PLANT PATHOLOGY

19

Butler, E.J



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PREFACE

THIS book is the outcome of a request made to Sir Edwin J. Butler, to consider a revision of his book *Fungi and Disease in Plants*, written when he was Imperial Mycologist at the Pusa Agricultural Research Institute, India, and published in 1918. It had previously been suggested to Sir Edwin by many interested friends that he should revise the book, substituting for the tropical diseases with which the second part of the book is concerned the plant diseases which are of importance in Great Britain, including those common also in other countries. After due deliberation Sir Edwin acceded to the writer's request and collaboration followed.

It is a matter of profound regret that Sir Edwin Butler did not live to see the publication of this volume. Despite ill-health he was able, by dint of his wonderful courage and pertinacity, to complete his accepted task with the exception of the bibliographies and illustrations. He died in April of 1943, and the book was completed a few months later. It was inevitable that the difficulties of the war and post-war years should have delayed publication, but every effort has been made to bring the text up to date.

In view of the great amount of research on plant pathology that had accumulated during the quarter-century since the publication of the old book, it was decided that the volume should be a new one. Much thought was given to the plan of the book. This remains much the same; namely, Part I dealing with 'General Principles', and Part II with 'Selected Diseases'. The former was written mainly by Sir Edwin Butler and the latter by the present writer, but there are various contributions by each of us in both parts. For the second part, two methods were considered: firstly, that of giving very brief descriptions of a large number of diseases under the causal parasites, thus following roughly a classification of the fungi, etc.; and secondly, describing in some detail only the more important diseases under the common host attacked, a plan of which Sir Edwin was strongly in favour and one which the writer has found, after long experience in teaching mycology to University students, to give a better grasp of the principles of the subject. This method, which was finally adopted, rightly gives prominence to the host, and is perhaps preferable when dealing with the disorders of agricultural and horticultural crops. Owing to the increasing attention now being given to virus and non-parasitic diseases and the uncertainty prevailing with regard to specific symptom-pictures, the properties of the viruses implicated and virus nomenclature, it was thought advisable to deal with these diseases first in a general way, for which reasons they appear in the first part, and to write specifically only on a few of the better-established, though still difficult types in the second part of the book. Though a few deficiency and physiological diseases are described in the second part of the book, a general treatment of these disorders is also included in the first part, for the reason that in no instance has a clear

picture of the symptoms been given of the disturbances brought about by these diseases.

The book has been framed to meet the requirements of students of mycology, plant pathology, agriculture, horticulture, and forestry, and the opening chapters deal in a sufficiently elementary manner to make the subject easy of approach for those who are making a beginning of the study of disease in plants.

As the work of finding illustrations for the book devolved upon the writer, he desires to take this opportunity of placing on record the ready response and kindness of mycologists at home and abroad to his appeals for photographs and diagrams; many of these have not hitherto been published. In particular he is under deep obligation to Dr. C. E. Foister and Dr. Mary Noble of the Department of Agriculture for Scotland, who gave unstinted help with the illustrations, as well as to Mr. D. Walters Davies, Cardiff, Prof. Robert McKay, Dublin, Prof. K. O. Müller, Dr. Dillon Weston, Cambridge, Dr. W. M. Ware, Wye, Dr. Wormald, East Malling, Dr. Bewley, Cheshunt, Miss Blackwell, Royal Holloway College, and Mr. L. Ogilvie, Long Ashton, for numerous illustrations or the loan of literature.

Sincere thanks are also expressed to all mentioned below, who sent photographs or diagrams, or gave permission to use material from their published works: Dr. Ainsworth, Mrs. Alcock, Dr. Ruth Allen, Mr. Beaumont, Dr. Bawden, Dr. Bennett, Prof. Barker, Dr. G. Bond, Dr. Boyd, Dr. M. R. Brown (Gilsen), Mr. Buddin, Dr. Cadman, Dr. A. H. Campbell, Mr. K. St. G. Cartwright, Prof. Chesters, Dr. L. C. Cochran, Dr. Ivimey Cook, Dr. Cornford, Prof. Craigie, Dr. G. H. Cunningham, Mr. W. R. Day, Dr. Dennis, Dr. Dovaston, Dr. F. L. Drayton, Dr. Bayliss-Elliott, Dr. A. R. Gemmell, Dr. Garrett, Dr. Mary Glynne, Mr. H. P. Gould, Mr. D. E. Green, Dr. Gregory, Prof. Dame Helen Gwynne Vaughan, Dr. Harris, Dr. Hickman, Dr. G. G. Hedgcock, Prof. Howitt, Dr. Hutchinson, Prof. Ingold, Mr. A. Powell Jones, Dr. L. W. Koch, Dr. L. O. Kunkel, Mr. A. J. Louw, Dr. J. H. J. van de Laar, Dr. du Plessis, Mr. Marsh, Dr. Millard, Mr. Moore, Mr. D. G. Milbrath, Dr. Nattrass, Mr. T. R. Peace, Dr. Pontecorvo, Dr. I. W. Prentice, Mr. Reid, Prof. M. C. Richards, Prof. Riker, Prof. Robertson, Prof. Salmon, Mr. Samuel, Dr. Selman, Prof. Stakman, Dr. Perley Spaulding, Mr. Searle, Mr. H. J. Edgar, Miss K. Sampson, Dr. F. M. L. Sheffield, Prof. N. J. G. Smith, Dr. Eric Taylor, Miss Tetley, Prof. H. E. Thomas, Rev. Dr. E. E. Thomas, Mr. P. H. Thomas, Dr. Thung, Prof. Wadham, Prof. Wardlaw, Miss Turner, Miss Wakefield, Prof. Waterhouse, Dr. Western, Dr. Woodward, Mr. Wilkins, Mr. Wilkinson, Dr. S. Williams, Dr. Wiltshire, Dr. A. R. Wilson, Prof. F. A. Wolf, and Dr. Scott Wylie.

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For permission to reproduce the various illustrations, acknowledged in the captions, thanks are due to the Controller of His Majesty's Stationery Office, the Ministry of Agriculture, the Department of Agriculture for Scotland, the United States Department of Agriculture, the Directors of the Research Stations at Harpenden, Princes Risborough, Long Ashton, Cheshunt, and East Malling, and to the Editors of the *Annals of Applied Biology*, *Annals of Botany*, *New Phytologist*, *Transactions of the British Mycological Society*, *Forestry*, *Phytopathology*, *Journal of the Royal Horticultural Society*, *Journal of Pomology*, *Journal of Agricultural Science*, *Transactions of the Royal Society*, *Transactions of the Royal Dublin Society*, *Farming in South Africa*, *Scientific Horticulture*, and *Gardeners' Chronicle*. The *Review of Applied Mycology* has been of inestimable value in connection with foreign literature.

The writer wishes to thank the Publishers, in particular Mr. L. J. F. Brimble, joint editor of *Nature*, and Mr. H. Cowdell, for help and advice. He is also under obligation to the Executive Committee of the Carnegie Trust for help to defray part cost of the illustrations, and to the Leverhulme Trust for a Grant that enabled him to devote full time to this work.

S. G. JONES

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BARLEY : Loose Smut (*Ustilago nuda*), Covered Smut (*Ustilago hordei*), Brown Rust (*Puccinia anomala*), Leaf Stripe (*Pyrenophora graminea*), Foot Rot (*Ophiobolus sativus*), Net-Blotch (*Pyrenophora teres*).

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PEACH : Leaf Curl (*Taphrina deformans*), Shot Hole (*Clasterosporium carpophilum*).

STRAWBERRY : Red Core (*Phytophthora fragariae*), Leaf Spot (*Mycosphaerella fragariae*), Yellow Edge (*Virus*), Crinkle (*Virus*).

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GOOSEBERRY : American Mildew (*Sphaerotheca mors-uvae*), European Mildew (*Microsphaera grossulariae*), Rust (*Puccinia pringsheimiana*), Die-back (*Botrytis cinerea*).

CURRANT : Leaf Spot (*Pseudopeziza ribis*), Rust (see *Cronartium ribicola*), Reversion (*Virus*).

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DAHLIA : Smut (*Entyloma dahliae*).

CARNATION : Leaf Rot (*Heteropatella valtellinensis*).

ROSE : Mildew (*Sphaerotheca pannosa*), Black Spot (*Diplocarpon rosae*), Brown Canker (*Cryptosporella umbrina*), Stem Canker (*Leptosphaeria coniothyrium*), Rust (*Phragmidium mucronatum*).

NARCISSUS : White Mould (*Ramularia vallisumbrosae*).

TULIP : Shanking (*Phytophthora cryptogea* and *P. erythroseptica*), Fire (*Botrytis tulipae*), ' Breaking ' (*Virus*).

HOP : Downy Mildew (*Pseudoperonospora humuli*), Powdery Mildew (*Sphaerotheca humuli*).

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OAK : Mildew (*Microsphaera alphitoides*).

SYCAMORE : Leaf Blotch (*Rhytisma acerinum*).

LARCH : Root Rot (*Armillaria mellea*), Heart Rot (*Fomes annosus*), Canker (*Dasyscypha willkommii*), Leaf Cast (*Meria laricis*).

SCOTS PINE : Needle Cast (*Lophodermium pinastri*).

WEYMOUTH PINE : Blister Rust (*Cronartium ribicola*).

DOUGLAS FIR : Needle Cast (*Rhabdocline pseudotsugae*), Swiss Needle Cast (*Phaeocryptopus gaeumannii*), Phomopsis Disease (*Phomopsis pseudotsugae*).

OTHER ROTS OF STANDING TIMBER (in Willow, Elm, Oak, Sycamore, Beech, Poplar, Sweet Chestnut, Birch, Walnut, Horse Chestnut, Ash, Larch, Scots Pine, Weymouth Pine, Spruce) :

Conifers : *Polyporus schweinitzii*, *Fomes pinicola*, *Trametes pini*, *Stereum sanguinolentum*.

Hardwoods : *Polyporus sulphureus*, *P. squamosus*, *P. betulina*, *P. hispidus*, *P. frondosus*, *P. dryadeus*, *Fomes fomentarius*, *F. igniarius*, *F. fraxinus*, *F. ulmarius*, *Ganoderma applanatum*, *Stereum hirsutum*, *S. gausapatum*, *S. frustulatum*, *Fistulina hepatica*.

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PART I

GENERAL PRINCIPLES

Chapter I

THE NATURE OF DISEASE ORGANISMS

Chlorophyll-free Plants

Plant diseases are due mainly to the action of fungi, but some are caused by bacteria, a few by plasmodia, while others in which no particulate living organisms have, so far, been discovered, are believed to be due to virus infection. In plant viruses that have been studied in any detail the invisible agent multiplies with great readiness only in the living host from which the virus has been isolated in the form of a nucleoprotein. Other diseases, again, but of a non-parasitic nature, are attributed to physiological or to deficiency troubles, and probably all are influenced by the factors of the environment.

Including for the moment the bacteria (fission fungi or Schizomycetes) with the fungi proper, one finds an enormous assemblage of species, at least as many as in the flowering plants, having few characters in common. They may be unicellular or composed of many cells, thread-like or forming sheets or masses, shapeless or beautiful in design, of limited or practically unlimited growth, of many colours. In the lowest group a cell wall — that standard criterion of the plant cell — may be present only in the sporing stage. Motility is not absent, either as the slow movement of plasmodia or the swimming of zoospores or the flagellar movements of many bacteria. In structure some approach closely to the green algae and, as the name Phycomycetes or algal-fungi reveals, it was long a popular suggestion and is still sometimes upheld that they were derived from algae ; the best modern view, however, is that they arose from colourless unicellular protozoa, and it is even maintained that, since the green algae probably arose from similar protozoa that had acquired chlorophyll, and since the rest of the green plants have developed along that path, it is logical to regard the fungi as a distinct phylum, neither animals nor plants but co-equal in their origin with the green organisms forming the plant kingdom. Normally, however, they are treated as plants, included in botanical works and courses, and they may be accepted as such.

The chief characteristic of the fungi is the absence of chlorophyll, even in those members which happen to be coloured green. This carries with it a profound difference in mode of life from other plants, for they take their food in the form of organic matter, whereas the chlorophyllous plants obtain their carbonaceous food from the carbon dioxide of the air, through the energy of sunlight (' photosynthesis '). The flowering plants, ferns, mosses, and algae build up their food out of dissolved inorganic salts from the soil or water, together with the constituents of the air. The fungi are unable to do this ; they are incapable of photosynthesis and so cannot make carbohydrates from carbon dioxide and water ; they must have organic matter or food already prepared

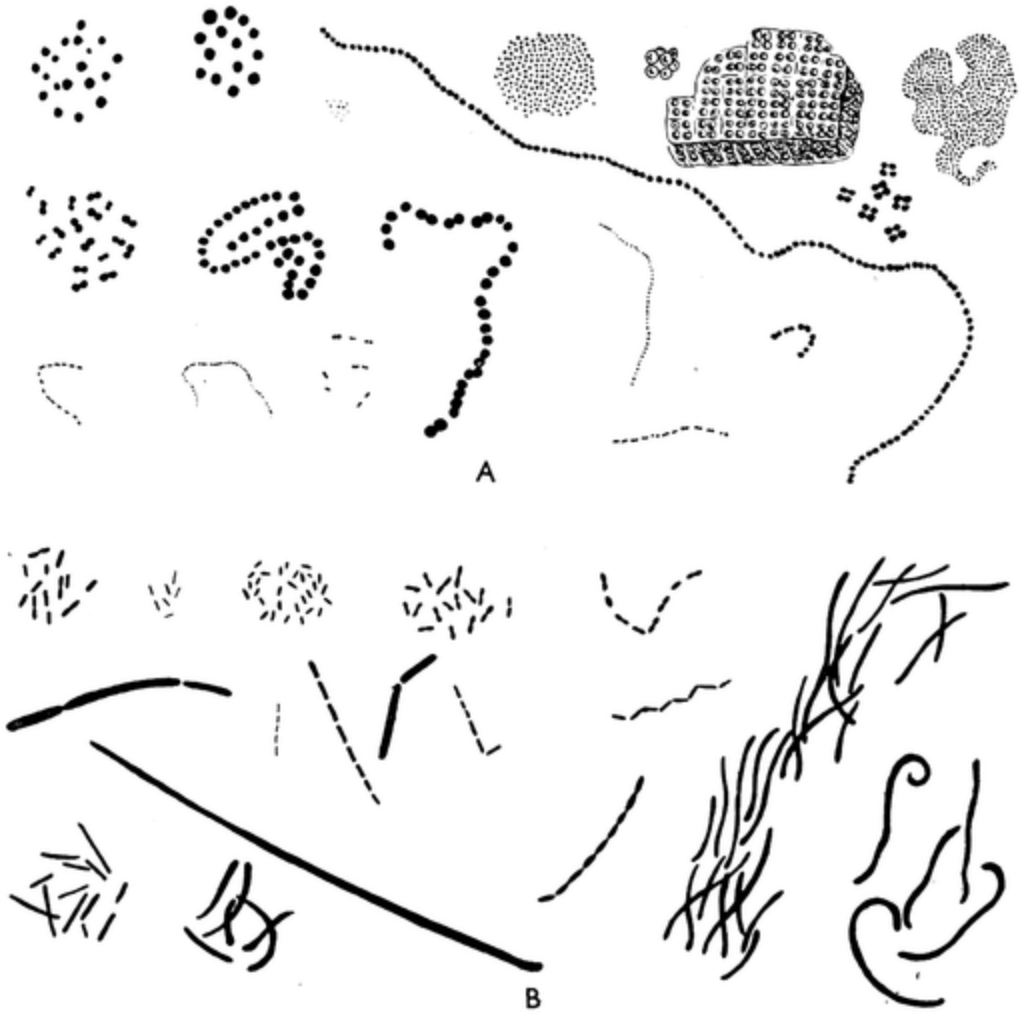


FIG. 1.—Bacteria. *A*, various types of cocci. *B*, rod, filamentous, and spiral types (after Baumgarten, from Lafar's *Tech. Mykol.*) (× 900)

by having been built up into the bodies of plants and animals, or having once formed part of these, like jam or leather. Incidentally, they are scavengers, without whose cleansing aid in decomposing organic matter, the earth would soon become unfit to sustain the higher forms of life.

BACTERIA

The bacteria really have very little in common with the fungi proper, except, in general, their mode of feeding. They are extremely minute, usually consisting of a single cell or a collection of more or less independent units (Fig. 1). Sometimes these units are arranged in branching threads which are hard to distinguish from those of the filamentous fungi. The fungal thread, however, is normally a part of the one individual, whereas a bacterial thread consists of a chain of individuals more or less loosely joined together. The branching is also different, the fungi having true branches, while in a bacterial column the spurious appearance of branching is due to imperfect separation of broken parts of the chain, which continues to give off new individuals above and below the break. The most important distinctions between the two groups are in the methods of growth and reproduction. Fungi normally grow only at the ends of the threads which compose their bodies, and multiply by forming spores at the ends of, or within special threads. Bacteria, on the other hand, have not got this purely end growth, and

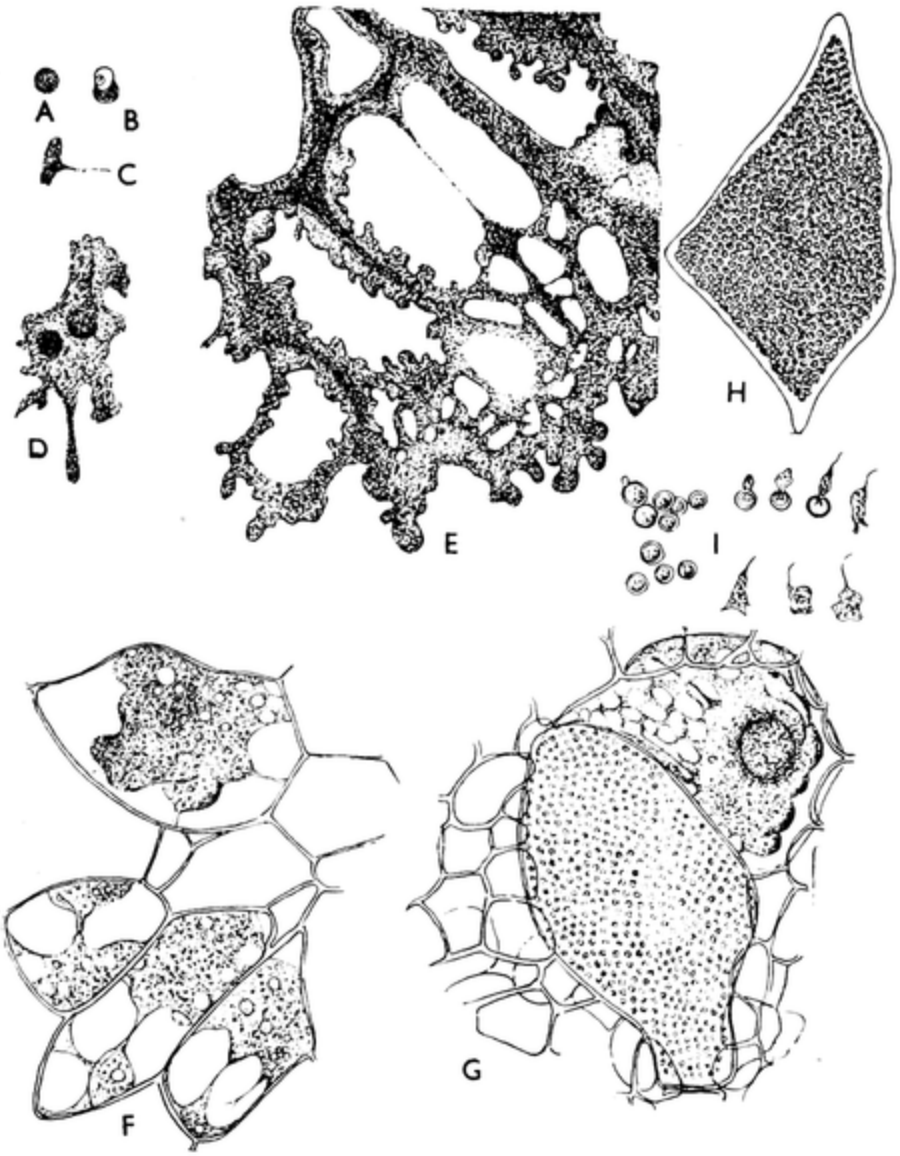


FIG. 2.—Plasmodia. *A–D, Chondrioderma difforme. A*, ripe spore. *B*, germinating spore. *C*, swarm cell. *D*, young plasmodium which has taken up two spores in its substance (× 230). *E, Didymium leucopus*, portion of a small reticulated plasmodium (× 33) (all after Cienkowski, from Sachs' *Lehrbuch*). *F–I, Plasmodiophora brassicae. F*, frothy plasmodia in the host cells. *G*, top cell with a frothy plasmodium, the lower cell showing the plasmodium segmenting to form spores. *H*, host cell full of the minute spores (× 213). *I*, spore germination and formation of swarm spores (× 470) (after Woronin)

usually multiply by each cell simply dividing into two cells, which frequently separate from each other and become independent. There are other less important differences as in the nuclei, cell wall, and so on. Being smaller and lighter than most spores of filamentous fungi the bacteria are even more efficient than the former in reaching and decomposing organic waste; as is well known, they were the chief subject of Pasteur's convincing demonstration against the old-fangled notions of spontaneous generation.

PLASMODIOPHORALES

The Plasmodiophorales are an aberrant group having apparent affinities with the chlorophyll-free mycetozoa or myxogastres, organisms close to the dividing line between

animals and plants. They are now usually placed near or in the primitive Chytridiaceae (Archimycetes). They have a power of spontaneous creeping movement not limited to the zoospores. Instead of a germ-tube (see next paragraph) the spore gives rise as a rule to motile swarm cells which fuse to form a small mass of naked protoplasm endowed with movement resembling that of an amoeba. This forms a multinucleate ' plasmodium ' or plasmatic body still without a wall and usually constituted by the amalgamation of several individuals (Fig. 2). In at least some of the genera the swarm cells (zoospores) function as gametes (see p. 563) and the plasmodium arising from the zygote forms sporangia, so that there are well-marked sexual and asexual phases in the life-cycle. Two important crop parasites belonging to this small family occur in the British Isles, namely *Plasmodiophora brassicae* and *Spongospora subterranea*, causing ' club root ' of cruciferous crops, and ' powdery scab ' of potatoes, respectively (see Part II, pp. 559 and 493).

The Fungi Proper

All known fungi, with few exceptions, originate from spores, bodies comparable with, though not strictly similar to, seeds, for they have no pre-formed embryo or germ. These spores germinate, requiring for the purpose much the same conditions as ordinary seeds. The result of germination is usually the protrusion of one or more fine filaments known as germ-tubes (Fig. 3) These grow and give off branches, the resulting branched thallus constituting the ' mycelium ' or vegetative part of the fungus, as distinct from the reproductive part. Each individual filament of the mycelium is termed a ' hypha '. In a limited number of primitive fungi the vegetative part consists of a single cell, usually round or oval, and this may become transformed into a reproductive cell with little change in shape, the life-history being a very simple one (Fig. 4).

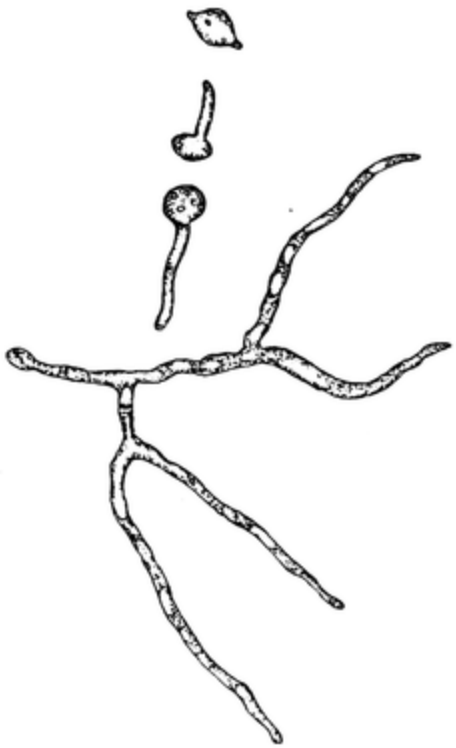


Fig. 3.—Germinating spores of *Clitocybe gigantea* (× 530) (after Bayliss-Elliott, *J. Econ. Biol.*)

The Vegetative Structure of Fungi

The Mycelium

At an early period in its growth the germ-tube and the hypha which arises from it usually become segmented into a row of cells by the formation of transverse walls (septa), each, when young, being perforated by a central pore sufficiently wide to allow protoplasmic granules and even nuclei to pass from cell to cell. Each cell is a hollow structure, bounded by walls of a transparent pliable material, which is sometimes cellulose as in the higher plants, but more often is a complex nitrogenous substance which resembles chitin, the material which forms the covering of insects. Within the walls, the cell is filled with a watery jelly, protoplasm, a material mainly protein in constitution, found in all living bodies and, in fact, the actual living matter itself. The cell cavity is not uniformly filled. Here and there little spaces occur (in older cells often united

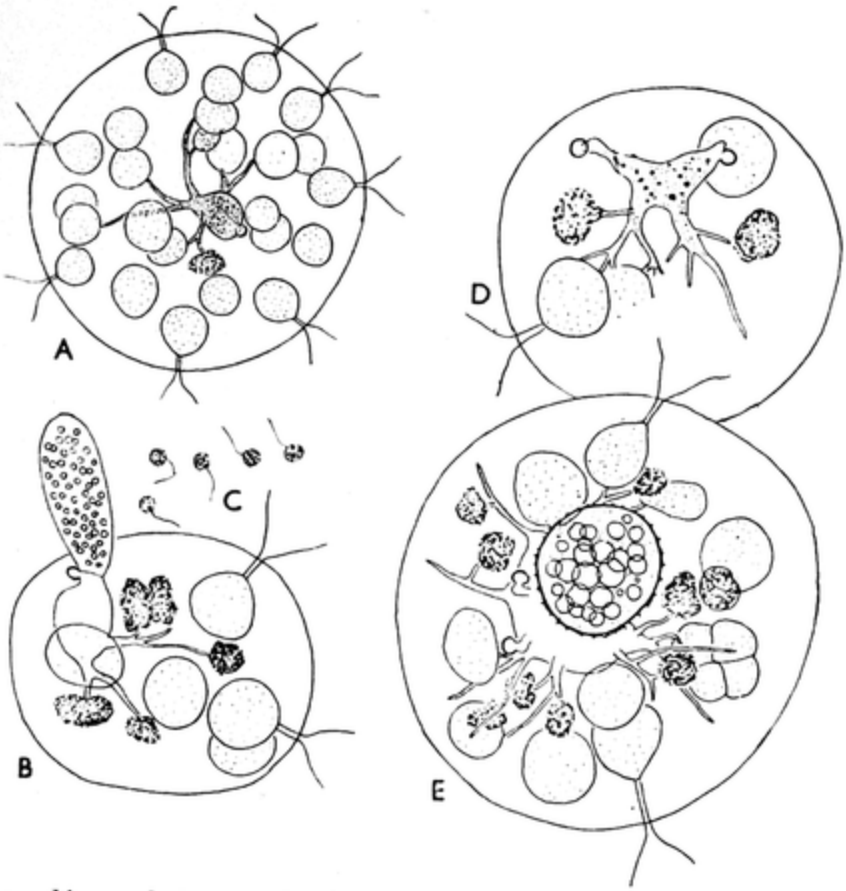


FIG. 4.—*Endocoenobium eudorineae*, a simple type of fungus (Chytridiaceae) parasitic on colonies of the green alga *Eudorina elegans*. *A*, the dark-shaded thallus of the fungus, in the colony (× 315). *B*, thallus with a mature zoosporangium (the small circles indicate oil drops and the position of the zoospores) (× 385). *C*, zoospores. *D*, fusion between two thalli to form a common thallus (× 430). *E*, a zygospore developed as a bud from the fusion cell (× 430) (after Ingold, *New Phytologist*)

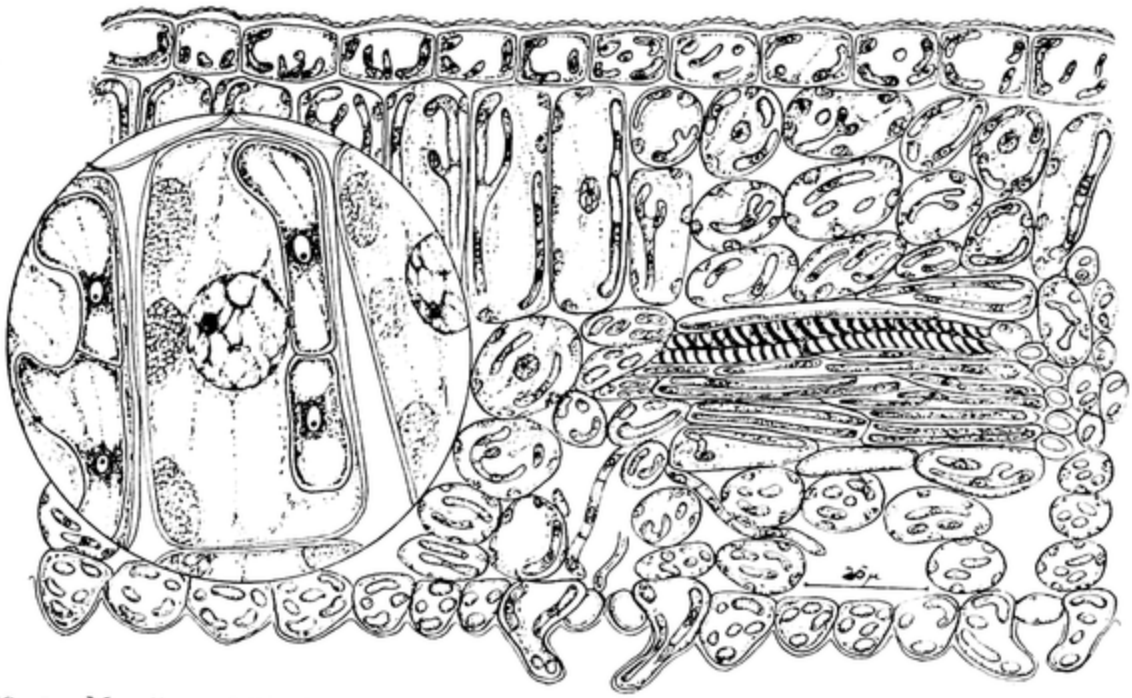


FIG. 5.—Mycelium of *Rhytisma acerinum*, causing ' tar spot ' disease, in the leaf of sycamore. The fungus occupies the host cells (intracellular mycelium); inset, uninucleate hyphal cells lying in the host cytoplasm along with the host nucleus and chloroplasts; note the vacuolated cytoplasm in the hyphal cells

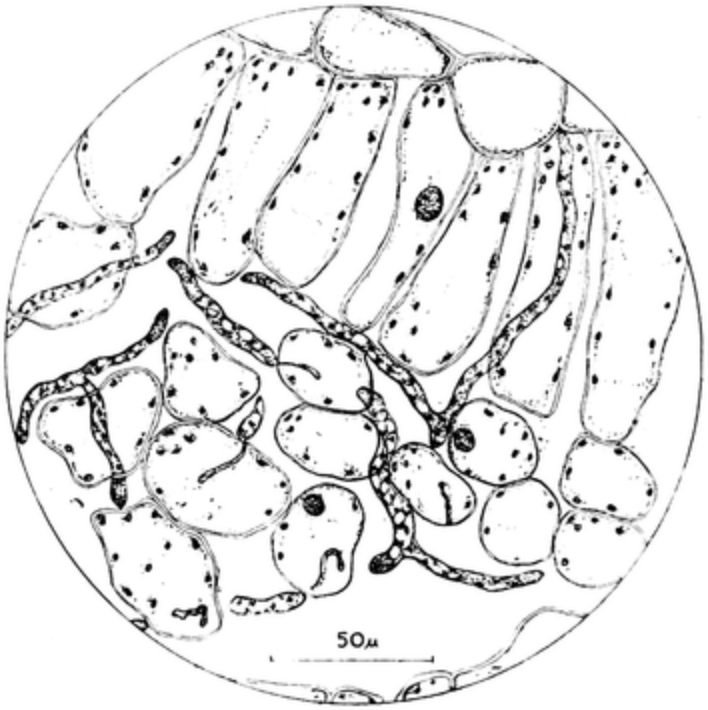


FIG. 6.—The young, coenocytic mycelium of *Phytophthora infestans* (causing potato blight) in the leaf of potato; haustoria are seen in some of the cells of the spongy mesophyll (from slides by Gonzalez and Lamont)

into a single space occupying much of the cell) and are known as 'vacuoles'; they are usually filled with water containing dissolved salts, sugar, and so forth, of the cell sap. Here and there also condensations of a peculiar kind of protoplasm form, which are termed nuclei (Fig. 5). One or more of these nuclei may occur in each

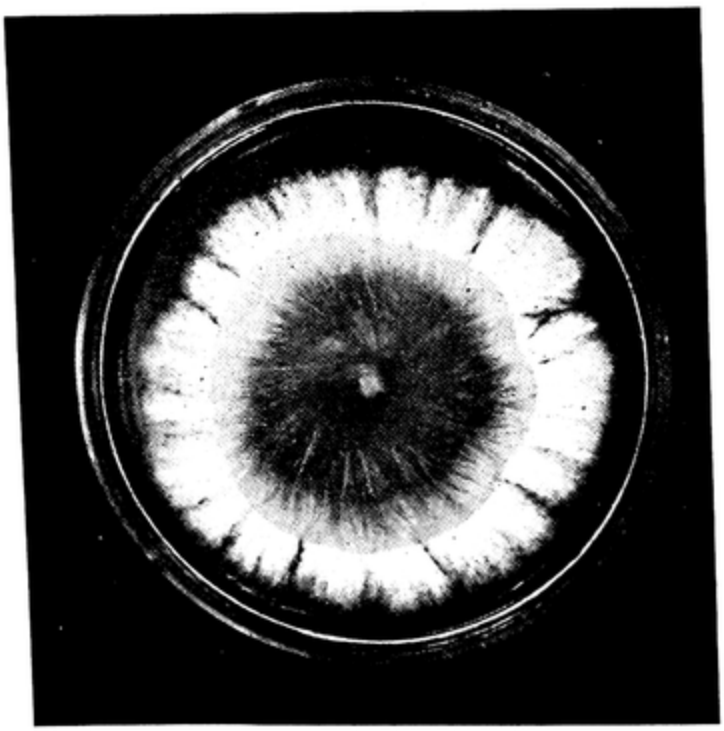


FIG. 7.—A colony of *Phomopsis aucubae* on malt agar showing adpressed feeding mycelium surrounded by flocculent subaerial mycelium (× ½)

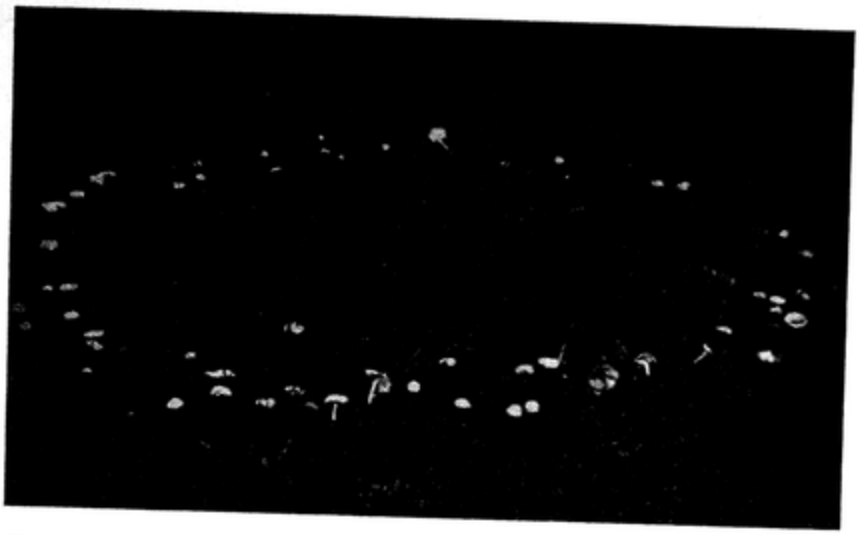


FIG. 8.—'Fairy ring.' Ring of fructifications caused by the perennial mycelium of *Marasmius oreades* (much reduced)

cell; two are habitually found in certain phases of the life-cycle of Ascomycetes and Basidiomycetes, playing a part in the complicated sexual reproduction of these fungi which will be further discussed below. In the lower fungi it is common, or even in some families habitual, to have no transverse walls in the active hyphae, which are 'coenocytic', that is to say, composed of a single large cell with many nuclei (Fig. 6). The nuclei are the bearers of the hereditary characters of the cell and also play an important part in regulating its metabolism.

Cells and spores are usually of microscopic size and contain not only proto-

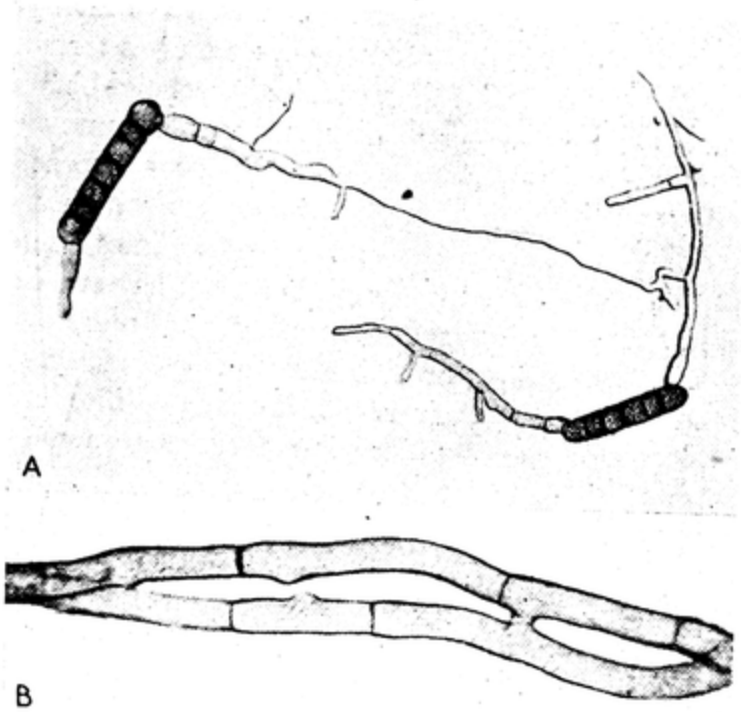


FIG. 9.—*A*, spores of *Helminthosporium* showing fusion of germ tubes, with *B*, showing anastomosis of hyphae (from microphotos by Dovaston)

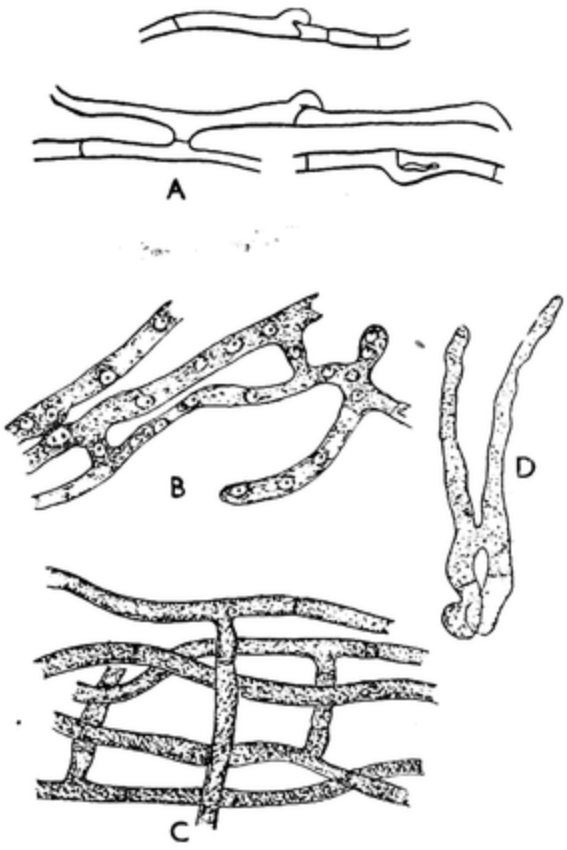


FIG. 10.—Clamp connections and anastomoses. *A*, clamp connection in *Hirneola auricula-judae* (×440) (after Green, *Ann. Bot.*). *B*, young hyphae of *Ascophanus aurora* showing lateral fusions; note the minute nuclei (×1066) (after Gwynne-Vaughan & Williamson, *Ann. Bot.*). *C*, hyphae of *Rhizoctonia crocorum* showing anastomoses (after Duggar). *D*, two conidia of *Lachnea cretea* with germ-tubes between which anastomosis has taken place (×280) (after Gwynne-Vaughan & Williamson, *Ann. Bot.*)

plasm with its nuclei but many other substances, such as reserves of sugars, fats, glycogen (a polysaccharide which replaces the starch of higher plants), crystals, resin, pigments, and so on. They are of many shapes, but on the whole the spores preserve considerable uniformity within the species. The prodigality of nature in its reproductive schemes is illustrated by the fact that a single giant puffball (*Calvatia gigantea*) has been estimated to bear some 100 million-million spores. It is little wonder that every substance suitable for colonisation by fungi is reached by air currents charged with spores, unless special precautions are taken to exclude them.

The body (' thallus ') of the simpler fungi usually consists of branching hyphae only (Fig. 7). It may be short or long lived. The ' fairy ring ' fungi, of which *Marasmius oreades* is the commonest in England, are the best examples of perennial mycelium, as they may survive for as many as four hundred years, producing a crop of toadstools each year (Fig. 8). In many cases unions termed ' anastomoses ' occur between neighbouring hyphae which may be from the same or different spores of the fungus concerned. These form bridges across which nutrient material and even sometimes nuclei pass from one hypha to another (Figs. 9, 10). Most fungi, however, have that part of the body which is involved in spore-bearing (the ' sporophore ') of more complex structure, formed of hyphae of special shape, or of bundles of hyphae, or of an interwoven tissue derived from hyphae by their repeated division into short cells. This tissue is termed ' plectenchyma ' or, since it resembles to some extent the parenchymatous tissues of higher plants, ' pseudoparenchyma ' (Fig. 16).

Chlamydospores and Gemmae

Hyphae of the ordinary vegetative mycelium may also become modified. Some of their cells may become provided with thickened walls, able to withstand adverse conditions, and may even assume a characteristic shape and colour. They are known as chlamydospores and, like true spores, are usually microscopic (Fig. 11). As they become separated from the mycelium when mature, and can germinate like spores, they play a part in the dissemination of the species which,

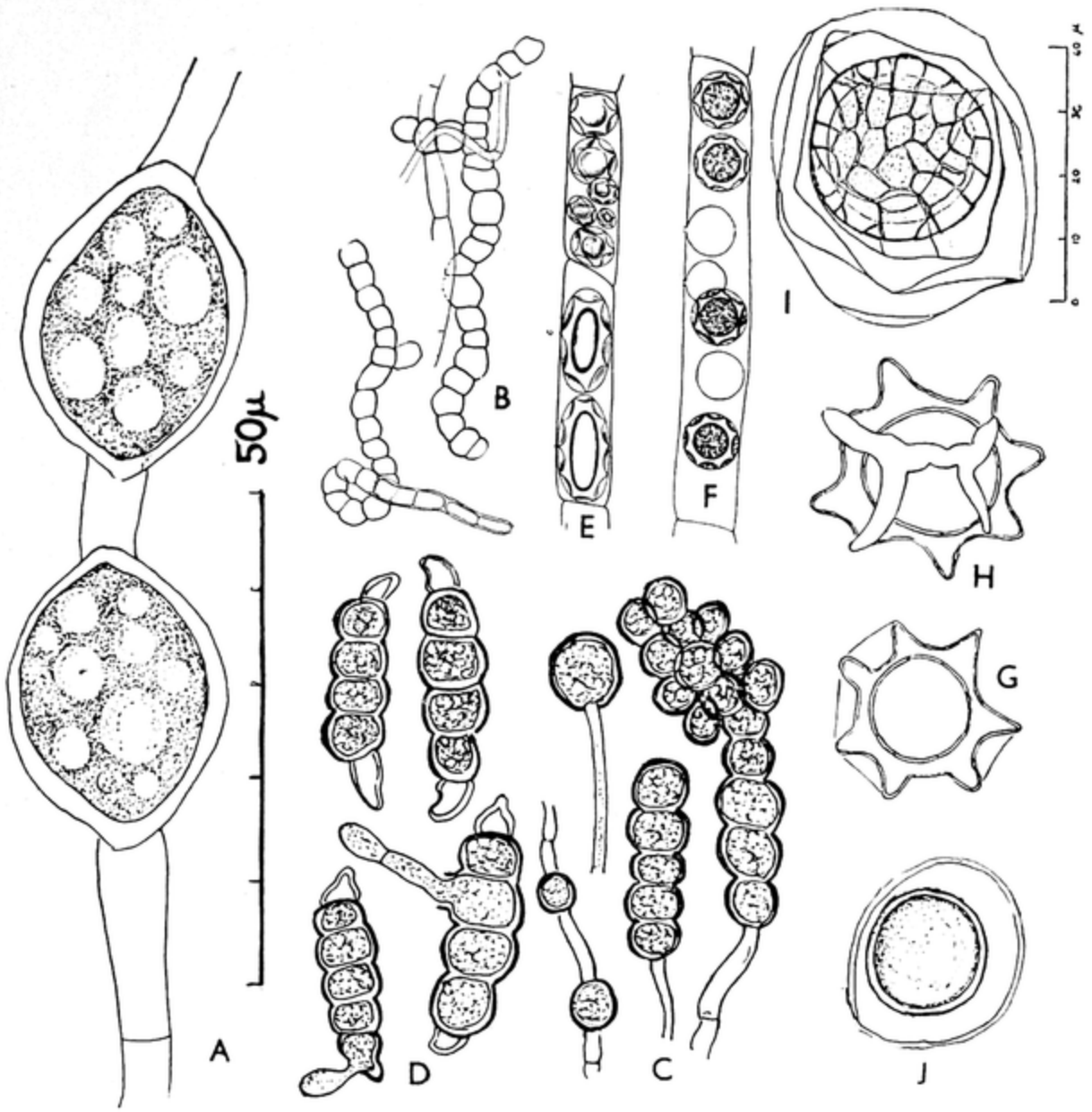


FIG. 11.—Resting spores. *A*, chlamydospores of *Azygozygum chlamydosporum* (after Chesters, *Trans. Brit. Myc. Soc.*). *B*, chlamydospores of *Helminthosporium avenae* (× 250) (after Dennis, *West. Scot. Coll. Agric., Bull.*). *C*, mycelial chlamydospores and sclerotial cluster of *Fusarium culmorum*, from wheat grain. *D*, conidial chlamydospores of same (both after Bennett, *Ann. App. Biol.*). *E*, cysts of *Asterocystis radicis*, from flax (after Marchal). *F*, cysts and zoosporangia of *Olpidium radicicolum*, from swede (after Bartlett). *G*, *H*, cysts of same from *Agrostis* (after Sampson, *Trans. Brit. Myc. Soc.*). *I*, oospore, within oogonium, of *Peronospora parasitica* (after Green, *J. Roy. Hort. Soc.*). *J*, oospore, within oogonium, of *Peronospora destructor* (after McKay, *J. Roy. Hort. Soc.*)



FIG. 12.—Gemmate mycelium of *Mucor prainii* (after Butler)

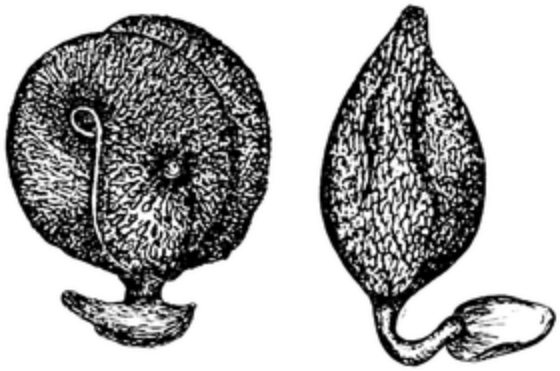


FIG. 13.—Two sclerotia of *Sclerotium stipitatum*, from white ants' nest (× 3) (after Butler)

on account of their durable nature, may be very important. Scarcely to be separated from the chlamydospores, except in being less durable, are the cells termed 'gemmae' (Fig. 12). They are usually thin-walled and continue to bud out cells similar to themselves instead of giving new hyphae. Under certain conditions the mycelium may be almost wholly transformed into these masses of rounded cells. Indeed, the familiar group of the yeast fungi (Fig. 57) may be regarded as an extreme case of the reduction of the mycelium to small gemmae endowed with a remarkable facility for budding out similar cells so as to form colonies which readily break up into their component units. This budding mycelium is frequently associated with fermentative activity.



A B

FIG. 14.—Sclerotia. *A*, of *Corticium kelerogа* on apple twigs (photo by F. A. Wolf). *B*, of *Botrytis* on inoculated leaves of *Gladiolus* (photo by Dennis)

Sclerotia

Much larger and more complex masses of vegetative hyphae than those producing chlamydospores may become united into 'sclerotia', sometimes measuring several inches across and as hard and heavy as a stone, but still formed wholly from vegetative hyphae variously thickened and divided so as to lose their filamentous character and become pseudoparenchymatous. The nests of white ants in hot countries often contain numerous rounded or spindle-shaped stalked sclerotia (*Sclerotium stipitatum*) belonging to the Ascomycete *Xylaria nigripes* (Fig. 13), while a sclerotium which may be as big as a toy football is formed by the basidiomycete *Polyporus mylittae* in

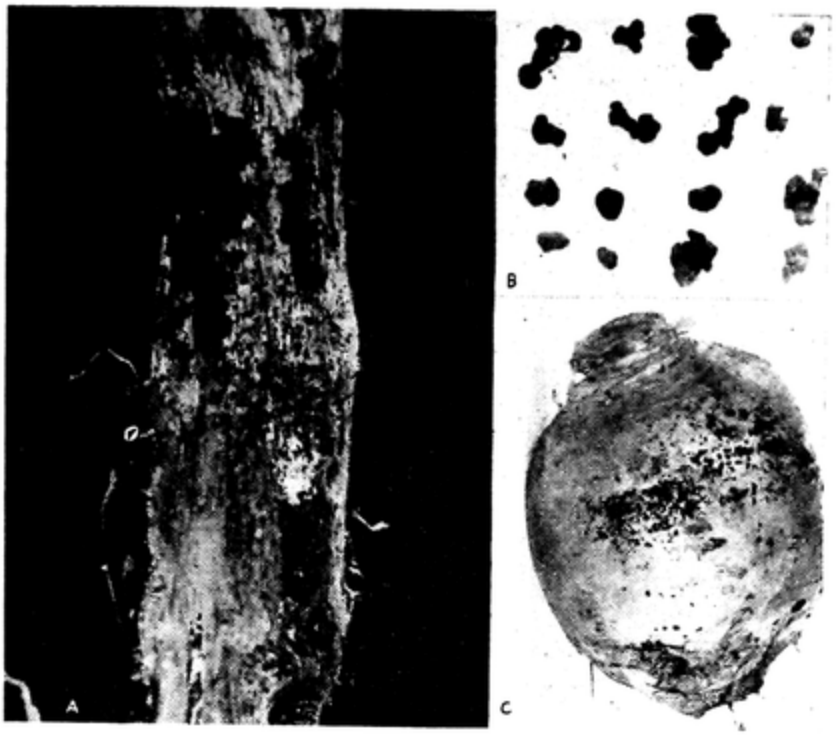


FIG. 15.—Sclerotia (*continued*). *A*, of *Colletotrichum atramentarium* on tomato stem (photo by Bewley). *B*, of *Sclerotinia trifoliorum* (photo by Wadham). *C*, of *Rhizoctonia solani* on swede (photo by Foister)

Australia, where it is known as 'black fellows' bread', as it is eaten by the aborigines. Several other Basidiomycetes (*Lentinus*, *Polystictus*, *Poria*) also form large sclerotia. Similar but much smaller types are known in both hot and temperate climates, the commonest in the British Isles being the often irregularly shaped sclerotia of *Botrytis* or *Sclerotinia*, of black-dot of potato and tomato *Colletotrichum atramentarium* and of *Corticium* (*Rhizoctonia*) *solani*, the cause of 'black scurf' of the potato, etc. (Figs. 14, 15). The round sclerotia of *Sclerotium rolfsii*, resembling very small seeds, are derived from a parasite destructive in the United States and elsewhere, while the still smaller, almost microscopic, *Sclerotium bataticolum* stage of the pycnidial (presumably ascomycetous) fungus *Macrophomina phaseoli* is ubiquitous in India and known in several other hot countries. In some sclerotia, such as those of *Botrytis* or *Sclerotinia*, a hard outer rind or cortex of thick-walled, often dark cells, with small cavities, may be distinguished from a softer pith (Figs. 16, 17). Besides being useful stores for reserve nutrient material, sclerotia are particularly well suited to withstand unfavourable extremes of temperature, dryness, and the like, and are valuable in carrying the organism over long periods of adverse conditions.

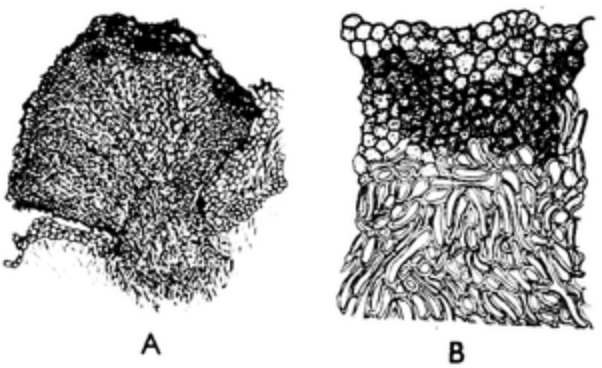


FIG. 16.—Sclerotial structure. *A*, section of a sclerotium of *Rhizoctonia* on root of sea-kale showing mycelial hyphae penetrating the cortical tissues of the root (after Salmon). *B*, section of a sclerotium of *Sclerotinia*; note the thick rind and 'cortex', and the formation of a pseudoparenchyma (after Brefeld)

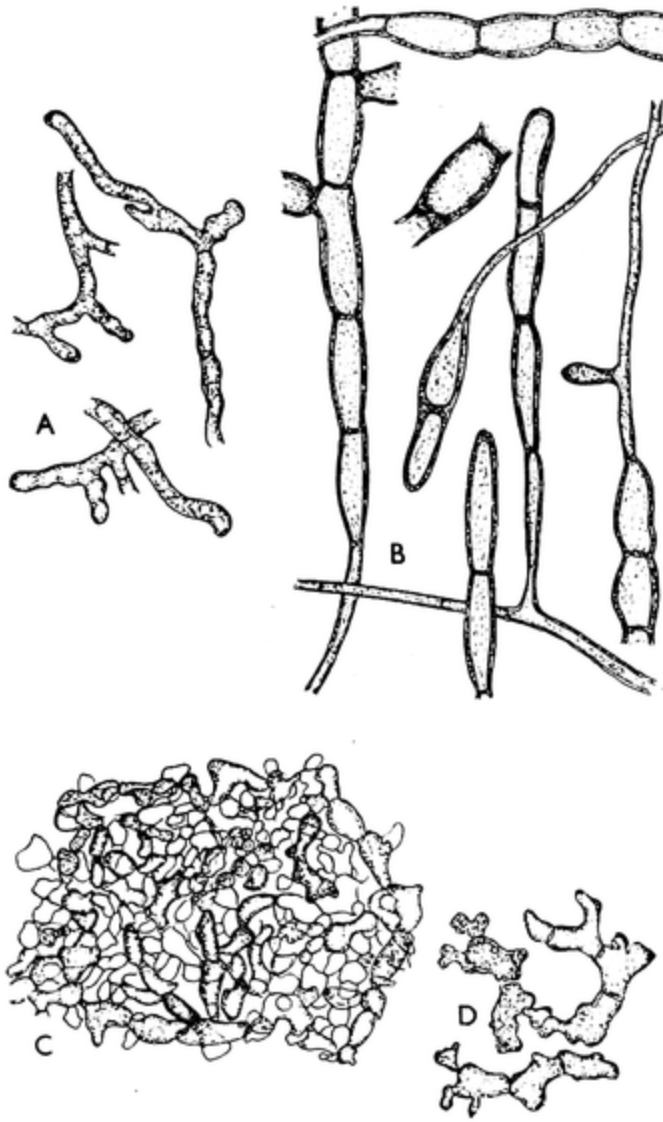


FIG. 17.—Sclerotial development. *A*, young hyphae of *Rhizoctonia crocorum*. *B*, characteristic hyphae of same from tufted growth covering the surface of the large sclerotia and, to a certain extent, of the 'infective cushions'. *C*, section of a large sclerotium of same. *D*, cells from a macerated sclerotium of same (after Duggar)

Rhizomorphs

Visible strands or cords of mycelium can often be seen on turning over heaps of leaves or at the base of puffballs and other large fungi. The simplest of these are generally white, and beyond being intertwined or sometimes laterally united they do not appreciably differ from an ordinary bundle of vegetative hyphae. More complex strands, known as 'rhizomorphs', are found in the vegetative thallus of some fungi. In the less differentiated types of these, ordinary filaments unite with specially tough hyphae and sometimes with hyphae adapted for the conduction of nutrients to constitute a part of the plant fitted for extensive exploration in search of new colonisation grounds. In the dry-rot fungus *Merulius lacrymans* (Fig. 18), they have been found passing through the mortar of walls to reach fresh structural woodwork on the other side. The more highly differentiated rhizomorphs are represented in the common parasite of tree roots *Armillaria mellea* (p. 907). These have been likened to purplish-brown or black leather shoe-strings, but they are often much branched, rather uneven in diameter, smooth and shiny when fresh, and luminous when young (Fig. 422 B). They consist of an outer black, rather brittle rind, composed of brown, thick-walled pseudoparenchyma, and a light-coloured tough flexible pith in which the filamentous character of the hyphae is largely preserved (Fig. 19). Though they grow normally by definite apical growth, keeping their root-like form, they can spread out into sheets of mycelium in certain positions, especially when they pass under the bark, or they may burrow deeply into the tissues of the root, forming tubular structures in the long axis of the root; these are still differentiated into a harder cortex and a loosely interwoven pith, and grow only at the apex. Similarly embedded rhizo-

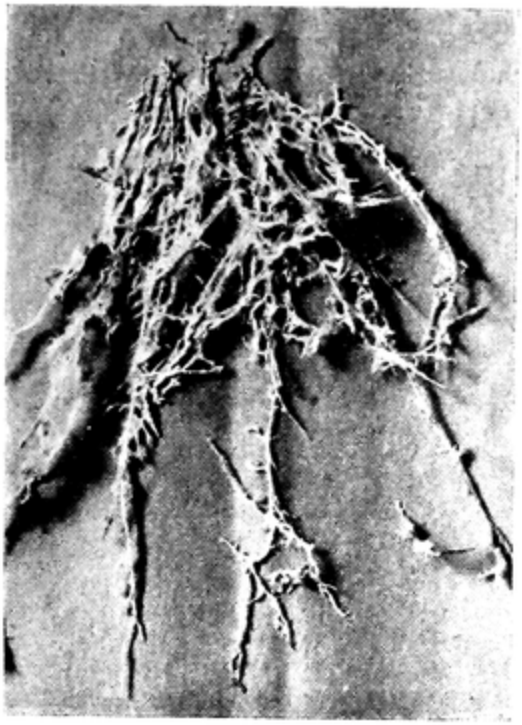


FIG. 18.—Rhizomorphs of *Merulius lacrymans* ($\times \frac{1}{2}$) (photo by Edgar)

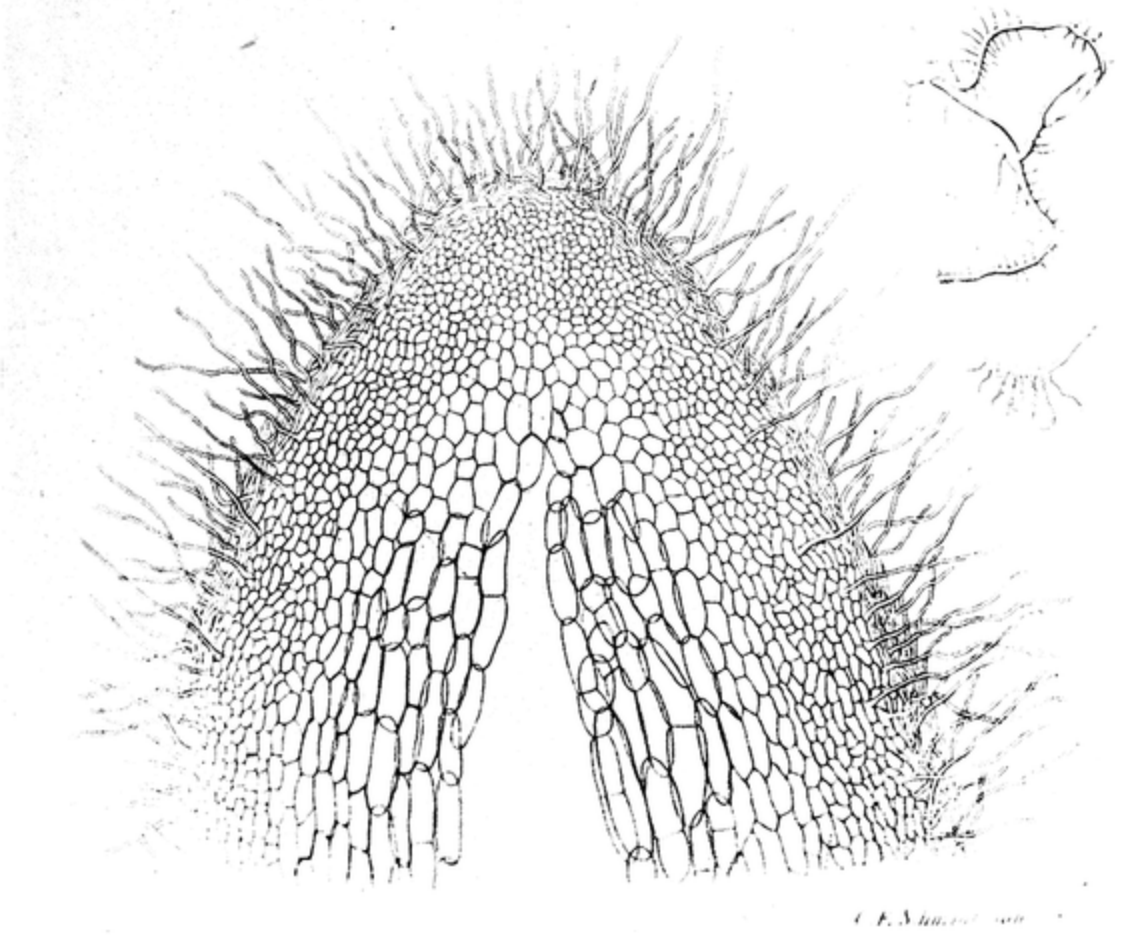


FIG. 19.—*Armillaria mellea*. Tip of a rhizomorph showing the superficial loose hyphae, the interwoven hyphae of the ' limiting layer ' covering a looser cortex, and a central medulla with an aerating cavity; on right, two portions of a young branching rhizomorph (after Zopf, from Brefeld's *Lehrbuch*)

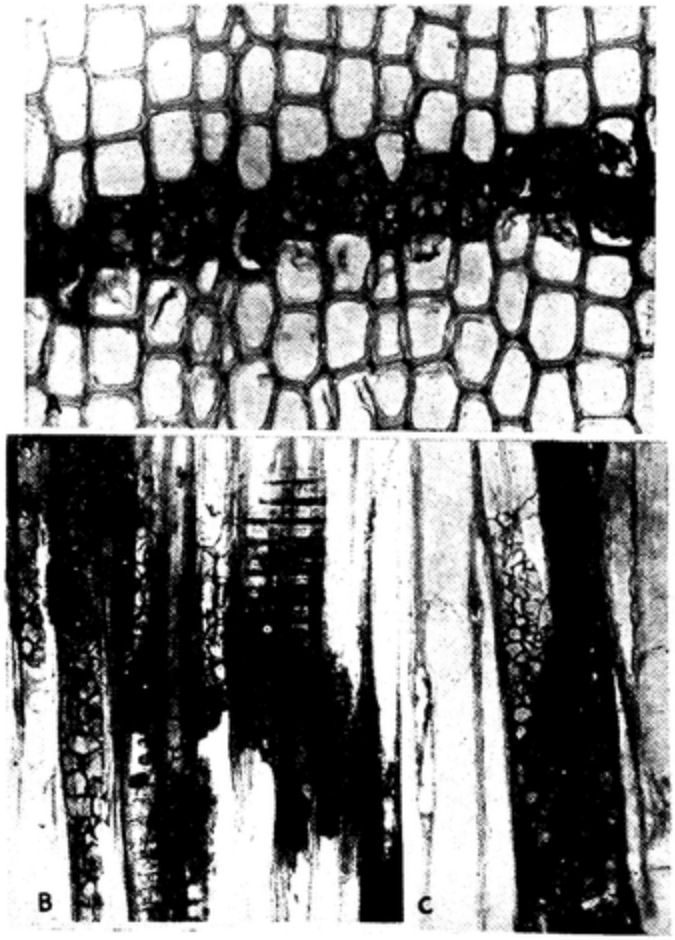


FIG. 20.—Black, or zone lines, due to *Armillaria mellea*. *A*, transverse section of pine wood showing the packing of the bladder hyphae in the tracheids (× 113). *B*, longitudinal radial section of same (× 66). *C*, longitudinal section of the black line showing close packing of the bladder hyphae (× 66) (after Campbell, *Ann. App. Biol.*)



FIG. 21.—Section of a larch stem attacked by *Armillaria mellea*; the black lines can be seen, especially on the right-hand side of the figure (after Hiley, from *Fungal Diseases of the Common Larch*, by permission of Oxford Clarendon Press)

morphs are formed by the Ascomycete, *Sphaerostilbe repens*, a widely distributed tropical fungus causing root rot of tea and other crops.

In the more serious of the root diseases of tropical plantation crops, such as rubber and tea, the rhizomorphs, which are the chief organs of spread, remain on or in the roots or other buried timber (whether alive or dead), and even in *Armillaria mellea* their ability to grow away from such supports and food-base is limited, and extension through firm soil, other than along wood, is almost negligible. Their function in the parasitism of the last-mentioned fungus is discussed in a later chapter (pp. 119, 131).

Allied to rhizomorphs are the raised black lines or narrow bands running longitudinally on the surface of roots attacked by *Armillaria mellea*, each representing the free margin of a dingy white sheet of mycelium (' xylostroma ') which disrupts the wood, often but not invariably in a radial direction. The xylostromata are capable of growth at their free edges and of causing infection when a diseased root comes into contact with a healthy one. They are much less often seen in temperate than in tropical forms of the fungus, in contradistinction to the rhizomorphs, which are common in temperate, but may be hard to find in tropical countries. The xylostromata arise by proliferation from a narrow band of deep brown cells which form sharply defined black lines in the tissues invaded by *Armillaria mellea*. These bands are sections of continuous sheets crossing all the tissues of the host from near the surface to the pith. They begin by a development of thin-walled, swollen, colourless, bladder-like cells completely filling the host cell cavity (Fig. 20). The bladder cells soon thicken their walls and turn brown. When fully developed, the black line is composed of a dense mass of angular, often rather small cells of a deep brown colour, and this colour also stains the walls of the host cells. The result is a continuous layer surrounding an area rotted by the fungus, and it has been suggested that it encloses a mass of fungal tissue, mixed with elements of the host, which is protected from adverse influences and to which the term ' pseudosclerotium ' has been applied (Fig. 21). Black lines of this type occur in the tissues invaded by various parasitic and saprophytic species of *Ustulina* (Figs. 153, 154), *Xylaria*, *Hypoxylon*, and the like.

Stromata

In many fungi, after a period of vegetative growth in the filamentous condition, the mycelium condenses here and there into pseudoparenchymatous masses termed ' stromata ', which are formed generally on or just below the surface of the substratum in which the fungus is growing (Fig. 22). These differ from sclerotia only in having usually a less regular shape and a margin in more frequent communication with the filaments of the rest of the thallus. They often, indeed, as in *Rhytisma*, only form flat crusts covering the deeper hyphae from which they have developed (Fig. 23). They commonly constitute a transitional stage between the purely vegetative and the reproductive parts of the fungus, and the term ' stroma ' is sometimes restricted to this pre-sporing stage. It is difficult, however, to accept this restriction, as stromatic crusts are common that seem to have no association with spore bearing.

The parts of the fungus plant so far considered are chiefly concerned with

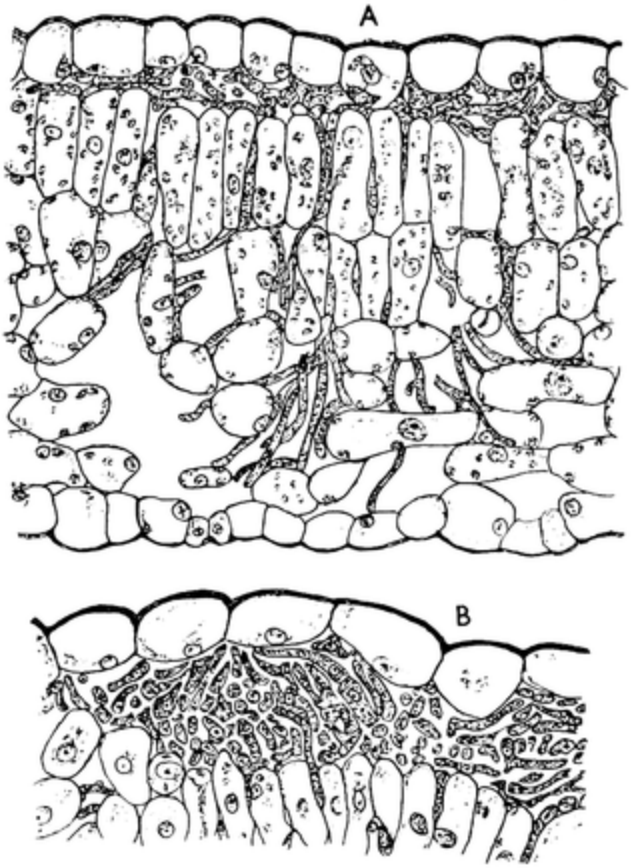


FIG. 22.—Development of the stroma. *A*, transverse section of leaf of *Rhamnus* showing the mycelium of *Puccinia coronata* in the tissues, the fungus aggregating under the upper epidermis to form a stroma, which, in *B*, is about to form a spermagonium (after Ruth Allen, *J. Agric. Res.*)

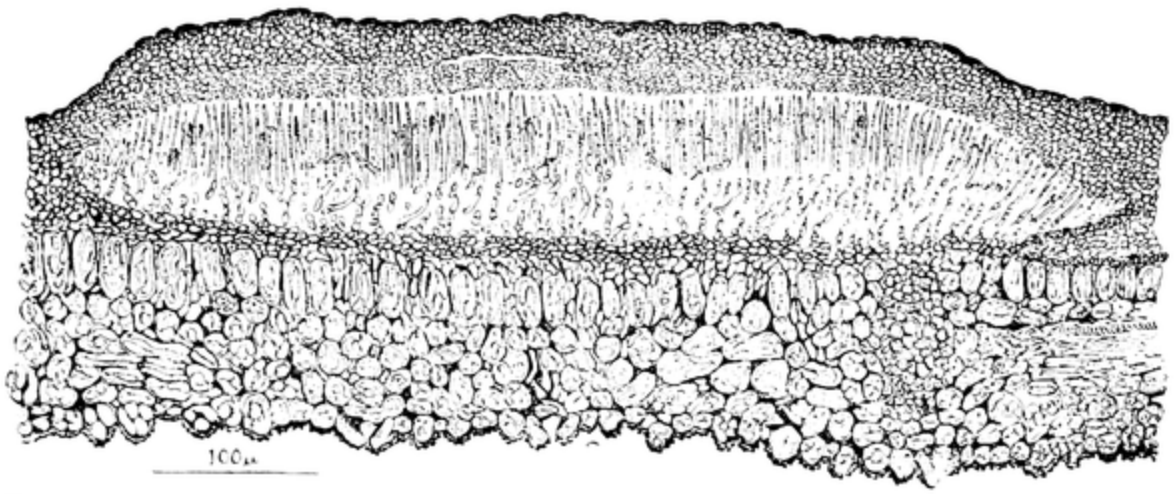


FIG. 23.—Development of the stroma (*continued*). Transverse section of leaf of sycamore showing affected cells of leaf filled with mycelium (intracellular mycelium) of *Rhytisma acerinum*; the mycelium has aggregated chiefly in the split upper epidermis in which a stromatic bed is formed, together with a thickened roof, prior to the development of an apothecium; the occupied leaf cells and hyphal bed (hypothecium) thus form a stroma. (See also Fig. 420 B)

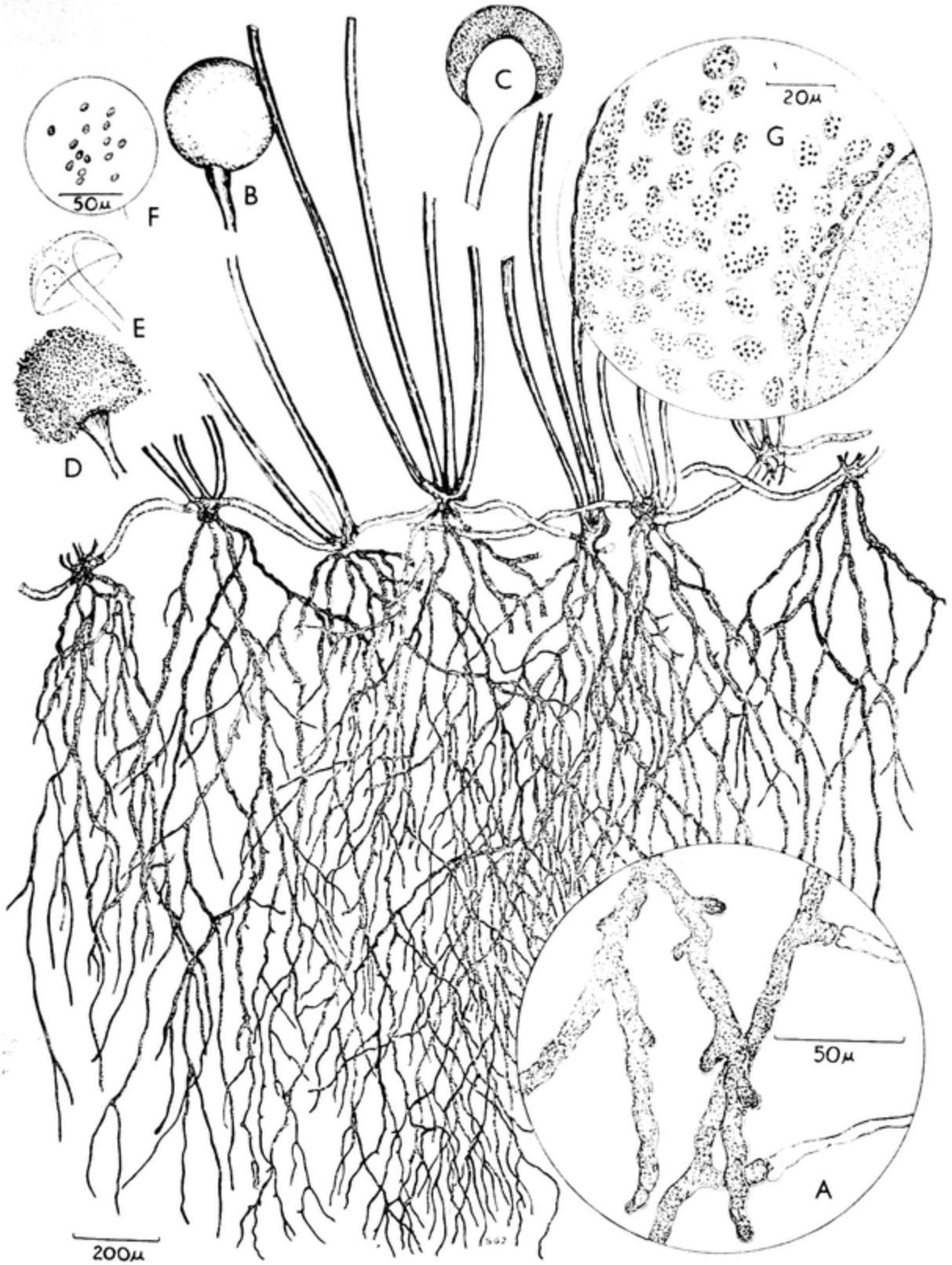


FIG. 24.—The black mould, *Rhizopus stolonifer*, growing in a deep medium of malt agar; note the richly branching rooting mycelium, without cross septa except when the hyphae become septated with age, as at *A* (inset), due presumably to a staling of the medium; the branching rhizome-like branches creep over the surface of the culture and give rise to groups of vertical, brownish sporangiophores which bear the spherical black sporangia, as at *B*. *C*, a sporangium in vertical section showing the central columella surrounded by spores. *D*, *E*, the dehiscence of a sporangium (see Fig. 73). *F*, the spores. *G*, a portion of the contents of a sporangium showing the multinucleate spores, sporangial wall, and portion of the columella

nutrition. The more specialised rhizomorphs, sclerotia, resting and budding cells, take part also in propagating the species, but this is ' vegetative ' propagation, on a par with propagation by cuttings, buds, suckers, and the like, amongst higher plants. The reproductive system proper is usually quite distinct and well defined.

Reproduction of Fungi

Sporophores

As the vegetative system is usually buried in the substance on which the fungus feeds — in the soil, in rotting organic matter, or in living plants — and as most spores should be formed in free access to the air to secure their dissemination, it is evident that some special arrangement must be made to bring the spore-forming

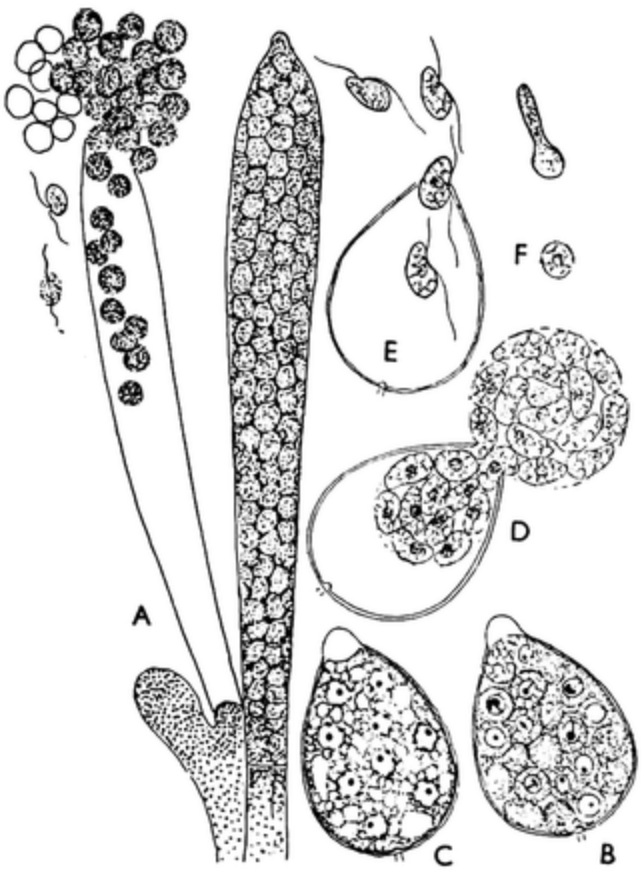


Fig. 25.—Zoosporangia. *A*, of *Achlya* (*Saprolegniaceae*). The zoospores collect at the top of the opened sporangium, and each becomes invested with a cell wall ; on the left, the zoospores are swarming away, leaving their cell walls behind (after de Bary). *C*, *B*, *D*, *E*, *F*, stages in the development of zoospores of *Phytophthora cactorum* (× about 700). *C*, the multinucleate sporangium. *B*, the zoospores well organised within the sporangium. *D*, zoospores passing into an evanescent vesicle before liberation, as in *E*. *F*, a zoospore and its germination (after Blackwell, *Trans. Brit. Myc. Soc.*)

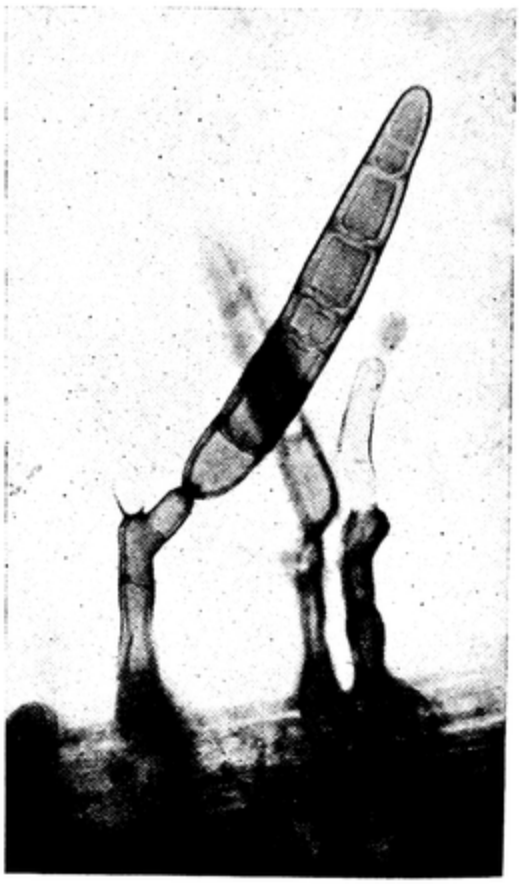


Fig. 26.—A conidiophore of *Helminthosporium* (from rye grass), from leaf kept in damp chamber for 3 days ; stained with lactophenol (× 250). Note the mature spore pushed aside and a new spore being developed in sympodial fashion (microphoto by Dovaston)

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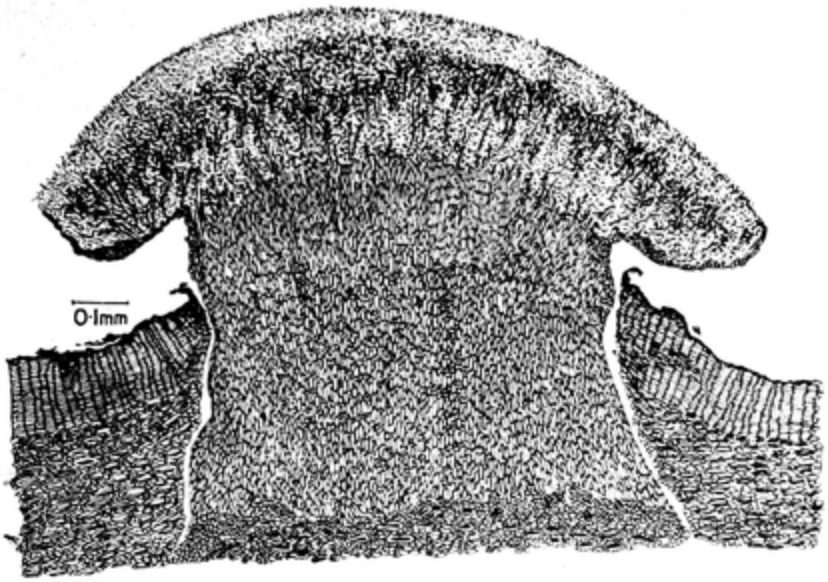


FIG. 27.—The sporodochium. Section of currant stem showing the conidial stage of *Nectria cinnabarina*. The entire hyphal mass of the sporodochium breaks through the bark of the dead stem (see Fig. 28)

parts where they will be exposed to the elements. This is done usually by the development of structures known as 'sporophores', raised vertically above the level of the general mycelium. The sporophore may consist either of a single hypha (simple sporophore) or of bundles or complex masses of hyphae (compound sporophore), and it bears on some part, usually towards the top, the spore of the fungus. The hyphae of which it is composed ordinarily differ from those of the vegetative mycelium in one or more characters, such as their vertical position, limited growth, or special structure (nature of cell wall, shape of the cells, characteristic mode of branching, and the like), and as they may be arranged in systems of great complexity, an almost endless series of different types of sporophore exists.

The simplest forms are found commonly in many of the lower fungi and in those moulds that are familiar on damp or rotting organic substances and that belong to several different families of the fungi (Fig. 24). In some of these, particularly such as have a considerable development of aerial mycelium in addition to the hyphae buried in the substratum and some of those that grow in water or in soil, special sporophores are almost or quite dispensed with, as being unnecessary to secure free dissemination. The spores are formed at the tip of, or laterally on, ordinary hyphae, or, in the aquatic species, in special cells ('sporangia'), on the hyphae, and are liberated in the air, the water, or the soil as the case may be (Fig. 25). More often, however, specialised sporophores are formed, and in the simplest cases these are single hyphae called sporangiophores, or conidiophores, often of distinct shape and size, which grow out into the air, and bear the spores on the tips or sides or on lateral branches (Figs. 24 B, 26).

In other cases, clumps of hyphae growing close together rise into the air. Sometimes these merely remain near together without lateral union (sporodochia) (Figs. 27, 28 A-I), sometimes they unite more or less closely into solid columns (coremia or synnemata) (Fig. 28 J-O).

Sporophores aggregated into crusts or layers are produced in many fungi.

Sometimes these are formed on the surface of the substratum in which the vegetative mycelium grows, sometimes, especially in parasitic and other fungi that grow in the stems and leaves of plants, just below the surface, which is eventually ruptured so as to expose the spore-bed (Fig. 29). In either case, the layer of sporophores may arise directly from the mycelium below, or there may first be formed a stromatic tissue composed wholly or in part of pseudoparenchyma. Common examples of spore-beds formed below the surface of the plant are the ' sori ' of the cereal and other rusts (Fig. 30), and the ' acervuli ' of many of the Ascomycetes and of the ' Fungi Imperfecti ' (Fig. 31). When formed on the surface, there may be a very large mass of stromatic tissue intervening between the vegetative mycelium and the layer of sporophores. Such compound sporo-



FIG. 28.—Development of sporodochium and coremium. *A*, branching sporodochial conidiophore of *Nectria cinnabarina*; the branches are close together, but free. *B–G*, stages in development of sporodochium of *Aegerita webberi*. *C*, early stage. *D–G*, chains of cells from the sporodochium (after Petch, *Trans. Brit. Myc. Soc.*). *H*, *I*, *Fusarium culmorum*. *H*, sporodochial elements. *I*, formation of conidia on the aerial mycelium (after Bennett, *Ann. App. Biol.*). *J–O*, coremial development in *Stysanus stemonites*. *J*, the origin from a single hypha. *K*, *L*, *M*, progressive branching of the vertical hyphae. *N*, *O*, the branches become erect and more or less coherent (after Hasselbring)

phores in the form of superficial stromatic crusts or tubercles may be found commonly on pieces of old wood, dead branches of trees, and the like, in damp situations. Sometimes the edge of the crust becomes free and curves over to form a sort of bracket (Fig. 32). This is the first stage in a process which has as its purpose the lifting of the spore-bearing part of the sporophore above the object on which it is growing. In more advanced types, so much of the crust may leave the surface that only a narrow band or stalk remains to join it to the latter (Figs. 33, 34, 35). In this way the bracket fungi found on trees and old wood, and the laterally attached toadstools, are formed. Finally, the whole spore-bearing part of the fructification may be raised from the surface on a central stalk (Fig. 36) and may be expanded into a cap at the top, as in the well-known mushrooms, or may remain as a simple or branched, vertically elongated body of various shapes.

In another type of compound sporophore, the spore-bearing hyphae are enclosed in roundish or cup-shaped or flask-shaped receptacles. To a common form of this type the name ' pycnidium ' is applied when it bears spores on the

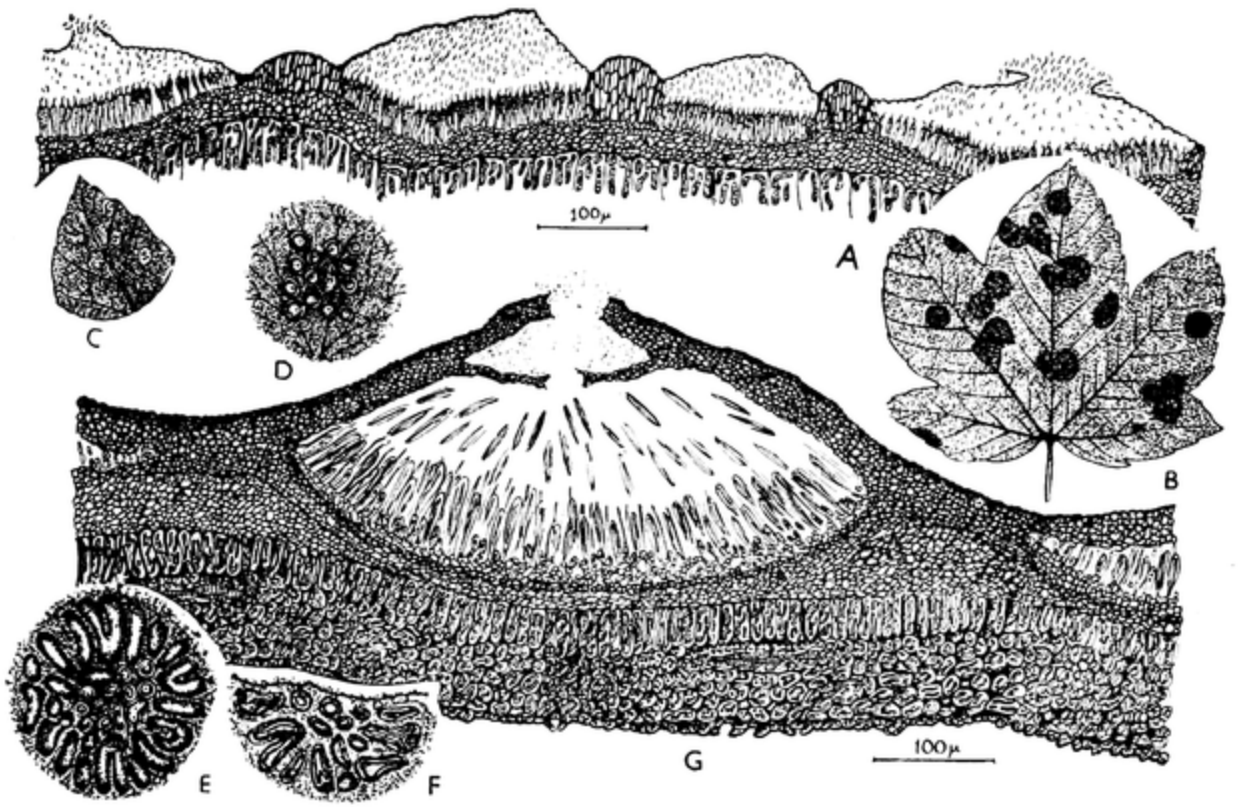


FIG. 29.—Sporophores aggregated into crusts. *Rhytisma acerinum* on sycamore. *A*, section of leaf stroma showing four acervuli (spermagonia); note the vertical spermatiophores arising from the stromatic bed and the very numerous, minute, comma-like spermatia. *B*, general appearance of the black stromata on the leaf. *C*, early infection showing small black spots (fringed with yellow; see Fig. 419). *D*, later, a group of acervuli on a stroma. *E*, surface view of a stroma at time of formation of apothecia around the margin of stroma; note, at centre, conversion of acervuli into small, round apothecia. *F*, surface view of an apothecial stroma at time of dehiscence. *G*, transverse section portion of stroma showing open apothecium in active discharge of ascospores; the median split in the roof is filled with mucilage and may open and close intermittently. The old leaf tissues below the ripe apothecium still form a firm stromatic bed. (See also Figs. 419, 420)

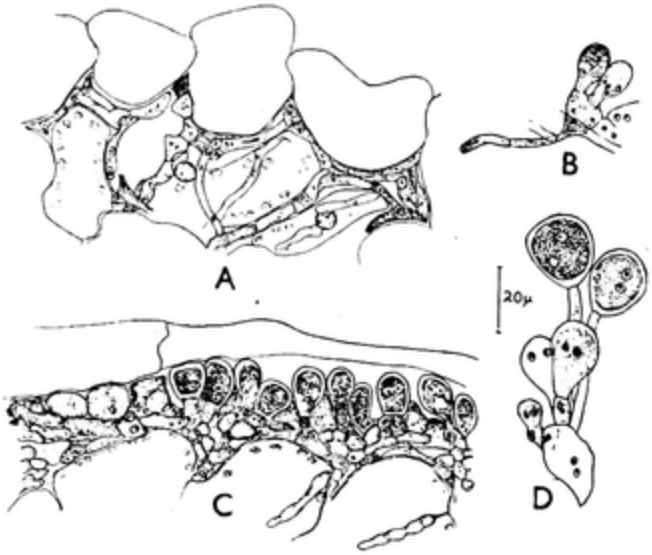


FIG. 30.—Development of the sorus. The uredosorus of *Puccinia anomala*. *A*, the sub-epidermal mycelium of binucleate cells. *B*, uredospore initials. *C*, portion of uredosorus; note the long hypha-like haustoria penetrating the host cells. *D*, a group of uredospores arising from a common basal cell (after D' Oliveira, from *Revista Agronomica*)

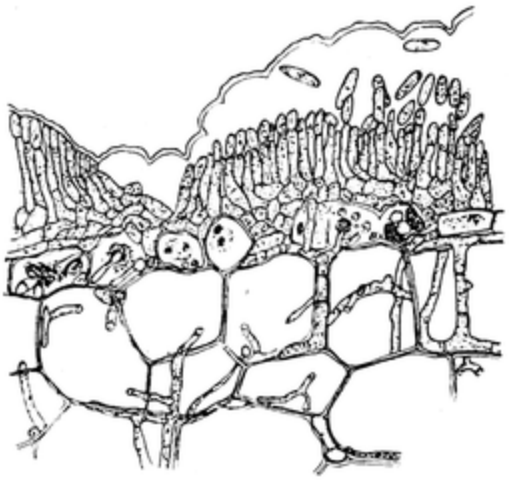


FIG. 31.—The acervulus of *Gloeosporium musarum* on banana. Formation of the spore bed below the cuticle (after Wardlaw, *Trinidad J. Agric.*)



FIG. 32.—Sporophores of *Stereum purpureum* on dead branch of a plum tree killed by 'silver leaf' disease. On the vertical branches, as here, the outer edges of the crust-like sporophores are free, but on horizontal branches, or fallen logs, the sporophores are resupinate flat crusts firmly attached all around the margin (reduced) (photo by Dillon Weston)



FIG. 33.—Sporophores of *Polystictus versicolor*. The bracket-like fructifications are free at the edge and curve, becoming more or less horizontal on the tree trunk (reduced) (photo by Bayliss-Elliott)



FIG. 34.—Sporophore of *Polyporus squamosus* on a dying trunk of elm. Note the dense porous hymenium on the under surface which, at the time the picture was taken, with a beam of strong sunlight falling across the sporophore, was sending out intermittently dense clouds of spores into the air ; note the short stalk of attachment

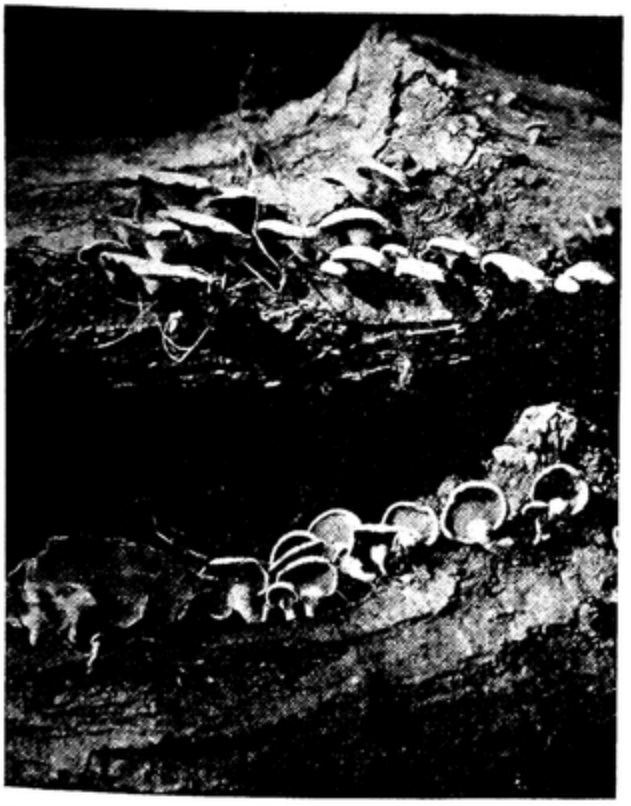


FIG. 35.—Sporophores of *Panus stypticus*. Top, the fructifications in side view, showing differentiation into a spore-bearing cap or pileus and a sterile stalk or stipe ; below, the same showing the gills and stalks (reduced) (photo by Bayliss-Elliott)



FIG. 36.—A group of the 'ink cap' fungus (*Coprinus*) showing the typical agaric kind of sporophore where a cap or pileus is clearly differentiated from a vertical stalk or stipe (reduced) (photo by Williams)

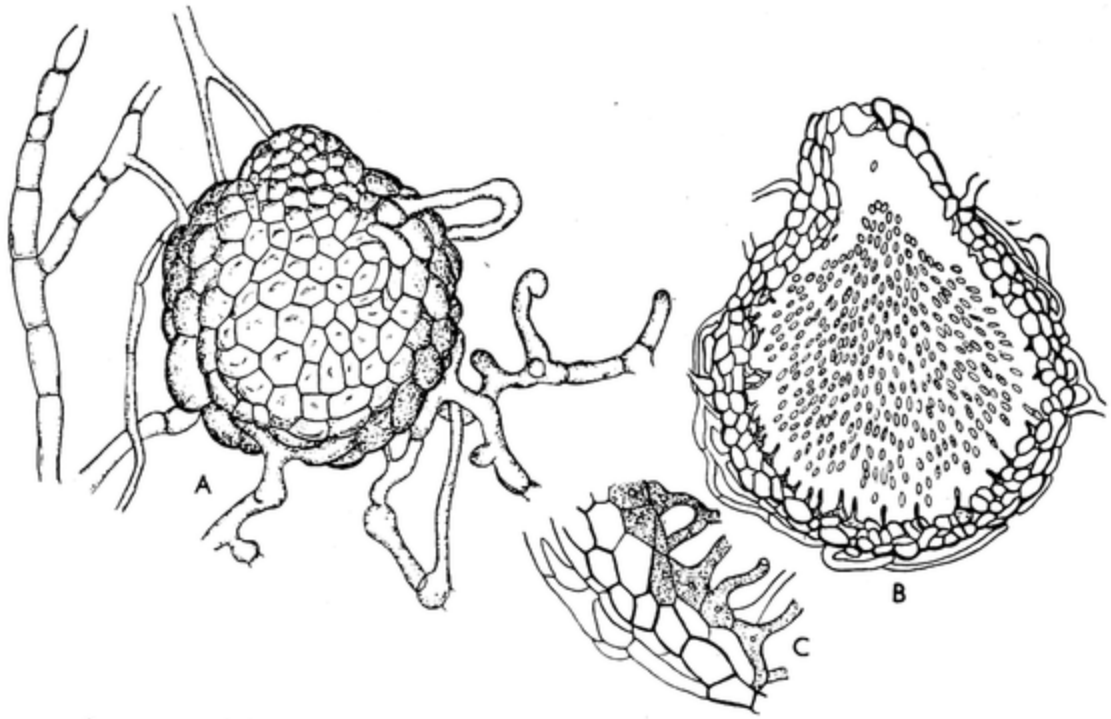


FIG. 37.—Structure of the pycnidium. The pycnidium of *Helminthosporium* sp., on *Cynodon*. *A*, the spherical pycnidium arising on the mycelium, with a short neck at the top. *B*, the same, in vertical section, showing a mass of pycnospores. *C*, details of wall structure and mode of origin of the short sporophores which abstrict the small spores (after Smith)

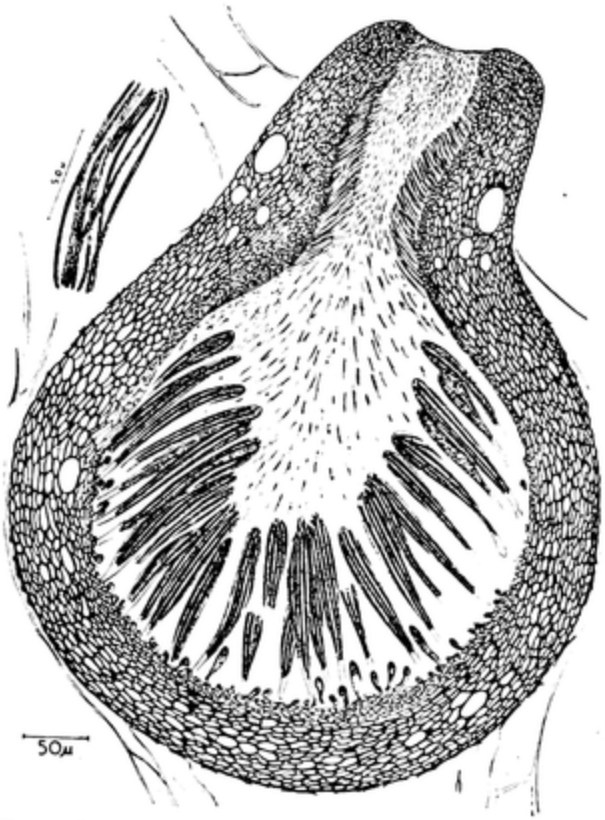


FIG. 38.—Structure of the perithecium. A perithecium of *Ophiobolus graminis* within the leaf sheath, from oat stubble. The interior is lined with club-shaped asci and the ostiole with periphyses ; on left, a bundle of the septated needle-shaped ascospores



FIG. 39.—The apothecium. Apothecia of the larch canker (*Dasyscypha willkommii* (× 2)

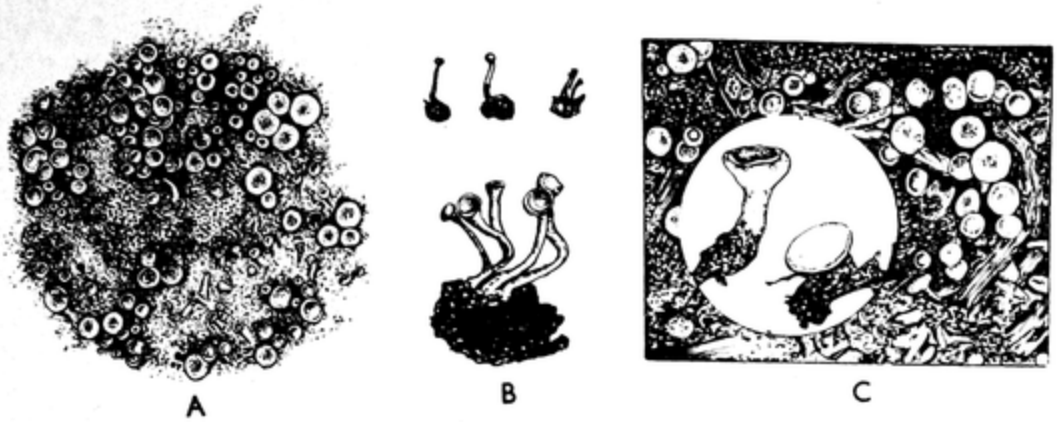


FIG. 40.—Apothecia. *A*, a group of apothecia of *Sclerotinia cinerea* from potato stem, developed in culture; note, towards the centre, their association with clusters of *Botrytis* conidiophores (adapted, after Groves and Drayton, *Mycologia*). *B*, a group of apothecia of *Sclerotinia minor*, a seed-borne fungus on clover; top, three seeds with young apothecia growing from sclerotia on the seeds (× 4); below, apothecia growing from a sclerotium in culture (× 5) (after Alcock, *Trans. Bot. Soc. Edin.*). *C*, apothecia of *Sclerotinia polyblastis*, on leaves of *Narcissus*; inset, apothecia produced singly from sclerotia (adapted, after Gregory, *Trans. Brit. Myc. Soc.*)

outside of the fertile hyphae (Fig. 37), and 'perithecium' when the spores are enclosed in the sac-like hyphae (asci) characteristic of the group of fungi known as the Ascomycetes. Frequently the term perithecium is reserved for these fructifications when they are distinctly flask-shaped and opening by a mouth or ostiole in the neck (Fig. 38), the term apothecium being given to the forms which open wider so that they appear like shallow cups or saucers (Figs. 39-41), and the term cleistocarp or cleistothecium for the forms which are more or less spherical and open irregularly or by a general breakdown of the wall (Figs. 43, 44). The perithecia may be formed either on the surface of the substratum, e.g. *Nectria* (Fig. 42 B),

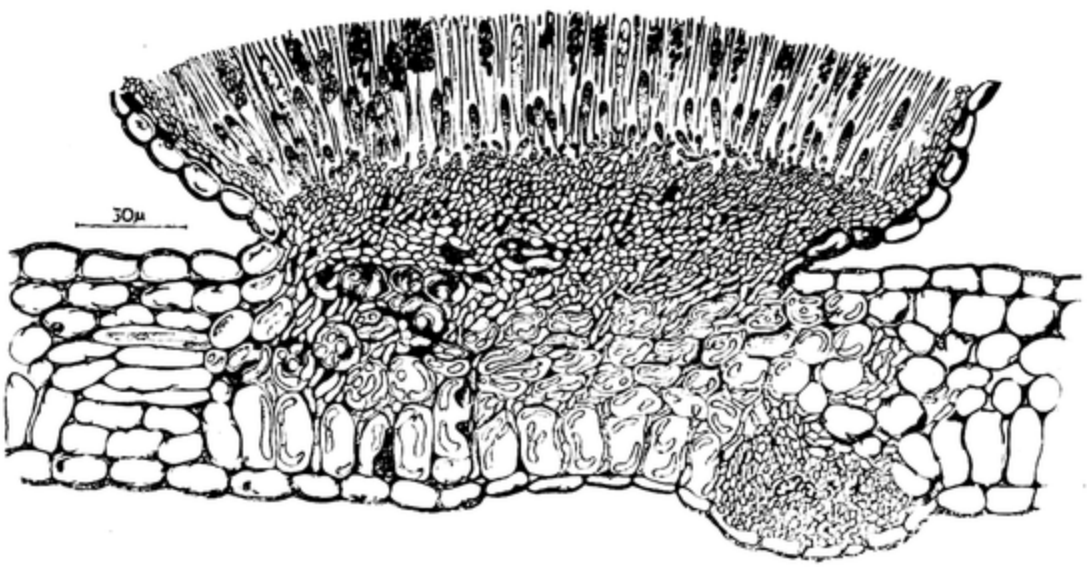


FIG. 41.—Structure of the apothecium. Transverse section leaf of clover showing fully developed apothecium of *Pseudopeziza trifolii*; the mycelium occupies the host cells between which is an accumulation of black substance formed by the fungus at the expense of the host cell-wall material; the club-shaped asci contain eight ascospores (mainly in two rows); the paraphyses are numerous and slender; note a young apothecium at the lower epidermis

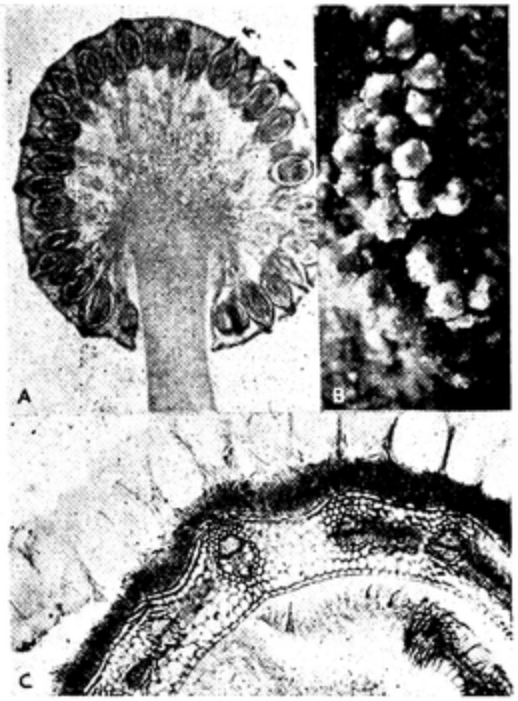


FIG. 42.—Perithecial stromata. *A*, spherical stromatic head of *Claviceps purpurea* showing the numerous sunken perithecia, with slightly protruding necks (×70). *B*, the spherical perithecia of *Nectria galligena*, more or less free from each other (not forming a continuous stroma), erumpent and attached to the host only at the base (×18). *C*, thin, continuous stroma of *Epichloe typhina*, forming a film covering the leaf sheath of a grass (cocksfoot), the perithecia being completely sunken (see also Figs. 223, 238, 341)

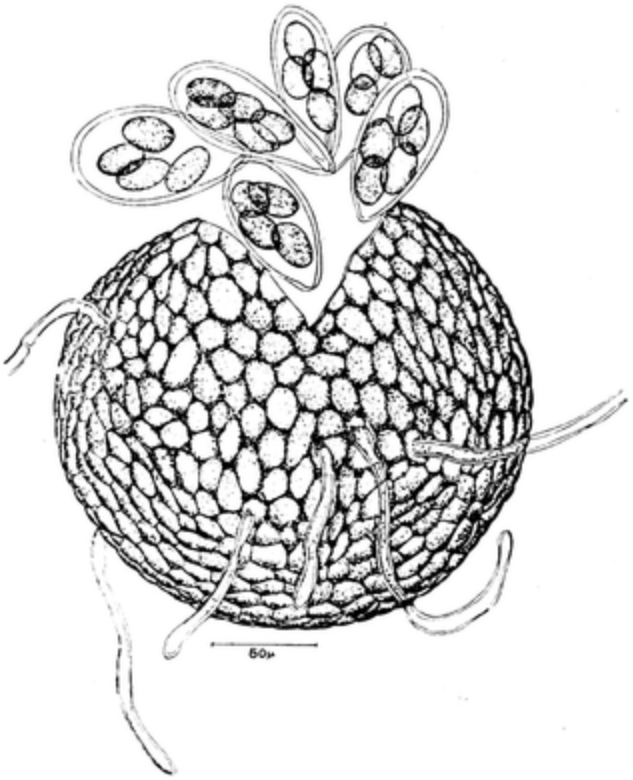


FIG. 43.—The cleistothecium or cleistocarp. The spherical fructification of *Erysiphe polygoni*, from *Lathyrus communis*. It contains several asci and opens by an irregular slit to set free the spores; note the simple appendages confined to the lower half of the body. The fructification was slightly crushed to cause the extrusion of the oval asci in which about four or five ascospores are usually developed

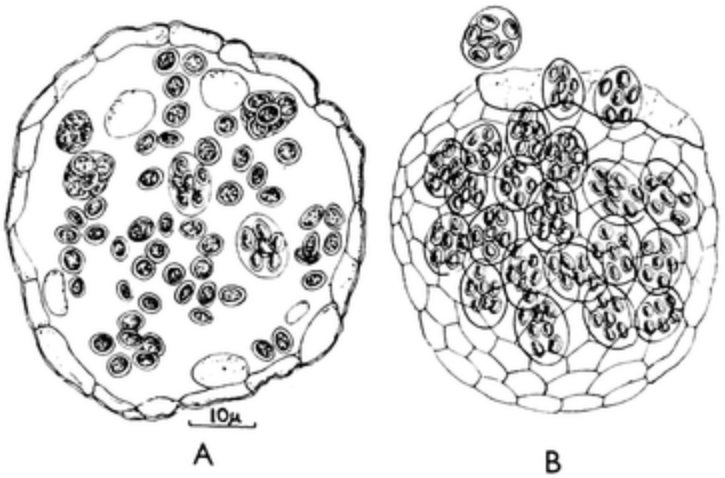


FIG. 44.—The cleistocarp of *Eurotium* (*Aspergillus*). The fructification is spherical and thin-walled; it opens irregularly, or by a general decay of the wall to release the spores; the oval asci contain from four to eight ascospores; some sterile fungal parenchyma cells still remain intermixed with the asci or spores in *A*, showing the fructification in section. *B*, a fructification in surface view

or immersed in it, e.g. *Epichloe*, *Claviceps* (Fig. 42, A, C), and opening to the surface by the narrow neck through which the spores are expelled when ripe. Sometimes they are formed close together but free from each other as in *Nectria* and *Ophiobolus* (Fig. 196), sometimes in groups immersed in a common stroma as in *Claviceps* and *Epichloe*, and, as in these two types, the stroma has in some cases become elevated or vertically elongated so as to raise the spore receptacles above the level of the substratum.



FIG. 45.—Complete section across the cap or pileus of a small sporophore of *Coprinus* showing the radiating gills covered with the blackened hymenium (× about 12). When the spores are ripe the gills become deliquescent and disappear in the form of inky drops; note that the spores mature in succession, and deliquescence proceeds from the margin of the pileus towards the centre, or stipe

In most compound sporophores, the spores are not produced on all parts of the organs. In the mushrooms, for instance, they are formed only on the surface of the gills found on the under side of the expanded 'cap' (Fig. 45). To the fertile part of the sporophore, i.e. that part which actually bears spores, the name 'hymenium' is given (Fig. 46).

In many cases, where the spore-bearing hyphae are so numerous as to form definite layers or a hymenium, certain sterile hyphae usually of characteristic form, occur intermingled with the fertile ones. These are frequently known as 'paraphyses' and are especially common in the Ascomycetes and Basidiomycetes (Figs. 41, 47). The basidia of the Hymenomycetes are often interspersed with sterile cells or 'cystidia' (Fig. 48).

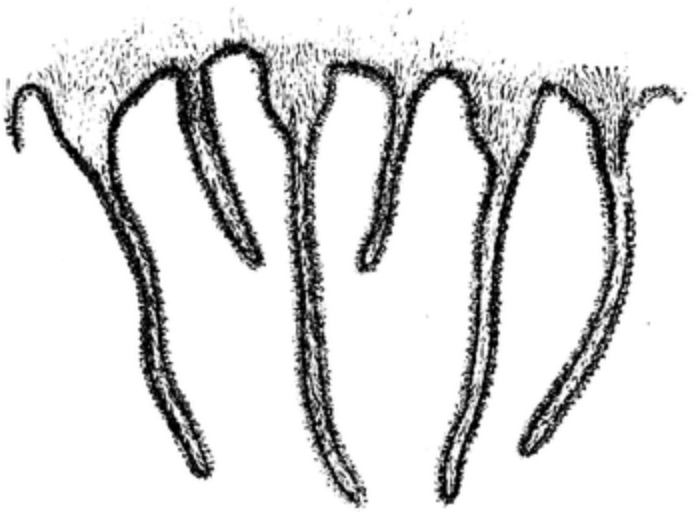


FIG. 46.—The typical Basidiomycete sporophyte. Portion of a section across the cap of a sporophore of *Coprinus* showing the gills covered by a continuous hymenium of basidia with dark-coloured basidiospores (diagrammatic)

From the above account of the fungus body, it will be seen that an ordinary fungus consists of a vegetative part, usually formed of thread-like hyphae buried in the substratum, and of a reproductive part which is sometimes not distinct from the vegetative except in bearing spores, but is more usually modified to form a simple or compound sporophore. As the production of spores provides for a new

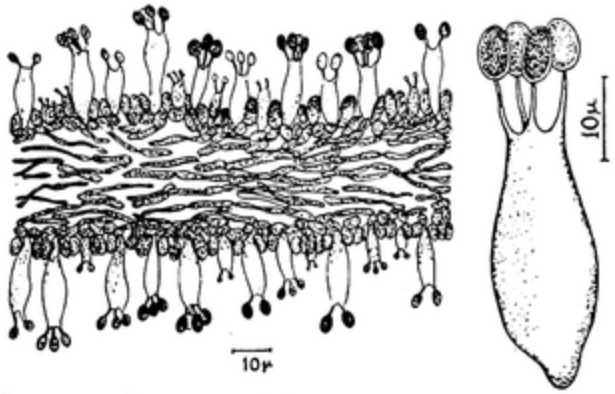


FIG. 47.—Structure of the gill-hymenophore. Portion of a gill of the previous figure showing the central, loose medulla (trama), a layer of small cells (sub-hymenium) supporting the continuous hymenium consisting of protruding basidia at all stages of maturity, furnished with basidiospores (ripe spores dark); some of the smaller, intervening cells amongst the basidia are sterile paraphyses; right, an enlarged basidium with four basidiospores borne on sterigmata

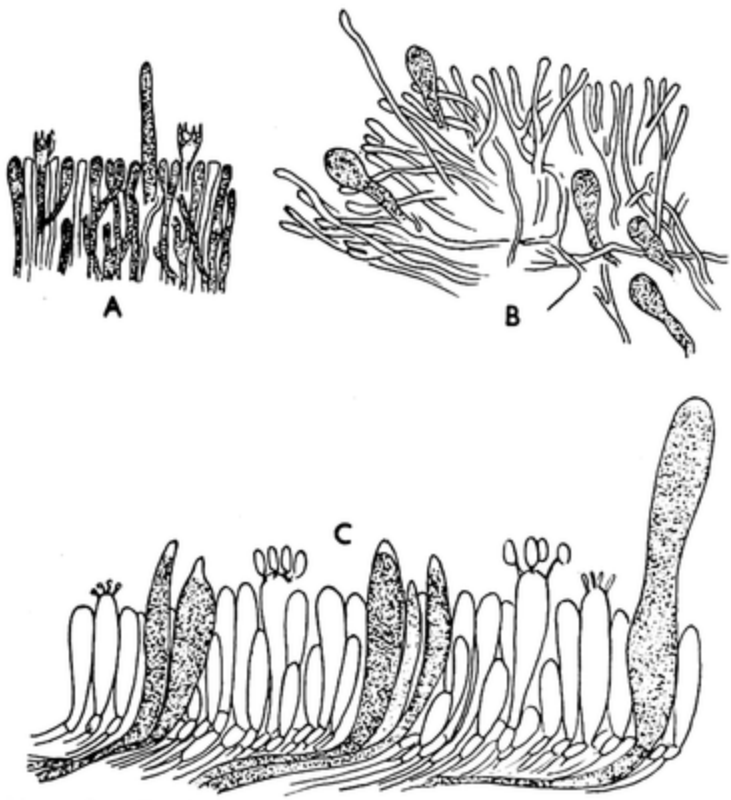


FIG. 48.—Cystidia. *A*, portion of the hymenium of *Stereum purpureum* showing two basidia with sterigmata, colourless hyphae, and a single projecting cystidium. *B*, section through the fructification of same, showing vesicular cells in the growing region (see also Fig. 88) (× 330) (after Exell, *Trans. Brit. Myc. Soc.*). *C*, portion of a hymenial layer of *Polyporus schweinitzii*, showing basidia, basidiospores, paraphyses, and cystidia (shaded) (after Hiley, *Fungal Diseases of the Common Larch*, by permission of Oxford Clarendon Press)

generation of the fungus, one may consider that the life-history of any species begins with the germination of the spore and ends with the development of new ripe spores, or, in the case of those fungi which have more than one kind of spore, with the development of the final or, as it is often termed, 'perfect' spore form, which is usually the result of a sexual process or its equivalent.

Formation of Spores

The spores of fungi present a wonderful range of variation, there being hundreds of types differing in size, shape, structure, and mode of formation (Figs. 49, 50). The one fungus may have several different kinds of spore during the course of its complete life-history. The vast majority differ sharply from the vegetative cells of the mycelium, but in any single species each kind of spore remains remarkably constant. Two main types may be distinguished: a 'perfect' form which is the result, immediate or delayed, of a sexual process (or its equivalent, for fungi have many ways of securing the object of sexuality — the bringing together of nuclei of different parentage); and an 'imperfect' or 'asexual' form to which the term 'sporangium,' or 'conidium' is usually applied. When the thallus which bears cells capable of sexual fusion is distinct from that carrying asexual spores, the former is termed the 'gametophyte', and the latter, the 'sporophyte'. The best

example of this alternation of generations amongst the fungi is found in the genus *Allomyces* (Fig. 51), but it is much less clear cut in fungi than in such plants as mosses and ferns [16].

Sexual Reproduction

The perfect form of fructification differs widely in the three main groups of fungi, Phycomycetes, Ascomycetes and Basidiomycetes. In the Phycomycetes it

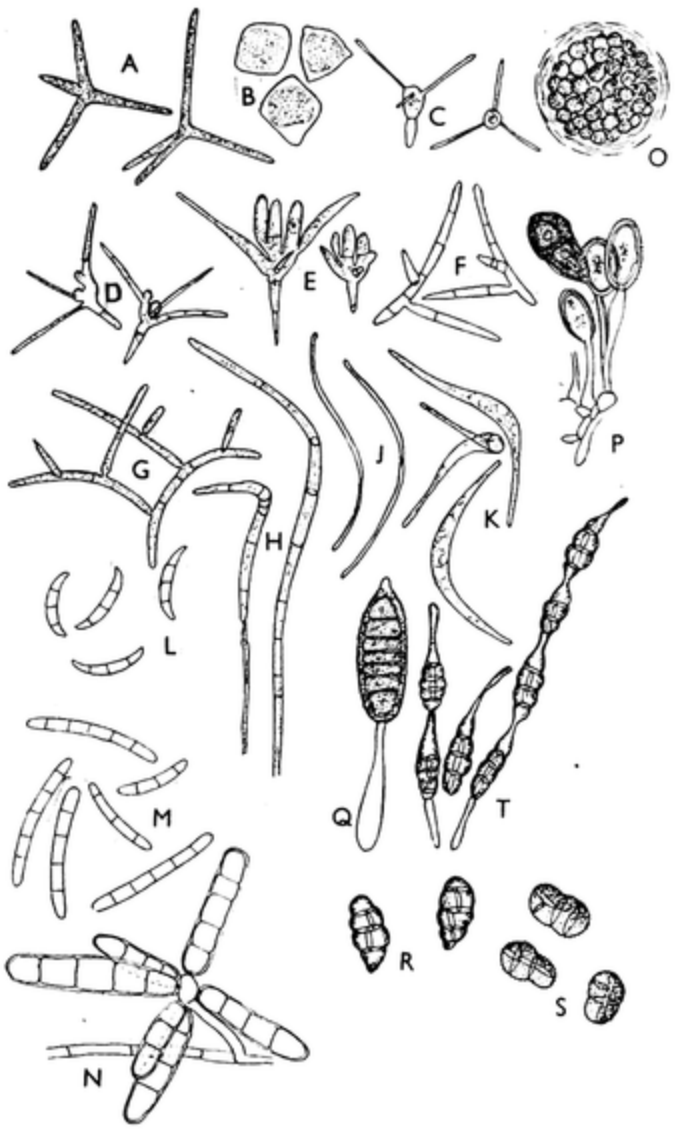


FIG. 49.—Variety of spores in fungi. *A–K*, spores of aquatic Hyphomycetes, on decaying alder leaves. *A*, *Lemonniera aquatica*. *B*, *Margaritispora aquatica*, showing glycogen contents (× 525). *C*, *Clavariopsis aquatica* (one on the left × 200, one on the right × 120). *D*, *Tetracladium marchalianum* (× 200). *E*, *Tetracladium setigerum* (× 300). *F*, *Tricladium splendens* (one on the left × 190, one on the right × 150). *G*, *Varicosporium elodeae* (× 200). *H*, *Anguillospora longissima*, two aleuriospores; it is difficult to say where the conidiophore stops and the conidium (aleuriospore) begins (× 220). *J*, *Flagellospora curvula* (× 210). *K*, *Lunulospora curvula* (× 190) (all after Ingold, *Trans. Brit. Myc. Soc.*). *L*, conidia of a species of *Fusarium* from buds of apple (after Salmon). *M*, conidia of *Nectria galligena* (× 230) (after Salmon). *N*, conidia of *Helminthosporium avenae* (after Dennis). *O*, spore ball of *Sorosporium saponariae* (× 170) (from Engler & Prantl). *P*, three uredospores and a teleutospore of *Puccinia graminis* (after Sachs). *Q*, *Phragmidium subcorticatum*, a teleutospore (× 350) (after Engler & Prantl). *R*, two muriform spores of *Stemphylium piriforme* (after Saccardo). *S*, *Macrosporium commune* (after Saccardo). *T*, *Alternaria tenuis* (after Saccardo)

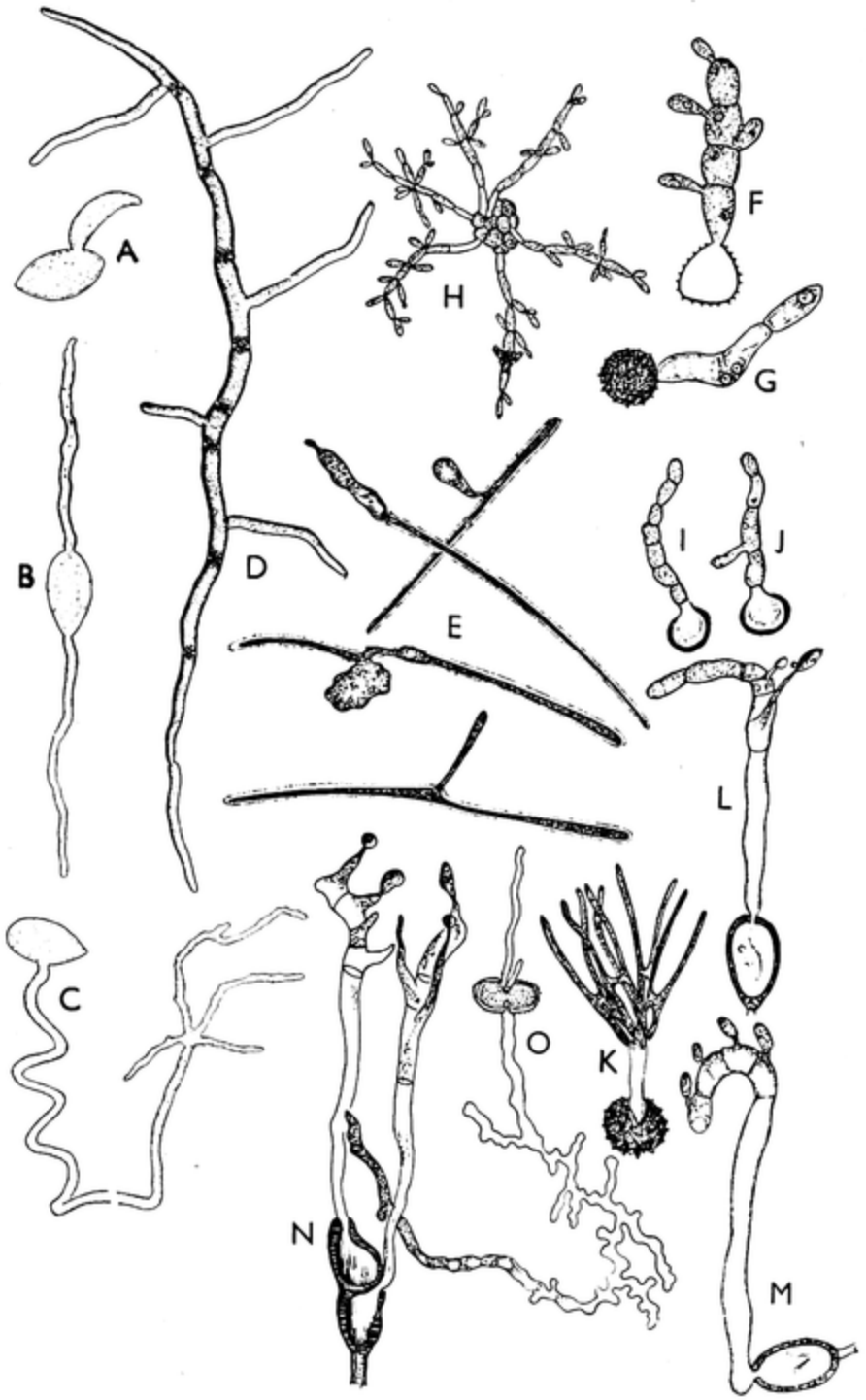


FIG. 50.—Spore germination. *A*, *B*, *C*, stages in germination of conidia of onion mildew (*Peronospora destructor*) (× 200) (after Murphy & McKay, *Sci. Proc. Roy. Dub. Soc.*). *D*, germination of the filiform, septated ascospore of *Ophiobolus heterostrophus* (after Drechsler, *J. Agric. Res.*). *E*, three germinating ascospores of *Lophodermium pinastri*, showing delicate spore sheath; three spores show vesicles (after Jones, *Ann. Bot.*). *F*, germinating smut spore of *Ustilago scabiosae* producing a septated basidium (promycelium) bearing sporidia or basidiospores (after Harper). *G*, germinating smut spore of *Ustilago carbo* showing passage of nucleus from one cell to another (after Rawitscher). *H*, a germinating spore ball of *Tolyposporium junci* (after Brefeld). *I*, *J*, germinating smut spores of *Ustilago avenae* (after Western, *Phytopathology*). *K*, germinating spore of *Tilletia tritici* (after Brefeld). *L*, *M*, germinating mesospores of *Puccinia sonchi* (after Lamb, *Hedwigia*). *N*, germinating teleutospore of stem rust (*Puccinia graminis*) showing the two cells producing a septated promycelium with sterigmata and sporidia (after Sachs). *O*, germinating uredospore of same, the hyphae passing out through the germ pores (after Sachs)

is a thick-walled, well-defined cell, to which the general term ' zygote ' is applied in some of the more primitive forms (*Chytridiaceae*), while in the Oomycetes the zygote is termed an ' oospore ', and in the Zygomycetes, a ' zygospore '. A zygote is simply the end product of the fusion of two cells with sexually differentiated nuclei which also fuse. These cells are known as ' gametes ' and in some of the *Chytridiaceae* are motile ; they may be outwardly alike, so that a ' male ' is not to be distinguished from a ' female ' gamete (Fig. 250, *16*). An oospore is a cell resulting from the fertilisation of a larger female cell or ' oogonium ' by a smaller male cell, the ' antheridium ', both borne on hyphae of the mycelium (Fig. 52). A zygospore results from the union of the tips of two branches of the mycelium, often quite similar to one another (Figs. 53, 54). In all these cases fertilisation is completed, as usual, by the fusion of the nuclei from the two uniting gametes to form the zygote [28]. At some stage prior to fusion the nuclei undergo a ' reduction ' division, which results in the halving of the number of ' chromosomes ' (the bearers of the characters of the species) and gives rise to what is termed the ' haploid ' phase of the life-cycle. When two haploid nuclei come together in a cell, the dikaryophytic, dikaryo-, or dikaryotic phase is initiated ; fusion of the two haploid nuclei (sometimes long delayed) restores the original number of chromosomes, half of which are now of different origin from the other half, and the resulting nucleus is termed ' diploid '. The process directed to bringing together the two haploid nuclei in one cell is sometimes called ' diploidisation '.

In the Ascomycetes the perfect stage is the ' ascus ' (Fig. 55), a sac-like,

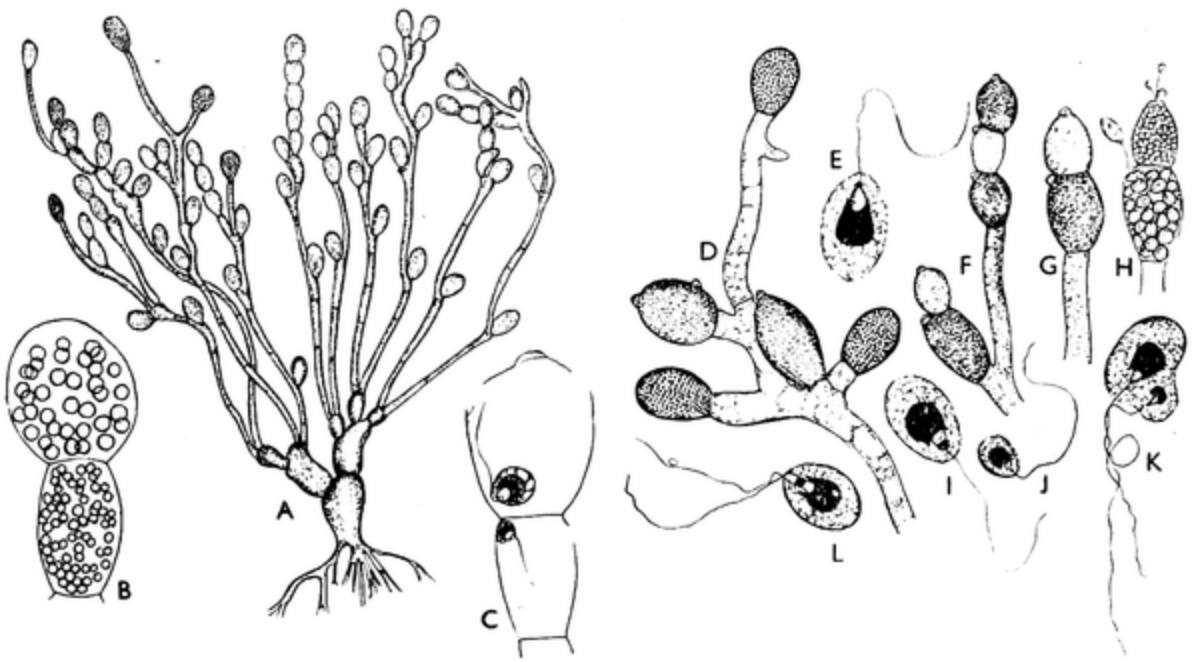


FIG. 51.—Alternation of generations in *Allomyces*. *A*, *Allomyces arbuscula*, the asexual thallus bearing sporangia (after Butler). *B*, *C*, upper cell, a female, lower cell, a male gametangium, from a sexual thallus of same (× 523) (after Hatch, *Ann. Bot.*). *D–L*, *Allomyces javanicus*. *D*, portion of an asexual thallus bearing zoosporangia. *E*, a zoospore. *F*, portion of a sexual thallus bearing the two kinds of gametangia. *G*, *H*, the sexual organs, the terminal cell a male, the penultimate cell a female, gametangium. *I*, the larger macrogamete. *J*, the smaller microgamete. *K*, early fusion of the gametes. *L*, complete fusion to form a zygote (after Kniep)

roundish or elongated cell containing ' ascospores ', which is sometimes immersed without orderly arrangement in a stroma or in meshes of the mycelium but usually lies side by side with other asci and, perhaps, paraphyses to form a definite hymenium. In some forms the ascus is the result of the fertilisation of a female cell, the ' ascogonium ', through a process or ' trichogyne ' which contacts an antheridium-like male cell (Fig. 56). Instead, however, of the two nuclei uniting as in the oogonium, they may divide several times and a male and a female pair pass into each of a group of cells (the ' ascogenous hyphae ') which grow out from the ascogonium ; from one or more of these cells the asci arise, each ascus still having a male and a female nucleus (Figs. 56, K-N). Fusion of the two nuclei occurs in the young ascus and the ascospores arise by the division of this sexual nucleus. Owing to their relative sizes the nuclei often have to lie in single file in the ascogenous hyphae, and an arrangement known as the ' crosier ' formation is formed by which the division of the nuclei brings together a pair

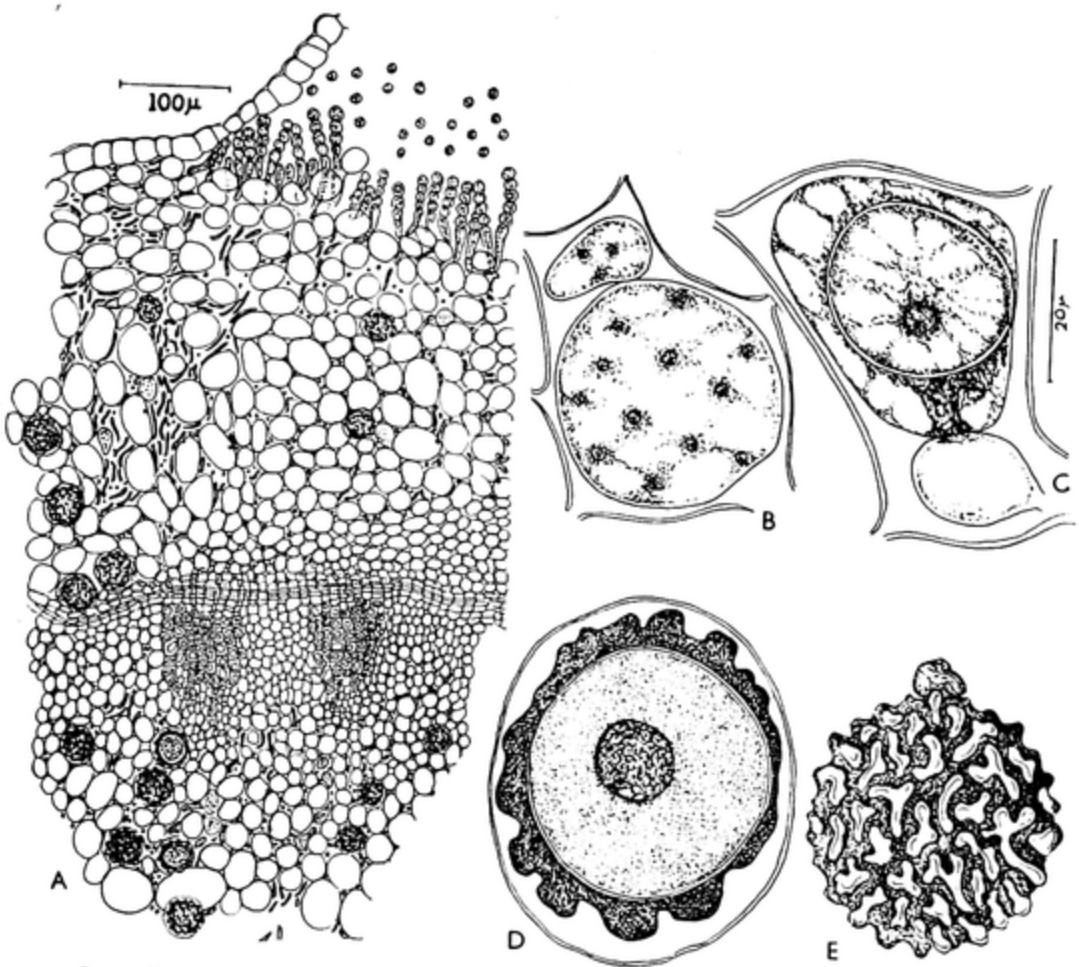


FIG. 52.—Sexuality in the Oomycetes. *Cystopus candidus*. *A*, portion of a transverse section of inflorescence axis of shepherd's purse showing a sporangial pustule breaking through the epidermis ; in the cortex and medulla note the intercellular mycelium (with haustoria) and sexual organs, oogonia and antheridia, and thick-walled oospores. *B*, a multinucleate oogonium (below) and antheridium (above) in an intercellular space. *C*, fertilisation of the uninucleate oogonium, the young oospore surrounded by vacuolated cytoplasm and remaining degenerated female nuclei ; note the dark-shaded remains of the fertilisation-tube and the empty antheridium below. *D*, the fully matured, thick-walled oospore within the oogonium, in section. *E*, the ridged, tuberculated epispore of the oospore

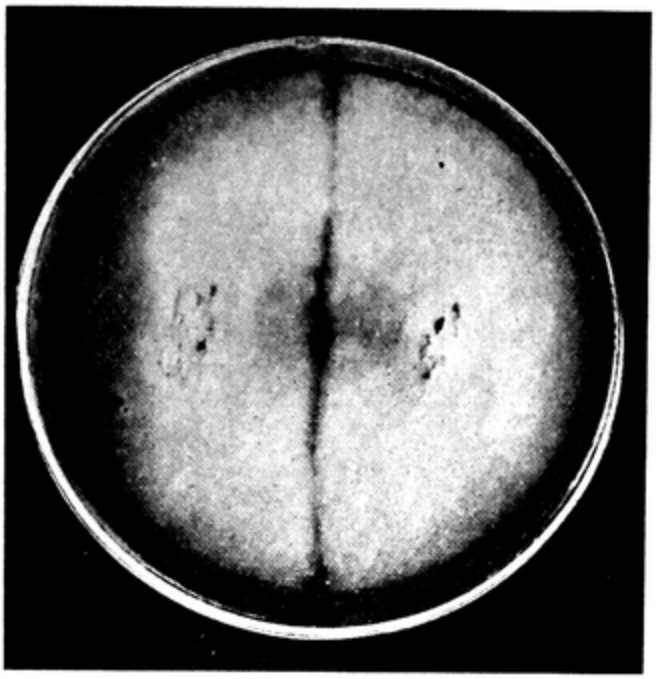


FIG. 53.—Sexuality in a heterothallic zygomycete. A colony of *Mucor hiemalis* derived from + and − strains, showing a zone of dark-coloured zygospores at the line of junction between mycelia of opposite signs; a culture on weak potato agar at 18° C. (× ½)

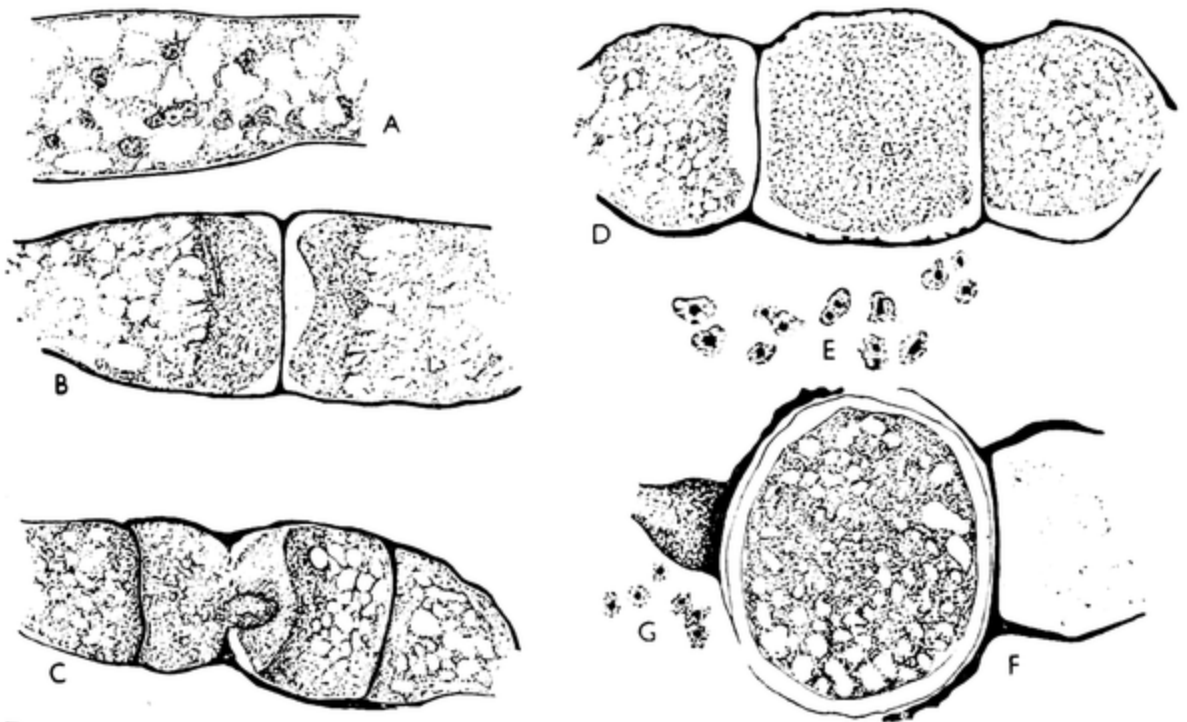


FIG. 54.—Sexuality in a homothallic zygomycete (*Sporodinia grandis*). *A*, a portion of the coenocytic mycelium. *B*, fusion of two hyphal tips (gametophores). *C*, the cutting-off of two terminal gametangia and the fusion of their contents following the breakdown of the intervening wall; the contents of the smaller gametangium appear to be penetrating the contents of the larger gametangium. *D*, the young zygospore at the time of nuclear fusions. *E*, a few of the nuclei in the zygospore showing various stages of fusion. *F*, a nearly mature zygospore showing increase in size, cytoplasm vacuolated and oily. *G*, nuclei of the zygospore (after Keene, *Ann. Bot.*)

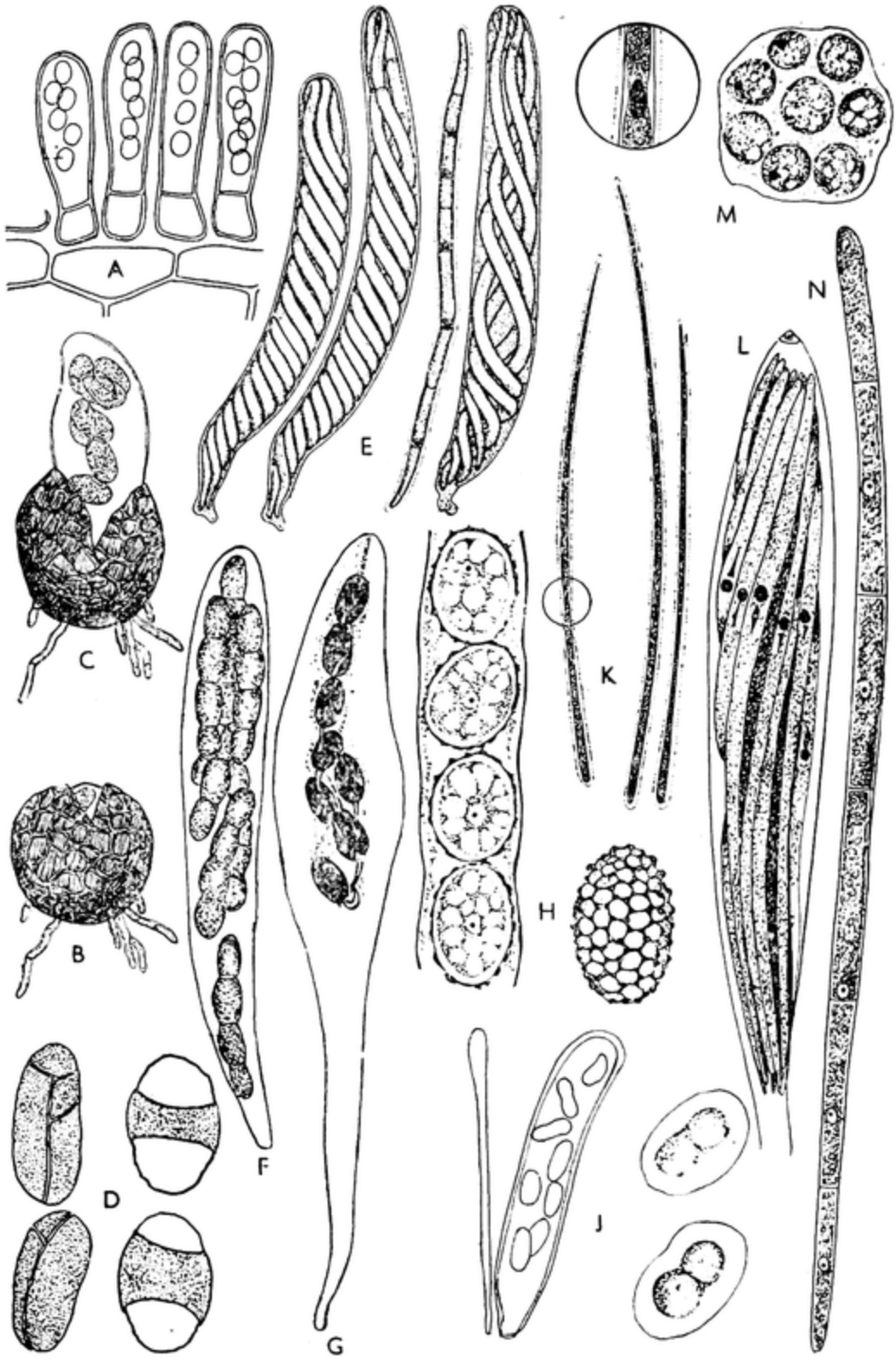


FIG. 55.—Morphology of the ascus and ascospores. *A*, *Exoascus minor*, four asci formed under the cuticle of a cherry leaf; note the sterile stalk cell (after Salmon). *B*, *C*, cleistocarps of *Sphaerotheca mors-uvae* showing liberation of the single ascus with ascospores (after Salmon). *D*, four ascospores of *Dasyobolus immersus*: the two on the left are normal purple spores, the two on the right have a purple band (×270) (after Gwynne-Vaughan & Williamson, *Trans. Brit. Myc. Soc.*). *E*, three asci from a perithecium of *Ophiobolus heterostrophus* showing the ascospores spirally coiled while within the ascus; in between, a released ascospore with a mucous sheath (×314) (after Drechsler, *J. Agric. Res.*). *F*, an ascus of *Sporomia intermedia* showing four-celled ascospores. *G*, an ascus of *Podospora curvula*, the eight ascospores more or less united by mucilage strands, just prior to dehiscence (after Ingold, *New Phytologist*).

of nuclei, male and female, in the penultimate cell, from which the ascus arises. The clamp connection of the Hymenomycetes, to be described below, serves the same purpose [34]. There is still some disagreement as to the number, location and significance of the nuclear fusions preceding the formation of ascospores, and the details of the sexual process are as yet uncertain in some forms. In some cases a fusion of the male and female nuclei has been observed in the ascogonium, in addition to the fusion in the young ascus, and two reduction divisions following this double fusion have been described.

In the unicellular yeasts the single cell may become converted as a whole into an ascus (Fig. 57), the formation of which may be preceded by union with another cell by means of a short bridge across which the nucleus of one cell passes to fuse with that of the other. In cultures of these fungi some cells are haploid, some diploid.

The great bulk of the Ascomycetes are contained in two main groups, the Pyrenomycetes in which the sporophore is a flask-shaped perithecium (Fig. 38), often immersed in the tissues or in a stroma and opening by a narrow neck, and the Discomycetes in which the hymenium lines the inner surface of a cup-shaped sporophore or apothecium (Fig. 41). In the Pyrenomycetes, which are the most numerous and diverse group of Ascomycetes, the hymenium lines the inner surface of the perithecium and the spores may be extruded while they are still enclosed in their asci or after they have been set free into the cavity of the sporophore. In the heterogeneous group, the Plectomycetes, the powdery mildews (Erysiphaceae) are furnished with a characteristic type of fructification, the cleistocarp or cleistothecium, which is not provided with an opening and the ascospores are only liberated after the protective wall ruptures or decays (Figs. 43, 44). The Exoascaceae are included in this group although (as the name implies) the asci are not enclosed in a special body at all, but — like *Taphrina* (*Exoascus*) *deformans*, the cause of peach-leaf curl (p. 770) — are exposed on the surface of the host once they break through the cuticle at maturity (Fig. 143). The typical number of spores in an ascus is eight but there may be fewer (in the yeasts, often only one to four) or more, when the number is increased by division of the original nuclei, usually to a higher power of 2, as 16, 32, 64, 128, but sometimes irregularly from suppression of one or more spores.

In many of the higher Ascomycetes (Pyrenomycetes and Discomycetes) the ascogonium which eventually gives rise to the ascogenous hyphae appears often to be fertilised by microconidia (oidia or spermatia) (Fig. 58). The oidia may be borne laterally on small awl-shaped or verticillate sporophores and in certain fungi — as in the genus *Sclerotinia* — do not function as ordinary spores as they do not give rise to a mycelium. When set free in *Sclerotinia* they adhere to cells either in

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H, *Lachnea scutellata*, four nearly mature ascospores showing vacuolated cytoplasm; the pattern on the epispore corresponds to the position of the vacuoles: on the right, a spore in surface view (after Gwynne-Vaughan, *Ann. Bot.*). *J*, ascus, paraphysis, and ascospores of *Rhabdocline pseudotsugae* (after M. and M. J. F. Wilson, *Trans. Roy. Scot. Arbor. Soc.*). *K*, three ascospores of *Lophodermium pinastri*; inset, the spore nucleus (after Jones, *Ann. Bot.*). *L*, *M*, *N*, *Ophiobolus graminis*. *L*, young ascus showing the eight filiform spores spirally twisted and, so far, uninucleate. *M*, cross-section of an ascus. *N*, a mature, septated ascospore (after Jones, *Ann. Bot.*)

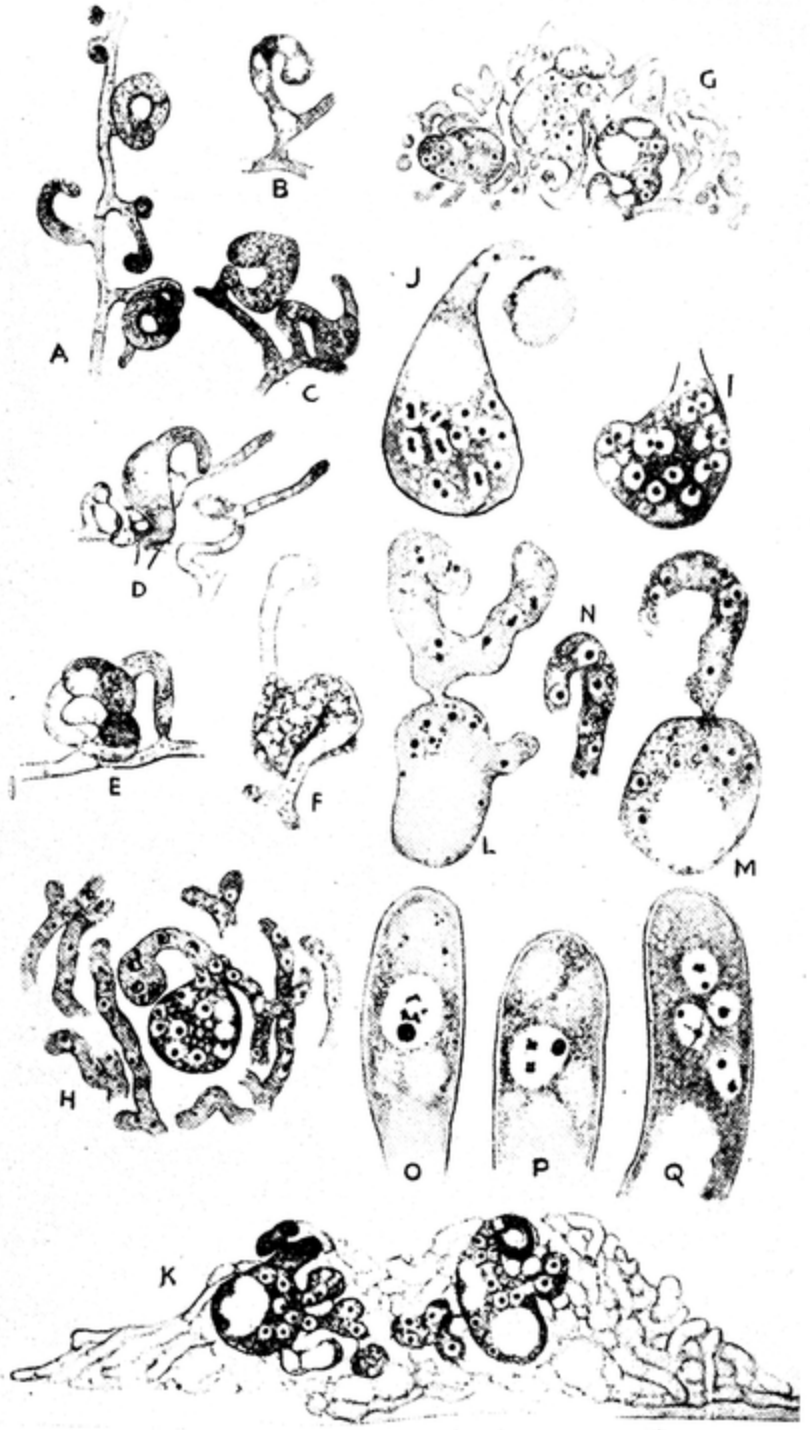


FIG. 56.—Sexuality in the Ascomycetes. *Ascophanus aurora*. *A*, hypha with developing sexual branches; the branched filament half-way down on the right will probably form an antheridium, the other branches will form oogonia. *B*, a male branch ending in an antheridium. *C*, two female branches with developing oogonia, from one of which a trichogyne is being put out. *D*, a young oogonium with trichogyne and male branch; the latter is shown again to the right. *E*, a pair of sexual branches, just before union of trichogyne and antheridium. *F*, a twisted oogonium after fertilisation. *G*, a group of three oogonia (all × approx. 930). *H*, passage of male nuclei from antheridium into oogonium (× 2600). *I*, pairing of nuclei in the oogonium. *J*, six fusion pairs, and some inactive nuclei (× approx. 1500). *K*, two oogonia with ascogenous hyphae (× 930). *L*, *M*, *N*, further development in the ascogenous hyphae. *O*, *P*, the fusion nucleus in the young ascus. *Q*, third division in the young ascus (× approx. 1500) (after Gwynne-Vaughan & Williamson, *Ann. Bot.*)



FIG. 57.—Ascus formation in the yeasts (Saccharomycetes). Budding cells of yeast (*Saccharomyces cervisae*); below, stages in segmentation of the cell contents to form, usually, four ascospores in the improvised cell which becomes an ascus (× 700) (after Hansen)



FIG. 58.—The probable function of microspores (microconidia, oidia, and spermatia) in the Ascomycetes, as shown in *Sclerotinia convoluta*; the genetic connection between the *Botrytis* stage and the perfect *Sclerotinia* stage. *A*, asci, ascospores, and paraphyses from the apothecium of *S. convoluta* shown in vertical section in *E*. *B*, a germinating ascospore producing microconidia. *C*, a branched conidiophore with immature macroconidia. *D*, mature macroconidia. *F*, a group of apothecia in association with bunches of the conidiophores of the *Botrytis* stage. *G*, a group of the stalked apothecia (after Drayton, *Mycologia*)

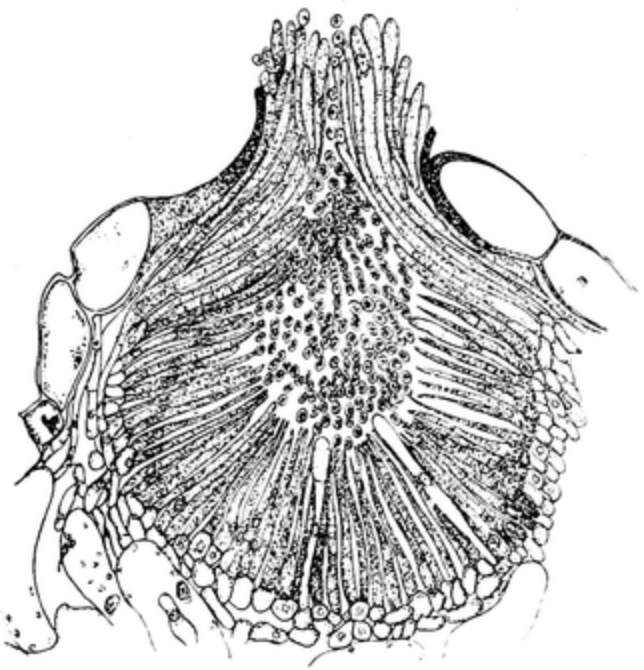


FIG. 59.—The spermagonium. A typical spermagonium of the rusts. The flask-shaped organ is lined with narrow spermatial hyphae which abstrict in great numbers the minute, oval, or round spermatia (male cells); some of the hyphae with blunt tips issuing at the ostiole may be paraphyses; the few club-shaped hyphae with sparse contents interspersed among the spermatial hyphae are ' buffer ' cells (× 480) (after Ruth Allen, *J. Agric. Res.*)

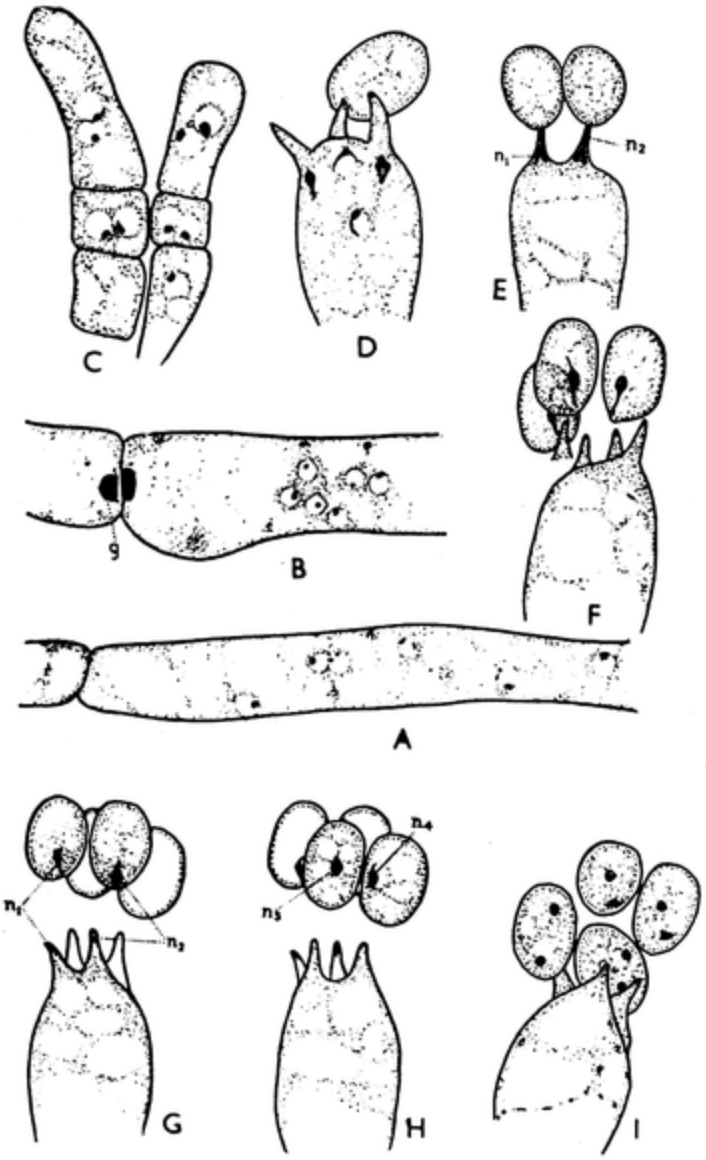


FIG. 60.—Sexuality in the higher Basidiomycetes (Eumycetes). Development of the four-spored basidium and spores in the mushroom. *A*, multinucleate portion of a mycelial strand connecting the fructification to the subterranean mycelium. *B*, part of a cell from undifferentiated fructification, showing six nuclei, with granules near the septum. *C*, binucleate cells of the subhymenium, the terminal cells being young basidia. *D–I*, stages in the development of a basidium, with four sterigmata bearing basidiospores which eventually become binucleate (all approx. × 2000) (after Colson, *Ann. Bot.*)

special receptive bodies raised above the level of the stroma, or contained in the sclerotia which are characteristic of these fungi. Fusion presumably results in the transference of a nucleus from a hypha in one mycelium to another, and apothecial development follows[22]. Spermatia borne in special spermagonia have long been known to occur in the life-cycle of many rusts (Fig. 59) Pyrenomycetes and Discomycetes (Fig. 29, A, D) and to have no apparent function as spores capable of reproducing the species. In a few cases they have also been found fusing with cells of the primordia of perithecia, and though the evidence is largely circumstantial, it is probable that both oidia and spermatia ordinarily function as haploid ' male ' cells.

Turning to the Basidiomycetes, a still greater complexity is found in the processes which seem to serve the ends of a sexual union. In place of the ascus of the Ascomycetes, the ' basidium ' marks the perfect stage in the life-cycle. The term ' promycelium ' is also applied to the basidium in this group. It is a round, club-shaped or baton-shaped body in which the gametic nuclei fuse. Subsequently it gives out a group (typically four) of spores (' sporidia ' or ' basidiospores ') on short outgrowths (' sterigmata ') from its surface (Fig. 60). In the wide sense, the Basidiomycetes include the smuts and rusts as well as the mushrooms and bracket fungi, and the different groups vary considerably in the details of their perfect stages since the sporidia of the two former groups do not arise immediately from the zygote.

The sporidia of the smuts are borne typically on a promycelium which resembles a stout germ-tube arising from the smut spore (Fig. 61). In the majority of the

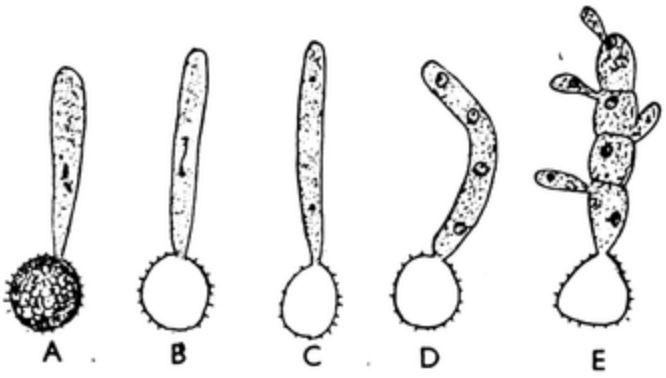


FIG. 61.—Sexuality in the smut fungi (Ustilaginales). Stages in the germination of a spore of *Ustilago scabiosae*, the germ-tube becoming a four-celled promycelium or basidium, each cell, after nuclear division, producing one or more sporidia or basidiospores (after Harper)

species the promycelium is divided by transverse septa into four cells, each of which contains a single nucleus (haploid). Usually a single sporidium or hyphal branch is produced from each promycelial cell, but sometimes two or more are developed; the promycelial nuclei do not pass out into the sporidia but divide, and each of the daughter nuclei enters a sporidium. In some cases this nuclear division may be repeated several times so that crops of sporidia arise directly from a promycelium. Sometimes the four cells of the promycelium fuse in pairs by means of small buckle-like outgrowths through which a haploid nucleus passes from one cell to the other (Fig. 62). Thus the binucleate, dikaryotic condition in the smuts may arise from the union of two sporidia or hyphal branches (germ-tubes), or from the union of two promycelial cells. In the genus *Tilletia*, the nuclei usually pass into the sporidia or hyphal branches, none remaining in the promycelium which in this type usually remains unseptated (Fig. 63). In *Tilletia* the primary sporidia formed at the tip of the promycelium become binulceate by fusion in pairs; they give rise to fine hyphae on which are borne secondary sporidia which are usually also binucleate but on germination give uninucleate and presumably haploid sporidia. These secondary and tertiary sporidia are considered by some to be the true basidiospores (Fig. 63 c) [9]. The smuts are parasites, and in a good many species it is the binucleate germ-tubes (' fusion hyphae ') which enter the host plant, the cells of the mycelium in the host usually again appearing binucleate just before the smut spores form, when the two nuclei fuse, transforming the smut spores into diploid zygotes. In some species the fusion hyphae are formed within the tissues of the host and there is some uncertainty as to the details of the change from the haploid to the diploid phase in these. The completion of the sexual process from the time the two nuclei are brought together in a cell (' dikaryon ') until their fusion in the young spore may take several months. The sporidial stage can be readily grown in culture, and experiments with cultures derived from single sporidia have established that fusions will only occur between certain pairs, and that the four promycelial cells usually represent two ' males ' and two ' females ' so that a simple form of heterothallism (see below) exists. Since in some species more than one pair have been found capable of fusing, it is necessary to recognise the existence of more than two kinds of sexually functionable gametes in fungi or else to use another terminology. Thus, some speak of the ' A ', ' B ', ' C ', etc., ' genders ' instead of sexes, or of compatible and incompatible groups. At least nine such groups appear to be recognisable in maize smut [12, 52].

It has long been known that the rusts have a dikaryophytic phase in which two nuclei occur in each cell. This commences as a rule somewhere in the

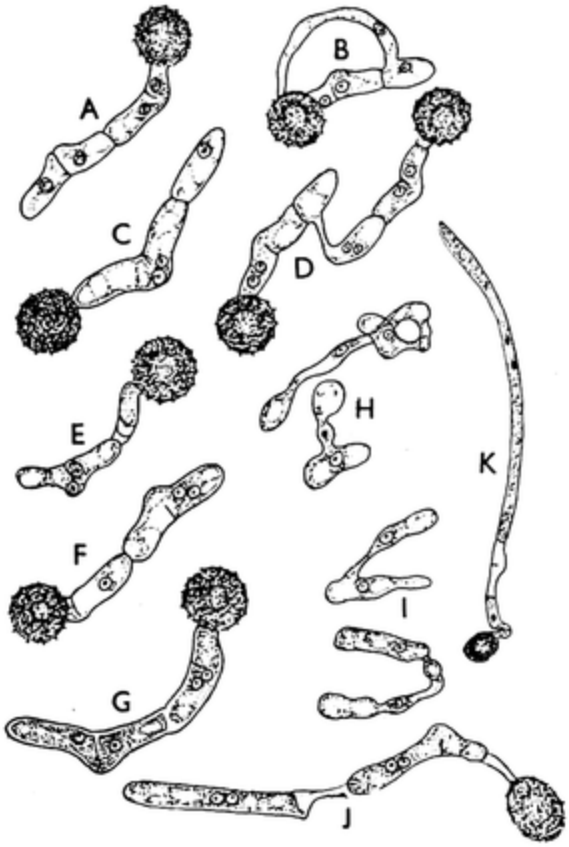


FIG. 62.—Sexuality in the smut fungi (*continued*). *A–K*, stages in the germination of the spore of *Ustilago carbo*, without the formation of sporidia. *A*, cross-wall near the spore breaking down to admit passage of nucleus to the next cell. *B*, a 'buckle-joint' hypha sent out from the terminal cell of a basidium to receive a nucleus from the spore, the terminal cell thus becoming binucleate (dikaryophytic). *C*, *E*, *F*, *G*, stages similar to *A*, showing binucleate cells. *D*, fusion of cells from different promycelia. *H*, *I*, *J*, various fusions of promycelia, or of promycelial cells. *K*, a spore germinating forthwith to form a germ-tube, the long cell becoming binucleate (× 400) (after Rawitscher)

primordia of the aecidia and terminates in the ripe teleutospore (Fig. 64) where the two nuclei fuse to form the zygote.

Rusts are amongst the most pleomorphic of the fungi, the full life-cycle often possessing no less than five spore stages. The basidium (promycelium) arises from the germination of the teleutospore — a resistant spore-stage homologous with the smut spore — and the haploid phase is initiated during the process of nuclear division in the promycelium, and which normally results in the production of four sporidia (basidiospores), one from each of the four cells into which the promycelium becomes divided by transverse septa (Fig. 64). As in the smuts the four sporidia belong to two 'sexes'. The haploid sporidia infect the host (all the rusts are parasitic) and cause the development of immersed flask-shaped spermagonia containing masses of tiny spermatia which are extruded in a drop of sweet 'honey dew' or nectar through the neck or pore to the surface of the plant. Here they are visited by flies or other insects which carry them to the openings of other spermagonia where they adhere to certain emergent hyphae (Figs. 65 c, 66, 185) in or at the margin of the spermagonium [14]. The next stage is difficult to follow and seems to differ in different species. In some, at least, the nucleus of the spermatium appears to be able to pass back through the hypha with which it fused to reach the deeper levels of the haploid mycelium developed from the sporidium (Fig. 66 B) [10, 52]. The general outcome is the establishment of a binucleate (dikaryophytic) condition of certain haploid cells at the base of the future sporophore which develops in the vicinity of the spermagonia, often on the opposite side of the leaf. This is the aecidium, bearing binucleate aecidiospores in chains (Fig. 67). From the dispersed aecidiospores arises a parasitic mycelium, the paired nuclei in the cells of which are maintained by conjugate division. It is this dikaryophytic mycelium that gives rise to binucleate uredospores, often in repeated crops of uredosori in such quantity that the yellowish or rusty spores colour the surface of the soil under the infected plants. Ultimately teleutosori replace the uredosori and the paired nuclei at last

fuse in the mature teleutospores (see life-history of *Puccinia graminis*, p. 347). The rusts frequently overwinter in the teleuto-stage, producing their promycelia (basidia) when the teleutospores germinate in the spring. In a considerable number of species the haploid sporidia from the promycelium are unable to infect the species of host plant on which the uredo-teleutospore stage develops, but attack some other species, often far removed systematically from the first, and cause the development in it of a haploid vegetative mycelium leading to the aecidial stage, while the binucleate aecidiospores carry the fungus back to the original host. This, which is known as ' heteroecism ', will be further discussed in a later section (p. 87) and seems to be a step in advance along the path already entered by the smuts, where the haploid germ-tube may be devoid of parasitic capacity.

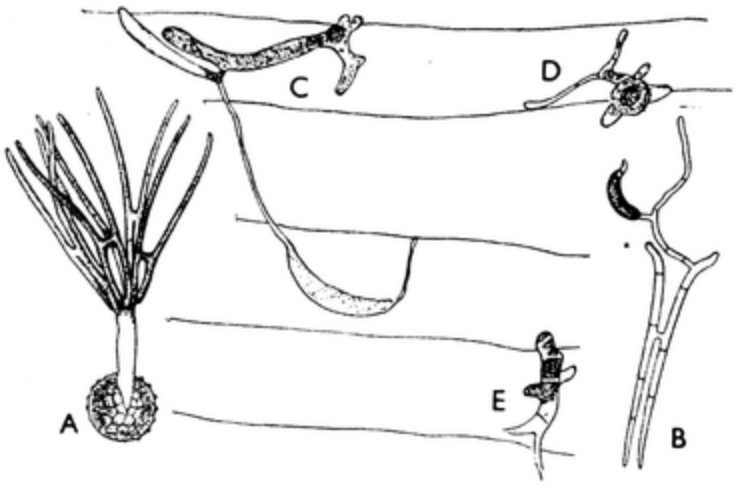


FIG. 63.—Sexuality in the bunt of wheat (*Tilletia caries*). *A*, the germinating spore, showing a stout basidium or promycelium bearing, on short sterigmata, long ' primary sporidia ' which fuse in pairs in H-fashion. *B*, a pair of primary sporidia germinating after such a fusion, to produce a thin hypha which is giving rise to a ' secondary sporidium ', this probably being a true basidiospore (after Brefeld). *C*, fusion of thin germ-tubes arising from secondary basidiospores to form a thick, dense ' fusion hypha ', prior to infection. *D*, *E*, fusion hyphae arising from fused, thin haploid hyphae from which the basidiospores have become detached. *C*, *D*, *E*, on wheat coleoptile (after Churchward, *Ann. App. Biol.*)

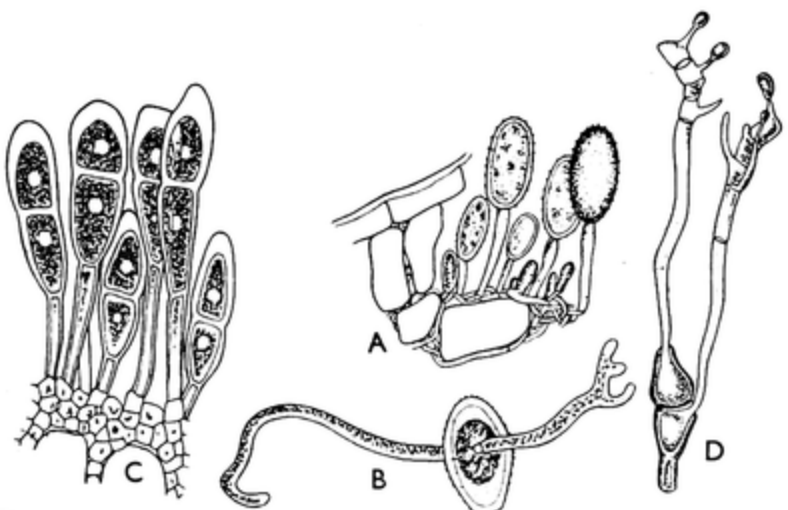


FIG. 64.—The spore cycle of *Puccinia graminis* on the cereal host. *A*, part of a uredosorus (after Duggar). *B*, germinating uredospore. *C*, part of a teleutosorus showing mature teleutospores in the cells of which the long-deferred fusion of paired nuclei takes place (after Eriksson & Henning). *D*, germination of the teleutospore, resulting in a haploid condition of the sporidia which, in turn, infect the barberry (after Tavel, from de Bary)

In the Basidiomycetes proper the conception of sexuality must be further stretched if it is to cover all the known cases. Not only are the four sporidia or basidiospores that are normally borne on four sterigmata on the free end of the basidium often only able to form thalli that can fuse in pairs which are mutually inter-sterile, but isolation of sporidia from other sporophores or other regions may go on adding almost indefinitely to the number of sexually functional gametes found. In these

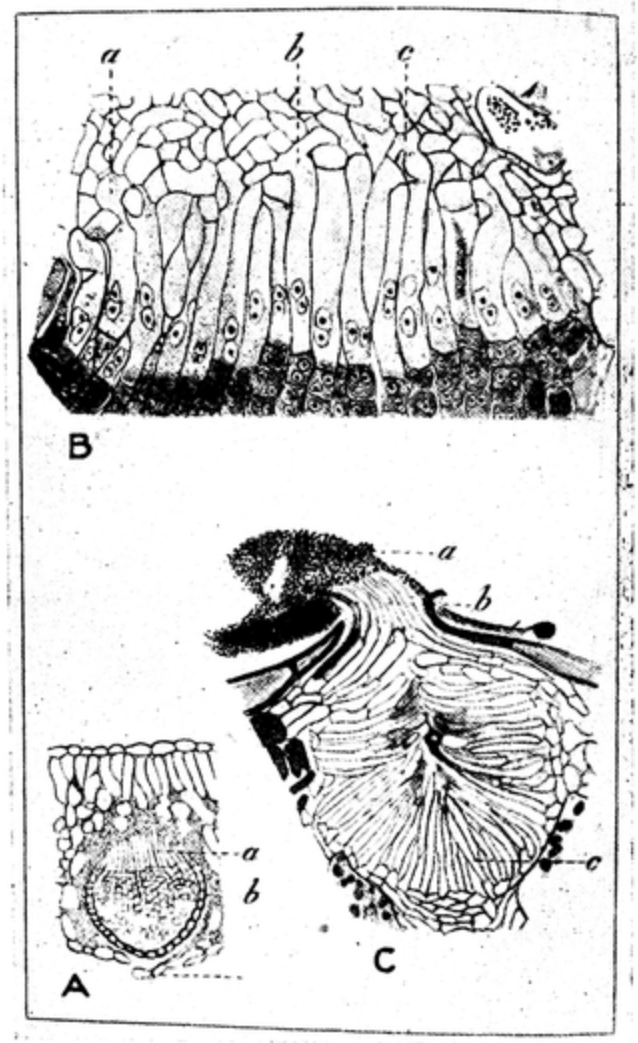


FIG. 65.—Sexuality in the rust fungi (Uredineae). The origin of the dikaryophytic phase in *Puccinia graminis*. *A*, a fertile aecidium in leaf of barberry still covered by its peridium *b*, and showing the 'fertile cells' *a*, at the base. *B*, enlarged portion showing the fertile cells in *A*; most of these cells are binucleate (dikaryophytic); at *a*, *b*, and *c*, probable places where passage of nuclei into the, at first, uninucleate cells, took place. *C*, a spermagonium, with spermatia embedded in matrix ('nectar') at *a*; paraphyses at *b* which probably pass back as at *c* (after Ruth Allen, *J. Agric. Res.*)

fungi the haploid spore stage may be represented by uninucleate oidia borne laterally on the mycelium (Fig. 68), which may fuse together (Fig. 69) or with the end cells of hyphae composed of uninucleate cells. The result is a binucleate cell and the binucleate condition in subsequently developed cells of the terminally growing hypha is maintained by a curious contrivance called a 'clamp connection' (9). This, whose presence marks the dikaryophytic phase of the mycelium, develops around the septum formed when the end cell of the hypha divides (Fig. 70). The clamp begins as a protuberance in the side of the end cell just above where the new septum will form. Both nuclei move to this point and one enters the protuberance. Both then divide while the septum separating the parent cell into two develops between the pair arising from the nucleus which did not migrate into the protuberance. The clamp completes its development by uniting at its tip with the cell below the new septum, and a nucleus from the pair formed in it passes to each of the two cells of the hypha. Thus the binucleate cells always contain a nucleus from each of their two components. Eventually the two fuse in the basidium and the perfect stage is achieved. Sometimes the dikaryophytic mycelium arises from the fusion of haploid mycelia derived from oidia of opposite sex (4), as well as from haploid mycelia of opposite sex derived from the germination of the basidiospores. These haploid mycelia are without clamp connections and are sometimes termed 'primary' mycelia, as distinct from the 'secondary, mycelia of the dikaryophytic phase. Oidia are sometimes developed on secondary mycelia, when they are usually binucleate and function as ordinary conidia. There appear to be many interesting variations in the diploidisation process, even to the transfer of nuclei from a diploid to a haploid mycelium, but it would be outside the scope of this survey to follow these or the

debates to which they have given rise as to the physiological significance of the nuclear behaviour [41, 56]. There have been several reports of clamp connections in the smuts, where the paired-nuclei cell (dikaryon) is less rigidly preserved during parasitism than in the rusts. It seems that both in smuts and Hymenomycetes the partnership of the two nuclei is liable to dissolution. In *Urocystis cepulae* (p. 702), the parasite of onion smut, the cells remain long uninucleate in onion tissues, becoming dikaryophytic just before sporulation.

Heterothallism

In all the main groups of fungi it has been found that individual thalli or vegetative bodies are not necessarily identical in all their attributes in any given species. The difference to which the term ' heterothallism ' is applied relates to sexual behaviour ; in ' homothallic ' forms (Fig. 54) thalli derived from a single spore can give the perfect stage, whereas in heterothallic species (Fig. 53) the perfect stage is only produced when union occurs between the cells or gametes of certain pairs of thalli. Thalli that can contribute to the development of the perfect stage are sometimes termed ' plus ' and ' minus ' thalli (Figs. 71, 72) but it is scarcely

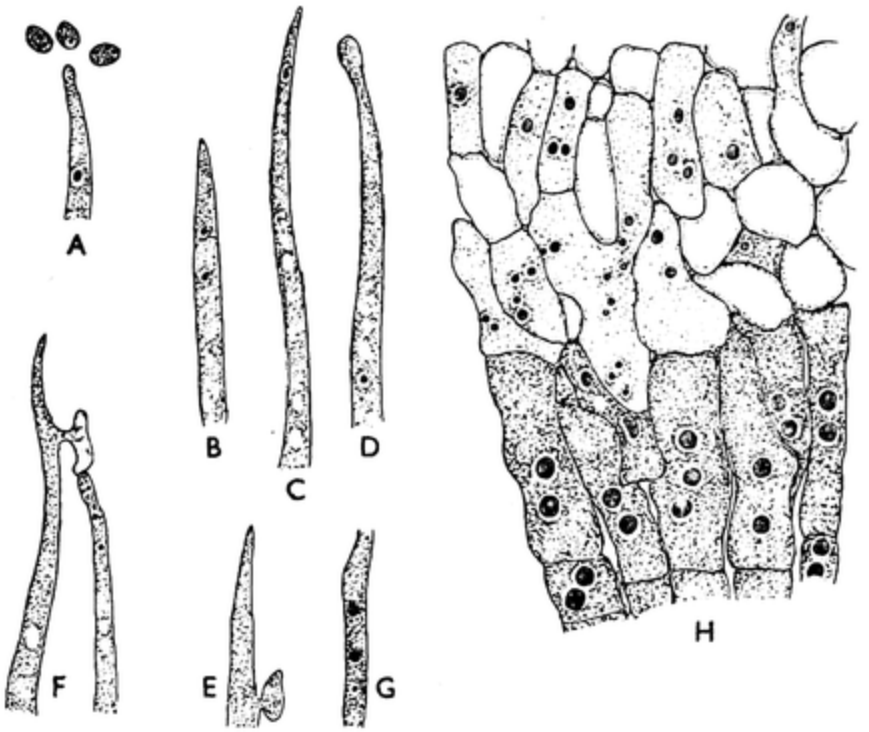


FIG. 66.—Spermatial fertilisation in the rusts. The initiation of the dikaryophase in *Puccinia phragmites*. *A*, a spermatial hypha (spermatiophore) with three spermatia ; *B*, *C*, ostiolar periphyses of the bluntly pointed type, in an unfertilised twelve-day-old monosporidial infection. *D*, an ostiolar periphysis of the bulbous type in an unfertilised fourteen-day-old monosporidial infection. *E*, a spermatium united laterally with an ostiolar periphysis in an eleven-day-old monosporidial infection which was fertilised with spermatia 48 hours prior to fixation. *F*, a spermatium united with two adjacent periphyses in an eleven-day-old monosporidial infection which was fertilised with spermatia 48 hours prior to fixation. *G*, part of a hypha of the vegetative mycelium in an eleven-day-old monosporidial infection, showing two thallus-nuclei and three spermatial nuclei. *H*, part of an aecidial primordium in a twenty-day-old infection fertilised with spermatia, showing binucleate sporogenous cells (dikaryophytic) ; some ' pathway ' cells contain, in addition to their own nuclei, residual spermatial nuclei (all × 1500) (after Lamb, *Ann. Bot.*)

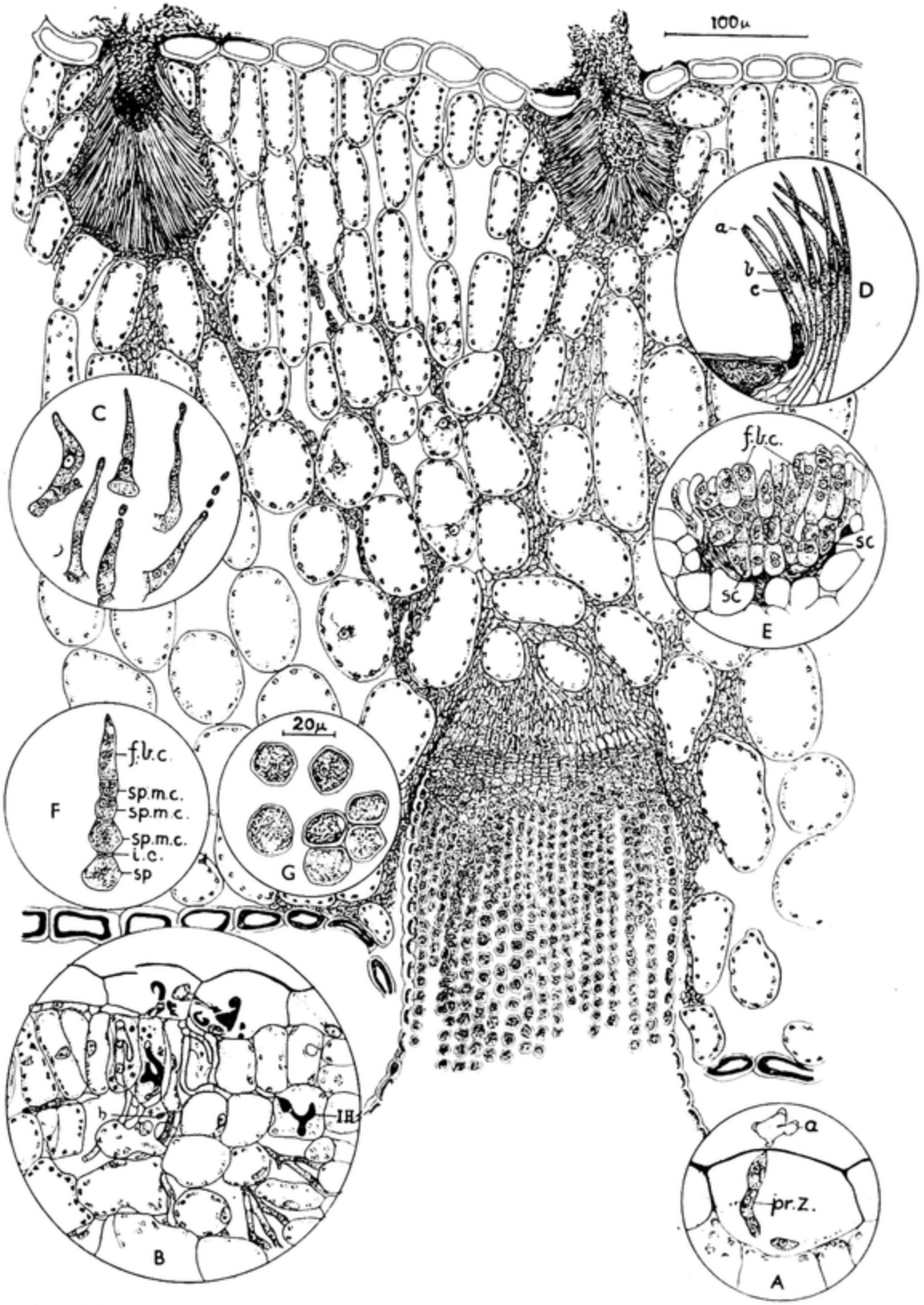


FIG. 67.—Life-cycle of *Puccinia graminis* on the barberry. Transverse section of the leaf showing two spermagonia at the upper epidermis and a mature aecidium at the lower epidermis; the intercellular mycelium appears to be in continuous connection between these organs across the leaf tissues; note the ' nectar ' and dense aggregation of spermatia at the ostioles of the spermagonia; the ' fertile cells ' are at the base of the aecidium, and cut off rows of aecidiospores, as shown, inset, in *F*; note the bell-shaped peridium which has ruptured the epidermis; insets. *A*, a two-day sporidial infection, empty sporidial wall at *a*, and a three-celled primary hypha within the epidermal cell (× approx. 565) (after Ruth Allen). *B*, a four-day infection showing

possible to restrict the conception to a single pair of signs. It is evident that heterothallism is a means of securing outbreeding, a matter the biological value of which is indicated by the many well-known adaptations directed to secure the cross-fertilisation of flowering plants (35). The apparent capacity of the different strains in heterothallic fungi to maintain themselves indefinitely in the imperfect stage, so that there is no way of knowing that they have the ability to produce the perfect form unless chance or deliberate experiment reveals it, suggests that some at least of the great mass of the Deuteromycetes or 'imperfect fungi' (see below, p. 338) may simply be strains that have lost their mates.

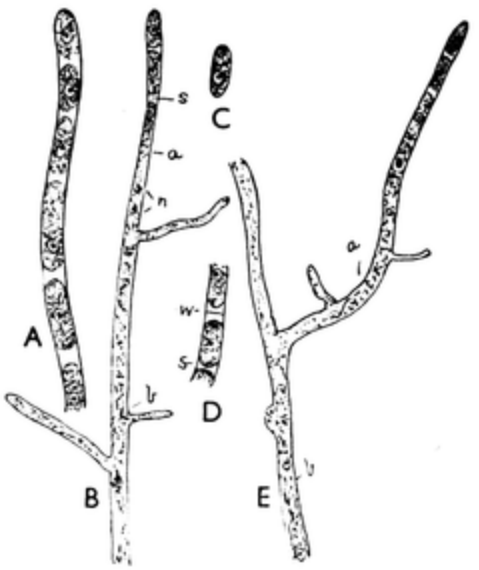


FIG. 68.—Formation of oidia in the Hymenomycetes. *A*, a chain of oidia formed by segmentation of contents of a hypha. *B*, a hypha breaking up into oidia at the apex with a clear space *s* between the oidia: *a*, indicating break in the cytoplasm; *n*, a pair of sister nuclei; *b*, a nucleus dividing. *C*, oidium. *D*, a portion of an oidial chain, with *w*, or without, *s*, a cross-wall. *E*, binucleate hypha *b*, bearing a uninucleate hypha *a* which has broken up into a chain of uninucleate oidia; note a clamp connection immediately above the letter *E*. (*A*, × approx. 900); (*B*, ×700); (*C*, ×760) (after Brodie, *Amer. J. Bot.*)

An alternative suggestion that nutrition rather than sexuality forms the basis of heterothallism has found some support. In this nutritive theory each of the various strains is thought to possess a gene or factor, absent from the others, enabling it to utilise some essential food material. Union of compatible strains brings ability to use all that is required (24).

Asexual Reproduction

Out of the multiplicity of asexual spore forms found in the fungi certain main types may be mentioned. The 'sporangia' of the Phycomycetes may contain unicellular 'zoospores', the animal-like attribute implied in the name being their power of swimming about in water by the movement of one or two 'cilia' or 'flagella', oar-like protrusions of the naked protoplast, attached terminally or laterally (Fig. 25). After this free-swimming period the zoospore comes to rest, rounds off, usually becomes provided with a cell wall, and then germinates by a germ-tube or by a short process which penetrates the host cell. Germination sometimes results

disintegration of primary hypha and intracellular hypha at *IH*; note small haustorium at *h*, the mycelium being now intercellular (× approx. 365) (after Ruth Allen). *C*, various spermatial hyphae showing abstriction of male cells (spermatia) (× approx. 730) (after Ruth Allen). *D*, some paraphyses at the ostiole of a spermagonium in a twenty-day infection; some contain a pair of nuclei *b*, *c*; sometimes, as at *a*, there is an additional smaller nucleus (× approx. 360) (after Ruth Allen). *E*, the basal, or fertile cells, *f.b.c.*, which abstrict the binucleate spore cells below; note, below the young spore cells *sc*, breakdown of sterile fungal tissue (dark-shaded) between these cells and the lower epidermis of leaf (after Ruth Allen). *F*, a spore chain with a multinucleate fertile basal cell *f.b.c.*, abstricting binucleate spore mother cells *sp.m.c.*, from which an aecidiospore *sp* and an interstitial cell *i.c.* are finally produced. *G*, at this early stage within the base of the aecidium the aecidiospores are hexagonal in outline, from mutual pressure of neighbouring chains

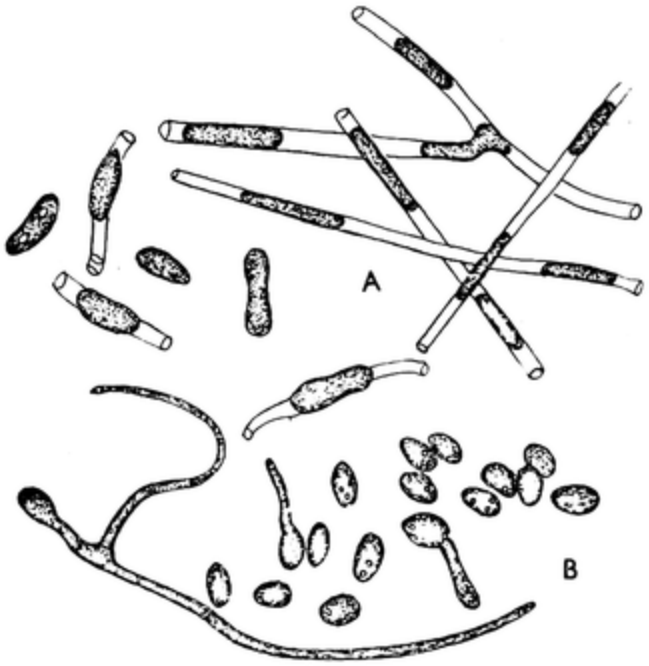


FIG. 69.—Hyphae of *Clitocybe gigantea* breaking up into oidia, some of which are copulating, thus initiating a dikaryophytic phase (× approx. 550) after Bayliss-Elliott, *J. Econ. Biol.*)

in the liberation of a second zoospore on which the position of the flagella may now be different, apical or lateral, from their previous position (' diplanetism '), so that the period of free-swimming life is prolonged (Fig. 290 D). A transitional stage in the adaptation of the aquatic fungi to life on land is, perhaps, to be observed in the *Peronosporaceae* (downy mildews) and some of their allies. In many of these the sporangium often fails to produce zoospores but germinates directly by one or more germ-tubes, just as if it were wholly transformed into a single spore. In the Zygomycetes, unlike the aquatic families of the Phycomycetes, the spores borne in the sporangia are not motile but are suitable for passive dissemination in the air or water (Fig. 73); these are amongst the commoner land moulds and include the well known *Mucoraceae* such as the bread moulds and others found on decaying organic matter in the soil and elsewhere. Most mucors have mucilaginous spore heads and are not readily dispersed until the head breaks up in water or in the soil solution and the individual spores adhere to soil particles and the like and are dispersed on these [17]. In some of the other genera these dried minute unicellular spores, when liberated by the disintregation of the sporangial wall, become air borne and are blown about as dust. *Pilobolus* has the whole sporangial head shot by a water jet generated in the sporangiophore to a distance said to be sometimes as much as a yard (Fig. 74 A-D).

Asexual spore types in the Ascomycetes are legion. To most of them the general term ' conidium ' is applied, and this name is also often given to those sporangia of the Phycomycetes which germinate directly by germ-tubes; they are, nevertheless, potential sporangia in this group. The conidia of the Ascomycetes may be composed of one or many cells, uni- or pluri-nucleate, and of the most diverse shapes from simple rounded or oval forms to branched bodies, sometimes furnished with slender cilium-like antennae from the end cells.

In the simplest case a conidium is merely the (usually swollen) end of the fertile hypha (' conidiophore ') or of a small stalk from the end or side of the latter. The spore is cut off by a septum from the parent hypha and may be called a thallospore. Very often, instead of a single spore being thus formed, a chain of spores arises from the sporophore or conidiophore. These chains may be developed in either of two ways; most commonly, each new spore of the chain is successively cut off from the tip of the parent hypha so that the end spore of the chain is the oldest (' basipetal ' formation) (Fig. 75); less often, the first-formed spore buds

out another from its tip, and this in its turn another, so that the end spore is always the youngest (' acropetal ' formation). As there may be more than one bud from the tip of a spore, the chains of the second type are often branched.

After a conidium or a chain of conidia is formed on the end of the conidiophore, the latter may continue to grow from a point just below the tip. The new growth usually tends to preserve the same direction as the original stalk, so that the spore is pushed over laterally. Slight kinks or knee-bends are often visible at the points where such lateral spores have been shed (Figs. 26, 76 A).

Instead of the tip of the conidiophore bearing a single spore or chain of spores,

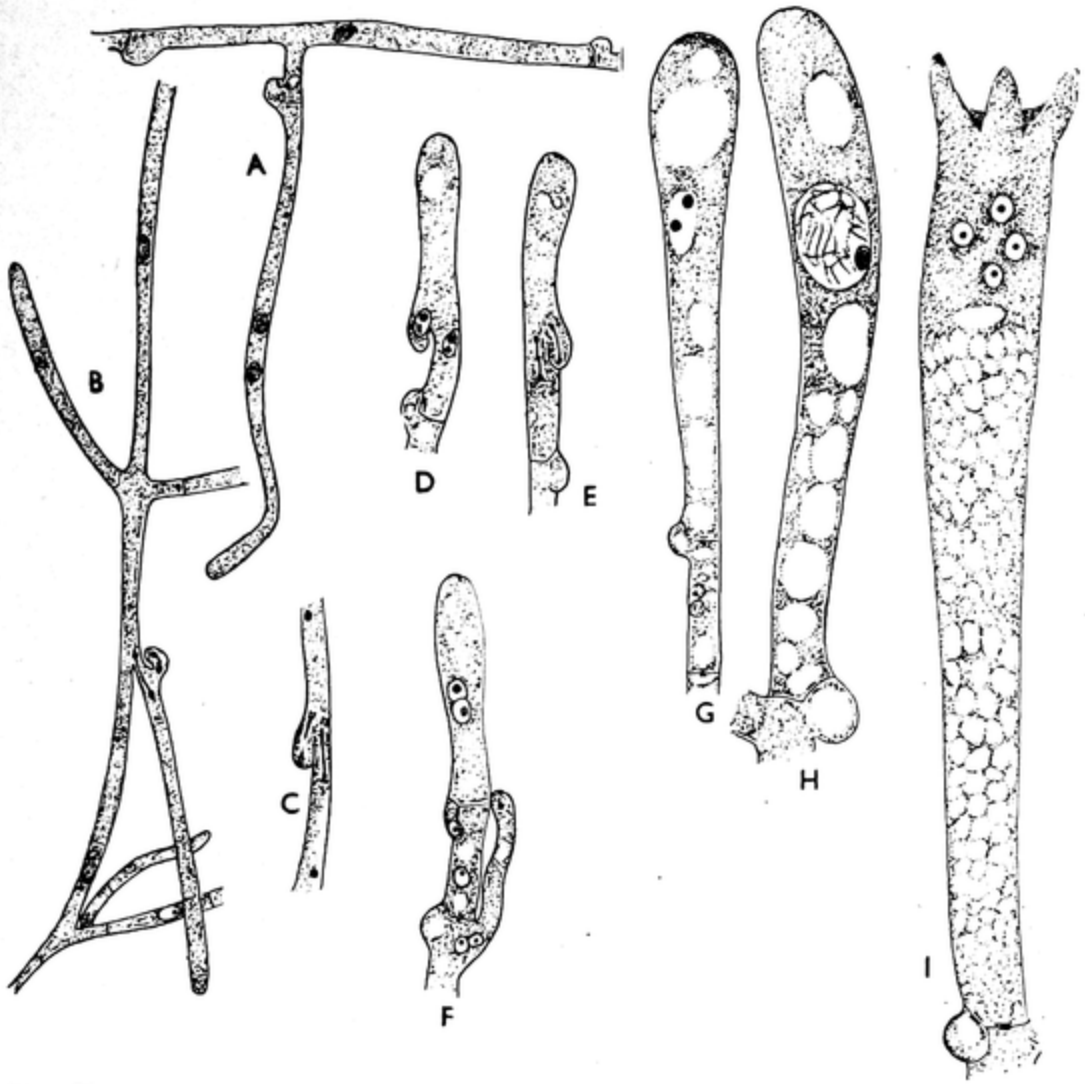


FIG. 70.—Clamp connections and the initiation of the dikaryophase. *A*, branching hypha of mycelium of *Corticium serum*, showing the parent cell uninucleate ; the initiation of two clamps at the end walls ; the branch hypha is binucleate, a nucleus having probably passed over into it by way of the clamp connection above, from the parent cell, after nuclear division. *B*, *C*, *Collybia conigena* showing conjugate division of paired nuclei in the clamp connection on the right, as shown at *C*. *D–I*, *Armillaria mucida* showing development of basidium, following transference of nuclei through clamp connection. *D*, the paired nuclei taking up position at the clamp. *E*, the conjugate division. *F*, two of the resulting four nuclei pairing in the young basidium ; of the other two, one remains in the bulge, the other in the stalk-hypha. *G*, *H*, basidium with the fusion nucleus (' deferred fertilisation '). *I*, division resulting in four haploid nuclei which finally pass into the four basidiospores (after Kniep)

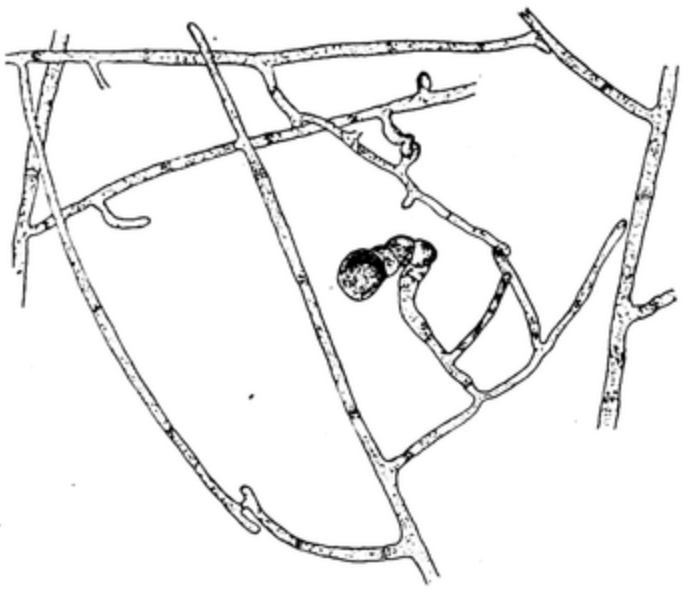


FIG. 71.—Heterothallism in the fungi. *Humaria granulata* (Ascomycetes) showing an archicarp developing in the neighbourhood of anastomosis between + and − mycelia (×200) (after Gwynne-Vaughan & Williamson, *Ann. Bot.*)

it often branches and the conidia are borne on the ends of the branches, which may be of limited growth and characteristic shape as in *Penicillium* (Fig. 77 C). Or instead of branching, the apex of the fertile hypha may enlarge into a head or columella as in *Eurotium* (Fig. 77 A) which bears a number of spores or chains of spores, again usually on special short stalks. The term sterigma is again often applied to such a spore-bearing stalk and, as it may branch one or more times before the spores develop, sterigmata of a second or third order may be recognised.

The sterigma, or hyphal branch, which abstricts conidia in succession, thus becoming constricted just below its apex, before cutting off the spore, is often called a phialide, and the abstricted conidium a phialospore, e.g. *Penicillium* (Fig. 77 C), *Margaritispora* (Fig. 76 B), *Flagellospora* (Fig. 76 C). During this development there is no formation of a cross-wall separating the spore from its phialide, and a cleavage line arises separating the two when the spore is fully grown. When, however, a newly developed spore is delimited from its parent hypha by a new transverse wall, it is called an aleuriospore (Fig. 76 D, E, F). In some forms (*Anguillospora longissima*, Fig. 76 F) it is not certain where the conidiophore and the aleuriospore begins, until the separating cell is differentiated. Again, spores formed on a sterigma or hypha, without reference to the growing point of the hypha, are called radulospores, e.g. *Botrytis cinerea* (Fig. 77 D). Some fungi develop their spores in two of these ways, thus the last-named type may also produce phialospores, and *Acrospeira levis* may have aleuriospores

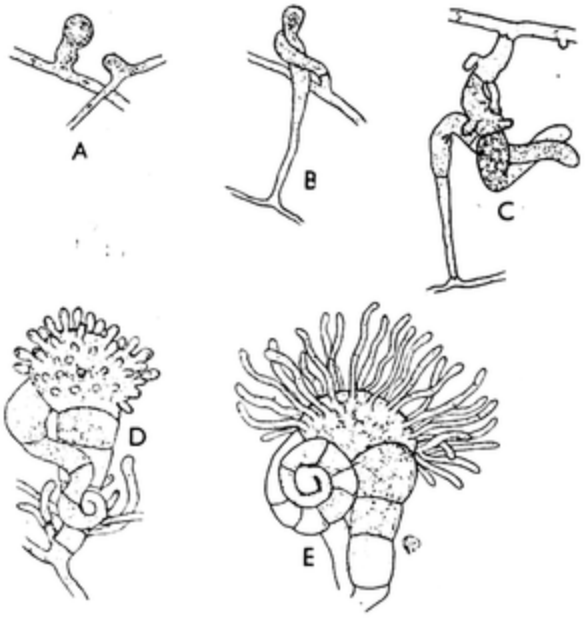


FIG. 72.—Heterothallism (*continued*). *Ascobolus magnificus*. *A–C*, pairs of young male and female branches; in *C*, the first hyphae of the sheath are growing out. *D*, the oogonium forming ascogenous hyphae; the tip of the empty antheridium is attached to end of trichogyne. *E*, oogonium with ascogenous hyphae and empty antheridium (all ×200) (after Gwynne-Vaughan & Williamson, *Ann. Bot.*)

and phialospores, but others, such as *Penicillium* produces, asexually, only phialospores. Thallospores, as above stated, are single spores derived from a mere swelling of the end of a hypha, and are not distinct structures, and while the distinction between radulospores and phialospores is not always sharp, that between thallospores and phialospores is fundamental (25 a). Some spores are formed more or less continuous along the hypha, with no sterile internodes in between as are found in sporodochia, and are called pionnotes; in pionnotal cultures sporulation takes place freely within the medium as well as on its surface, but in sporodochial growth, sporulation is almost entirely confined to the surface.

Outside the three main groups, Phycomycetes, Ascomycetes, and Basidiomycetes, there remains a vast assemblage of fungi known only in the conidial stages. These imperfect species, the Deuteromycetes or Fungi Imperfecti, are assumed on good grounds to be mainly asexual stages of Ascomycetes. Their numbers are slightly reduced from time to time by the discovery of the place of one of them in the life-cycle of some ascigerous species, but it seems probable that in many the perfect stage has been lost and the fungus is perpetuated indefinitely in the conidial condition. They include many parasites and amongst them are represented every type of asexual spore and sporophore (except sporangia and sporangiophores) mentioned above, the simplest forms being designated Hyphomycetes and the pycnidial forms Coelomycetes. Some hesitate to apply the terms genera and species to such imperfect organisms and prefer to qualify them as ' form genera ' and ' form species '. So long, however, as systematics has not reached the point of indicating true affinity (and that is still far off) there seems to be little reason for this refinement. Names based on characters that can again be identified are necessary to group organisms together into recognisable units, and a fungus capable of independent existence, perhaps



FIG. 73.—Spore dispersal. The Mucoraceae. *A*, much enlarged head of a typical mucor sporangium showing the columella and spores (diag.). *B–D*, *Rhizopus stolonifer*. *B*, dehisced sporangium projecting into the air; the collapsed columella bears a mass of spores and fragments of the sporangial wall; many of the spores have already been blown away. *C*, a similar sporangium from which nearly all the spores have disappeared. *D*, from a spore deposit collected on the lid of an inverted culture; the spores, being dry, have a wrinkled appearance (after Ingold, × 500). *B–D* (after Ingold, *New Phytologist*)

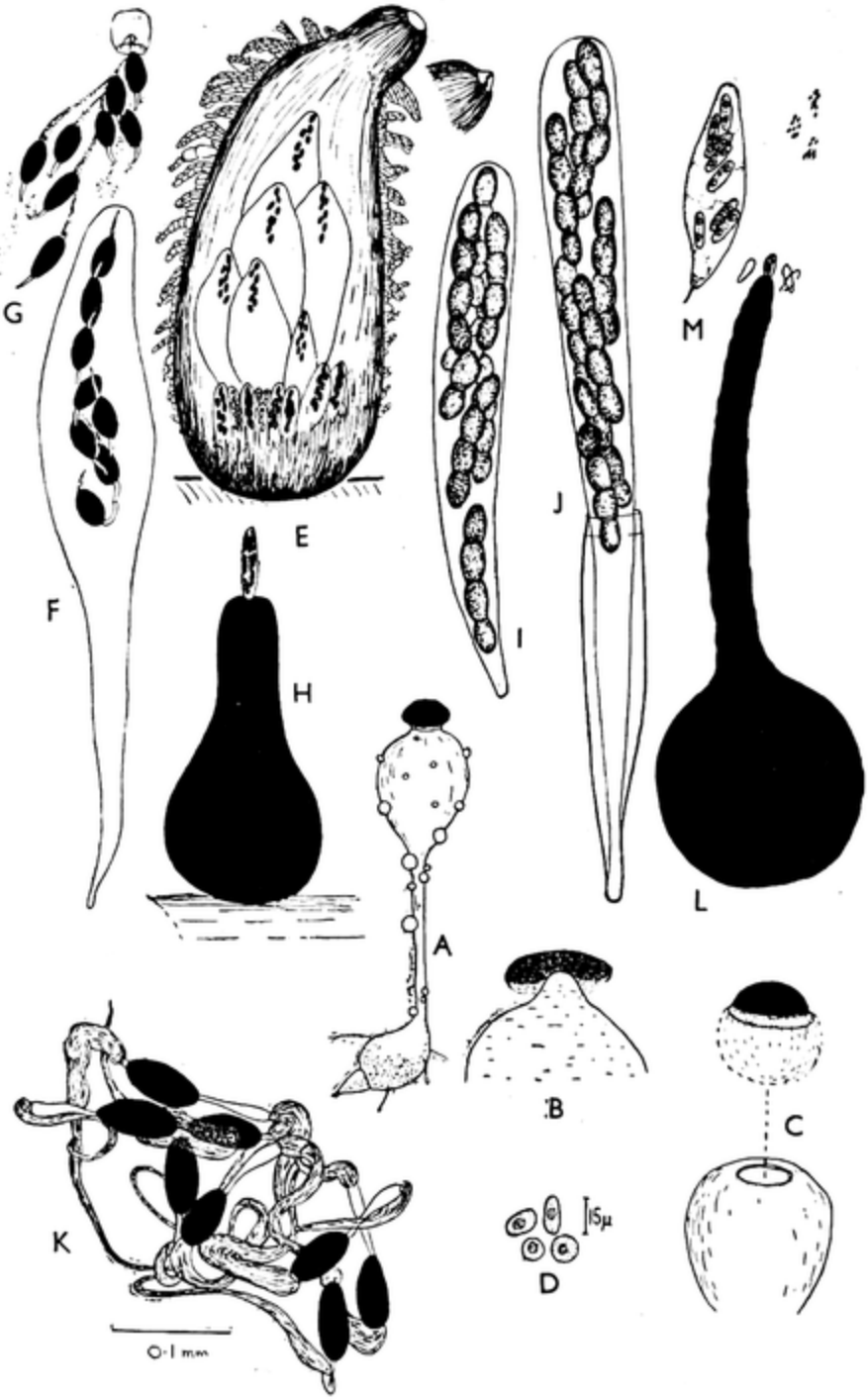


FIG. 74.—Spore dispersal (*continued*). *A–D*, *Pilobolus kleinii* (Mucoraceae). *A*, mature sporangiophore showing sporangium at apex, drops of liquid exuding from the stalk. *B*, the sporangium (black) filled with minute spores, with clear mucilage below; the cone-shaped tip of columella is in contact, through the mucilage, with the centre of the black-capped sporangial mass. *C*, the entire sporangium ejected. *D*, the spores. *E–G*, *Podospora curvula* (Ascomycetes). *E*, perithecium with asci showing through the transparent wall (×72); ostiolar region (10 min. later) shown on the right; the tip of the ascus which was leading in *E* has reached the ostiole and is about to discharge its spore mass. *F*, a single ascus which has commenced to swell (×167). *G*, a discharged spore mass showing the ascus-cap attached to the apical spore (×167). *H–J*, *Sporomia intermedia* (Ascomycetes). *H*, a perithecium (on a piece of straw) showing protruding ascus tip (×95). *I*, ascus, before rupture of the outer wall (×421). *J*, ascus, after rupture of the outer wall and elongation of the inner wall (×421). *K*, *Podospora fimiseda*, an ejected mass of spores held together by strands of mucilage exuded by the spore-apex and by the appendage cell. *L*, *M*, *Ceratostomella ampullasca* (Ascomycetes). *L*, a perithecium in active discharge; an ascus is still held in the ostiole and is about to explode; three empty asci close by have ejected their spore loads shown above (×40). *M*, a single, mature detached ascus (×308) (all after Ingold, *New Phytologist*)

indefinitely, in a conidial stage appears to be fully entitled to such dignity of status as may be implied by the use of a properly employed binomial terminology.

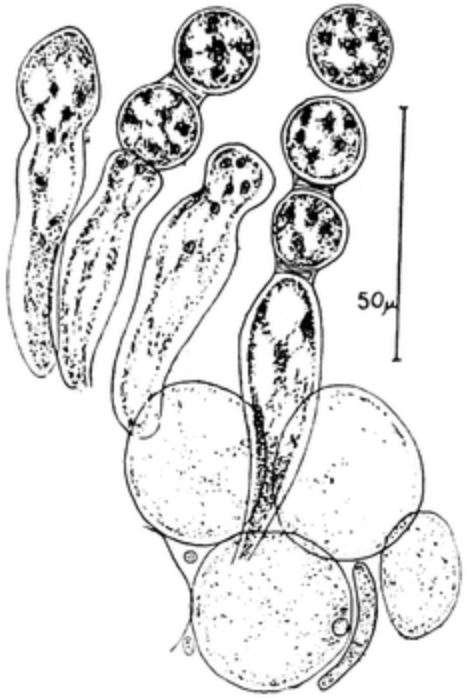


FIG. 75.—Asexual spores. *Cystopus candidus*. The multinucleate sporangiophores and sporangia; note the origin of latter from the swollen tip of a fertile hypha, and others formed in basipetal succession; cellulose joints or disjunctors, which break down prior to dispersal, are shown between the sporangia; note 'knob' haustorium in host cell

POLYMORPHISM

The occurrence of different types of spore and of spore-bearing structures in the life-history of an individual fungus has been indicated already, Ascomycetes, rusts and Fungi Imperfecti being particularly rich in examples. A rust, for instance, may have spermagonia, aecidia, uredosori, teleutosori, and the promycelium (bearing sporidia) in its full development. So also an Ascomycete may have a simple Hyphomycetal spore stage and a development of pycnidia, or spermagonia before asci are produced. In the life-history of some Hyphomycetes the type of spore found may vary, presumably as a result of age and the action of environmental conditions, to such an extent as to place the fungus in different genera according to the prevalent spore form encountered. Thus, the well-known *Heteropatella antirrhini*, the cause of 'shot hole' of cultivated snapdragon, may appear as a *Pseudodiscosia* or a *Cercosporella*, which is actually only a summer conidial (acervular) stage of the pycnidial form *Heteropatella* on the dead or moribund stems (Part II, p. 850): and *Ramularia vallisumbrosae* (Part II, p. 866), the cause of 'white mould' of narcissus, may appear as an *Ovularia* or a *Cercosporella* instead of a typical *Ramularia* [21]. Similarly there is reason to believe that *Ovularia primulae* and *Cercosporella primulae* are the same fungus as *Ramularia primulae* [38]. Amongst pycnidial fungi, again, it is probable that *Phoma destructiva* and *Diplodina lycopersici* refer to the same tomato parasite, the perfect stage of which is *Didymella lycopersici* (Part II, p. 667), while it seems likely that *Ascochyta lycopersici* is only another phase of this polymorphic species. Species of *Septoria* and *Stagonospora* are also stated at times to pass through an *Ascochyta* stage [23]. It is cases such as these that give force to the arguments against recognising imperfect forms as unqualified genera and species, but many of them are due to such notoriously variable characters as spore septation and sporophore type. Indeed, it is not unknown for variable characters in the sporophore to make it difficult to assign, say, one of the *Polyporaceae* to its proper genus.

In the opposite direction to the conidial forms mentioned in the last paragraph are those in which similar conidial stages may occur in fungi that are systematically far removed from one another. Thus both the Ascomycete

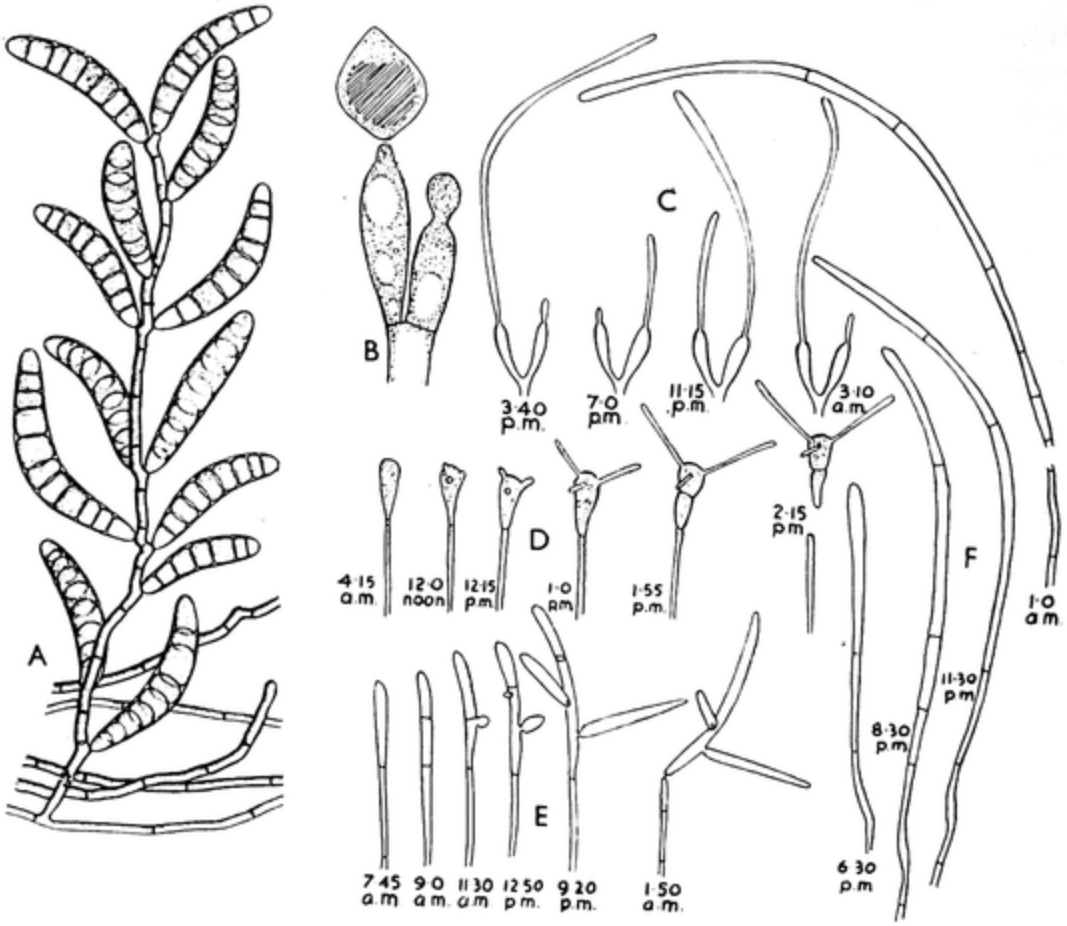


FIG. 76.—Types of conidiophores in asexual reproduction. *A*, a conidiophore of *Ophiobolus heterostrophus* showing ' knee joints ' (geniculate) at which septated conidia are developed in sympodial fashion (after Drechsler, *J. Agric. Res.*). *B*, development of phialides and phialospores in *Margaritispora aquatica*; note the shaded glycogen vacuole in the spore (×613). *C*, *Flagellospora curvula* showing four stages in the development of phialospores (×370). *D–F*, development of aleuriospores. *D*, *Clavariopsis aquatica*, six stages in the development of a spore, the last stage showing disjunction; upper cell with three processes (×233). *E*, *Tricladium splendens*, six stages, as above (×220). *F*, *Anguillospora longissima*, four stages; it is difficult to say where the conidiophore ends and the aleuriospore begins; note the indefinite breakage (×270) (after Ingold, *Trans. Brit. Myc. Soc.*)

Mycosphaerella and the smut *Entyloma* may bear a form referable to the genus *Ramularia*.

THE DISSEMINATION OF FUNGI

A few fungi are disseminated principally in the mycelial state as, for instance, the tropical thread blights which blow about with the leaves to which they adhere; the ' Corticium ' disease or ' red thread ', a common trouble of lawns and sports greens in Britain, produces on the leaves a mycelium of coral-like threads which adhere to the tips of the dying leaves (Part II, p. 481). Others are distributed as vegetative propagants such as the sclerotia and stromatic crusts of *Rhizoctonia solani*, of which the sporing form, *Corticium solani*, plays little part in spreading the fungus (Part II, p. 528). The long-distance dissemination of fungi is, no doubt, often to be traced to the transference of viable mycelium in plant produce from one country to another; this has happened in elm disease and very probably in potato blight and with many other fungi.

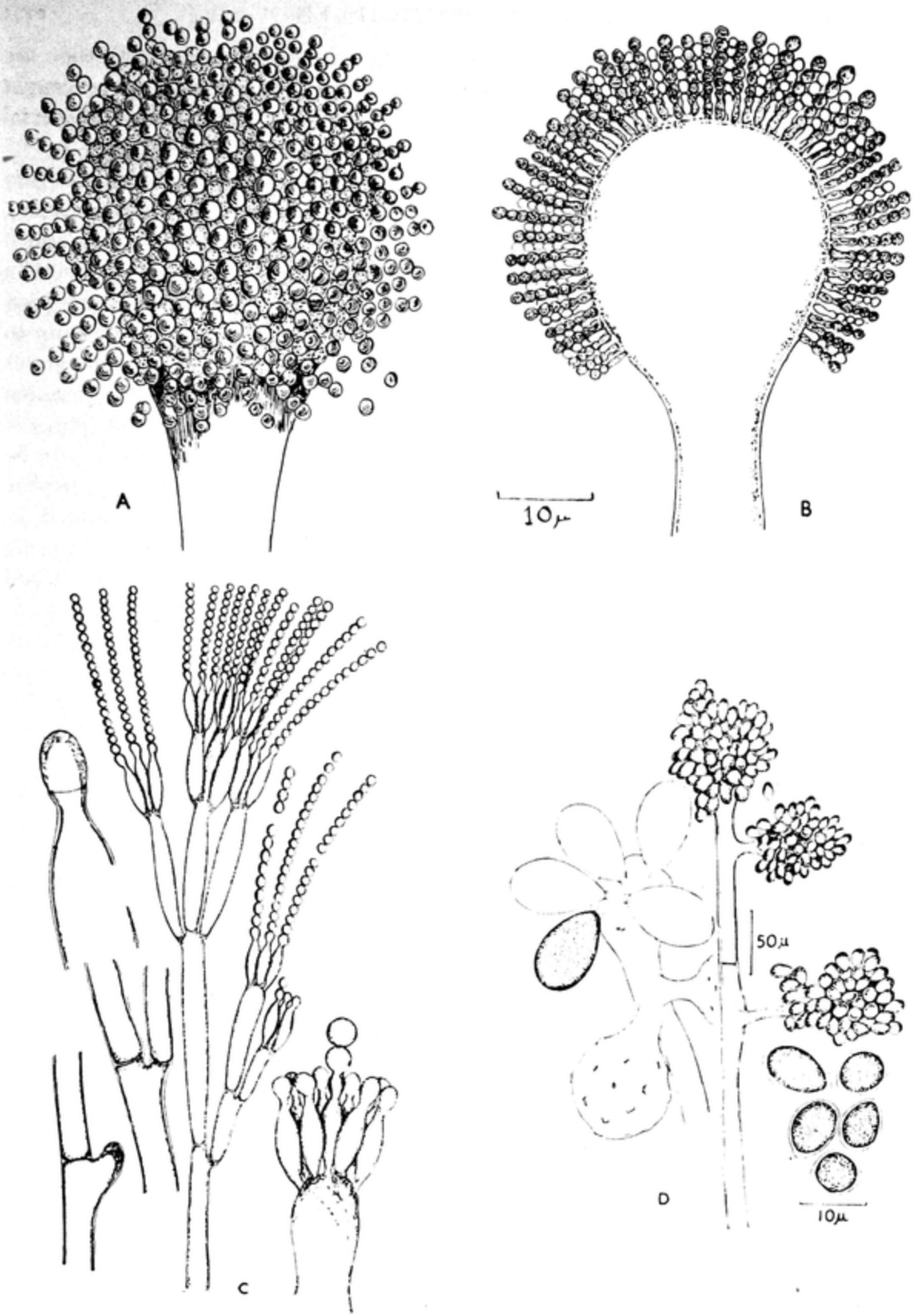


FIG. 77.—Types of conidiophores (*continued*). *A*, *B*, *Eurotium* (*Aspergillus*). *A*, the sporangial head in surface view. *B*, the same, in section, showing the origin of the sterigmata as small phialides over the whole surface of the swollen end of the conidiophore, each sterigma abstricting a chain of conidia. *C*, branching conidiophore and mode of development of phialides and conidia in *Penicillium*. *D*, the irregularly branched conidiophore of *Botrytis cinerea*, with a portion, to the left, enlarged

Usually, however, the spores are the organs of dissemination. As they are very small (invisible singly to the naked eye) and so light as easily to be caught up into the air as an impalpable dust, the wind is the chief agent in their dispersal (Fig. 79 A, E); there are, however, other important methods of spread.

The spores of fungi may retain their vitality for considerable periods (dry spore dust of *Aspergillus oryzae* up to 35, and of *Ustilago crameri*, up to 64 years), and they show amazing powers of resistance to extremes of heat and cold: sealed in vacuum tubes, slowly dried spores of *Mucor* and *Aspergillus* have withstood a temperature of liquid air (−190° C.) for nearly 500 hours, followed by immersion of the tubes in liquid hydrogen (−253° C.) for 77 hours [2]. Susceptibility to heat varies greatly, for some spores are killed in 10 to 15 minutes at temperatures far below the boiling point, whereas occasionally some can stand the direct action of boiling water or live steam for the same length of time. So also the spores of some species are killed by drying for 24 hours or even less, while others can be best preserved in a dry condition. As a rule spores are less sensitive than hyphae to adverse surrounding conditions, and vegetative growth is often inhibited at, or near, the freezing point, as is evident from the action of refrigeration in checking mould growth; slow growth on frozen foodstuffs, however, has been recorded, and very slight superficial mouldering of meat will occur at a temperature of −6° C.[5]. Unless special precautions are taken, mouldering as a result of spore germination quickly supervenes on removal from cold storage. These resistant properties of spores are of value in facilitating the aerial dissemination of many fungi, for desiccation in the air and extremes of heat and cold on the ground may have to be faced.

The vertical elongation of the sporophore is often a powerful aid to exposing the spores freely to air currents. Amongst other adaptations which seem to have the same object, the forcible projection of spores from their stalks is often important. When enclosed in a receptacle there are sometimes arrangements for expelling the spores actively into the air. These are often brought into action by changes in temperature or dryness, as in some Ascomycetes where 'puffing' (the expelling of a number of spores together in a cloud) can be induced by concentrating the sun's rays through a pocket lens on the receptacle. Some of the Discomycetes respond similarly if breathed upon when ripe. In the Basidiomycetes it is usual to find a mechanism for the violent release of the basidiospores based on the secretion of a drop of fluid at the junction of the spore and its sterigma (Fig. 78). For full advantage to be gained from these various adaptations it is necessary that the spores should be dry and free from mucilaginous envelopment.

In a considerable number of species, including many fungi whose spores are developed in acervuli, the spore beds are surrounded by a gummy secretion (Fig. 74). A great many of the bacterial plant pathogens also are immersed in mucilage as they are extruded to the surface of the host plant. When dry, the secretion tends to harden into flakes or sheets, but these are readily soluble in water, as in 'halo blight' of French beans, due to a bacterium (*Pseudomonas phaseolicola*) (Part II, p. 596). Dissemination may be effected by the blowing about of the dried gummy shreds, followed by the setting free of the individual cells in rain drops, as sometimes occurs in the American fireblight of fruit trees, due to *Bacterium amylovorum*, or

in *Xanthomonas malvacearum* in cotton, though the latter is often also carried as dust from adjacent cotton fields infected the previous season. The spore bed may be exposed to rain splashing which carries the spores to leaves or stems in the vicinity; species of *Colletotrichum* are often disseminated in this way. Amongst the Hyphomycetes it is possible to distinguish two main groups according to the slimy or dry character of the spores, and the method of their dispersal depends largely on this character, the dry spores being predominantly suited for distribution by the wind, while the slimy ones are carried by water, by insects, or by adhering to adjacent plants and the like. In a recent survey of the British Hyphomycetes three-fourths of the species belonged to the dry-spored group (57). The Coelomycetes, on the other hand, are generally representative of moist sporing fungi. In some of them and in many of the moist-spored Hyphomycetes, the spores are liberated in a mass held together by water or mucilage, and in this form may be subject to aerial dispersal before they are broken up by rain or dew.

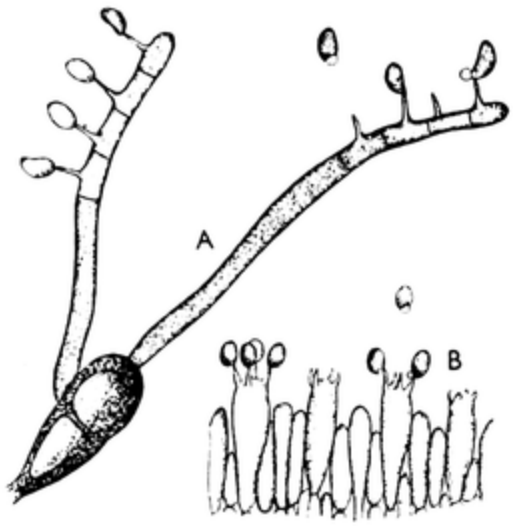


FIG. 78.—Mechanism of spore release in the Basidiomycetes. The 'water drop' mechanism. *A*, basidia of *Puccinia*, developed from the teleutospore, showing, on the right, a spore-projectile with an attached drop of water. *B*, basidia of an agaric type, showing the same mechanism (after Buller)

Knowledge of the long-distance air-borne dissemination of fungal spores has greatly increased of recent years (54a). Observations on the spread of white pine blister rust (*Cronartium ribicola*) in the west of the United States and Canada indicate that it is due to wind-blown infection of currant and gooseberry bushes by aecidiospores from pines that may be hundreds of miles away (see p. 934) (30). Similarly, cereals are infected over great areas by wind-borne uredospores of their rusts. Numerous examinations of the spore-content of the upper air have been made by aeroplane. In early flights, carried out in 1921, spores and pollen grains were found to be fairly abundant up to 11,000 feet, above which they became scarce. Uredospores of *Puccinia graminis* caught at 7,000 feet were germinated successfully. Above 10,000 feet, mould spores, such as those of *Aspergillus* and *Penicillium*, and bacteria, are most frequently found, occurring at times up to nearly 20,000 feet (40). There is circumstantial evidence that the distance to which such spores may be borne in a viable condition during storms is limited mainly by their ability to survive atmospheric conditions. These naturally vary greatly from year to year during the period of spread, but it is reported from Canada that there is rather conclusive evidence that under certain conditions the black rust of wheat has been carried for four or five hundred miles, or even farther, through the air (15). These spores (and, no doubt others) tend to be deposited in definite showers rather than in a continuous steady fall; in western Canada rust spore-showers occur after a day or two of strong south wind.

The long-distance dissemination of cereal rusts has been intensively studied.

Black rust due to the well-known parasite *Puccinia graminis* occupies a practically continuous wheat belt in North America from the Gulf of Mexico to the Prairie Provinces in Canada ; as the crop is maturing in the south at the same time that it is being sown in Canada, and the intervening areas are more or less intermediate, a regular succession of wheat ready for infection is available from south to north in the spring and summer. The spores seem to follow a main northward drift-path from an area in Texas, where the rust over-winters in the uredo stage and the wheat ripens in the late spring, through Kansas in May and June, to the upper Mississippi States. In Texas itself the summer is intensely hot and the rust is unable to survive until the new crop is sown in the autumn. Re-infection then takes place, apparently chiefly by a reverse flow of spores from the north. So there is an ebb and flow of infection to and from the area in which this fungus over-winters but cannot survive the summer. Confirmatory evidence of this movement is provided by the incidence in particular years of certain physiologic races (see p. 89) of the fungus in the areas concerned [36]. No doubt the spread is not ordinarily carried out in great jumps, though spores not of local origin have been caught as far north as Nebraska in May and June : usually there is time for one or more breaks and the renewal of the inoculum (spore dust) by the infection of local crops in the path from Texas to the northern Mississippi and Canada [54]. Some irregularity of incidence in the upper part of this tract may result from the local over-wintering of the fungus in the teleutospore stage and infection of the alternate host, the barberry, in the spring, for this rust is heteroecious and produces its aecidial stage on barberries, whence it can infect neighbouring wheat and possibly start an epidemic. But many of the barberries have been destroyed in the barberry eradication campaign (p. 225) and their disappearance in due course will, it is hoped, save much of the chief wheat-growing region in the northern States and Canada from all but the northward spore drift, for the region is much too cold to allow the rust to over-winter in the uredo stage. Attention can then be concentrated on the production of rust-resistant wheats in the comparatively restricted over-wintering area in the south.

Nearly all the wheat grown in India is found as a winter crop in the plains. In these areas rust is unable to survive the intense heat of the summer, and the wheat and barley crops are infected anew each year from rusted cereals grown in the Himalayan foot-hills and some of the hills of Peninsular India [37]. Barberries occur in these hills but not in the plains, and though the aecidial stage of *Puccinia graminis* has been found on several of the Himalayan species, it seems to play little part in the perpetuation of the rust. The uredo stage is abundant when the hill crops are harvested in May and June, and then persists on stubble shoots and volunteer plants until the new hill crops are sown in the autumn. Areas have been found in the Central Himalayas where the early crops are sown in August-September and may be heavily rusted by the first week of December. Inoculum is thus available in time to account for the first outbreaks in the Indo-Gangetic Plain, which usually occur in January-February. The uredospores have been trapped in the air by means of specially devised aeroscope slides well before the rust has been found on the crops in the plains. In Peninsular India similar foci of early infection of the growing hill crop have been found and seem to account for ex-

tensive distribution of the rust in Madras and the Deccan. A similar chain of infection serves to explain the distribution of wheat brown rust (*Puccinia triticina*), whereas *Puccinia glumarum*, the cause of yellow rust, is more sensitive to heat and its uredosori are not ordinarily found in the summer below about the 6,000 feet level in the Himalayas, though it may be the only rust seen above 7,000 feet in November-December.

The long-distance dissemination of cereal rusts in several parts of the world is effected on the same broad lines. Thus the wheat in the Amur region of eastern Siberia gets its inoculum from over-wintering areas in Manchuria; North Africa seems to be infected from Europe, the Balkans from Asia Minor, and the Argentine from Brazil. So to a great extent has been cleared up one of the greatest mysteries with which plant pathologists in certain parts of the world were faced before it was known that spores could be carried over long distances in the upper air.

Such long-distance spore flights are evidently limited to certain types of spores and of prevailing meteorological conditions, at least so far as the parasitic fungi are concerned. If they were general, the object of the plant quarantine services maintained by most countries would be defeated. Heavy wind-drifts or storms no doubt play an important part in them, as does the presence in a receptive condition of a susceptible crop in the area reached. It is known, furthermore, that spore-contaminated air not far from the ground purifies itself by the fall of the spores at rates varying from 0·5 to 5 mm. per second, while their presence over the ocean has been found to diminish with increasing distance from the land [26], and catches taken on shipboard may fall to nil in individual voyages fairly soon after reaching the open sea [3]; various favourable conditions must coincide to permit successful long-range spread, and they do not seem often to occur.

There is still a great weight of evidence from the known facts of the spread of parasitic fungi into new areas in support of the view that 'discontinuous' dissemination is usually effected by transport on or in the host plant, and is linked with the movement of nursery stock or other plant produce along recognised trade routes [7]. The discovery of spore-passage in the upper air and its bearing on the dissemination of the rusts shows that wind-borne distribution can be an important factor in certain circumstances and must always be regarded as a possible menace by those responsible for the protection of crops against foreign disease. It is also ordinarily accepted as responsible for much of the 'continuous' dispersal of fungi within any given land area. But sea-barriers and sometimes mountain ranges have proved effective checks to the dissemination of many plant parasites. Either the spores are unable to survive long passages in the air, or the timing of their flight is such that a suitable host in a receptive condition is not available when the new area is reached, or the meteorological conditions (prevailing wind, drought, etc.) are adverse, or other causes intervene to spoil a safe lodgement. Many favourable circumstances must combine to render the invasion successful, and experience suggests strongly that they seldom do. Supporting evidence can be found in the history of the spread of American gooseberry mildew (*Sphaerotheca mors-uvae*) (Part II, p. 817) in the British Isles and on the continent of Europe; of potato wart disease (*Synchytrium endobioticum*) (p. 499) in many parts of the world, though the resting sporangia of this fungus might be expected to reach

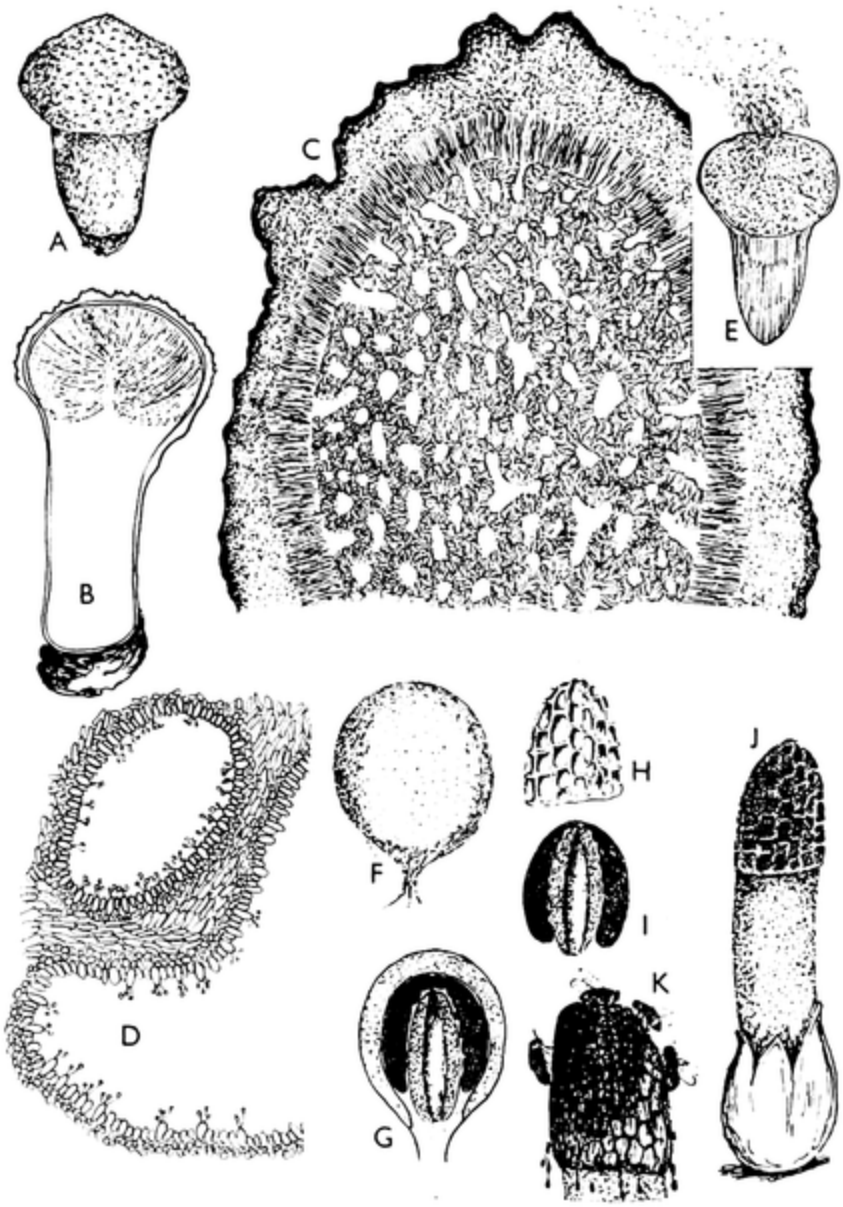


FIG. 79.—Spore dispersal in the glebal fungi (Gasteromycetes). *A–E*, *Lycoperdon*, a typical puff-ball. *A*, the fructification consists of a fertile head and a sterile stalk. *B*, the same, in section, showing the sporogenous ' gleba ' in *C* as a spongy mass, the cavities in which are lined with basidia as in *D*. Towards maturity the gleba dries to form a powdery mass, and when the thick wall or peridium opens at the top the spores are emitted, as at *E*, when the fructification is shaken (× ½). *F–K*, *Phallus* (Stinkhorn). *F*, the unopened fructification (about the size of a hen's egg) is partly concealed in the soil and is attached by long white rhizomorphs. *G*, the same, in vertical section, showing the dark gleba supported on a hollow, spongy stalk, the whole covered with a thick, gelatinous volva or peridium. *H*, the gleba is held in place on the reticulated surface of a thimble-like receptacle. *I*, the greenish gleba, in section, on the receptacle. *J*, the fructification at time of spore dispersal, the dark gleba on the receptacle, carried up by the elongation of the spongy stalk ; the ruptured volva at the base. *K*, deliquescence of the gleba, the basidiospores being held in the viscid, foetid drops which attract carrion flies (× ⅓) (after Fischer, from Engler & Prantl)

the air with dust from infected fields; of blister blight (*Exobasidium vexans*) on tea in India; of cacao witches' broom (*Marasmius perniciosus*) in South America and the West Indies; of several parasites of trees and of ornamental plants, and of many others. In general it may be said that if human agency be excluded, dispersal within a new area is gradual and continuous, each new generation extending the area by a rather limited distance. This happened when the vine mildews and that of the oak were introduced into Europe from America, though the latter at least seems to have had some jumps of about 100 miles, and also with several diseases such as chestnut blight (*Endothia parasitica*) introduced into America from abroad.

Some spores are disseminated largely with the seeds of the plants on which they grow. This is the chief means of spreading bunt of wheat and various other diseases against which seed disinfection is practised (see p. 242). Soil fungi are carried by irrigation water or surface-wash after heavy rain and are also liable to be distributed by farm implements, or plough cattle, or on the feet of farm workers.

The dissemination of the spores of certain fungi by insects has been noticed by botanists for very many years. Basidiomycetes belonging to the Phalloid or stinkhorn group were particularly marked examples (Fig. 79 F-K), for their strong and penetrating odour attracts carrion and other flies, and the special adaptation of this group for insect dissemination was early suggested and subsequently fully established [19]. The sweet honey-dew in which the spores of some fungi are extruded has also long been known to attract insects. These visit the '*Sphacelia*' stage of *Claviceps purpurea* (Part II, p. 445), the cause of ergot of rye, and carry spores away not only as an external contamination but also in the alimentary tract, through which they can pass uninjured. The more recently discovered function of insects in securing the fertilisation of the rusts has been mentioned above (p. 42).

Naturally, however, it is the rôle of insects in the spreading of parasitic diseases that has attracted most attention in modern times, and its importance is now fully recognised [31]. Insects are the main agents of dissemination of virus diseases, their activity as vectors of which is discussed in Chapter VIII, on these diseases. They also play a considerable part in the spread of bacterial diseases, such as fireblight (*Bacterium amylovorum*) in orchards, where they are attracted to the gummy exudate from twig cankers in the spring and carry the bacterium-laden matter to the blossoms and young growth of healthy apple and pear trees; the honey-bee is considered to be one of the chief agents in initiating the blossom

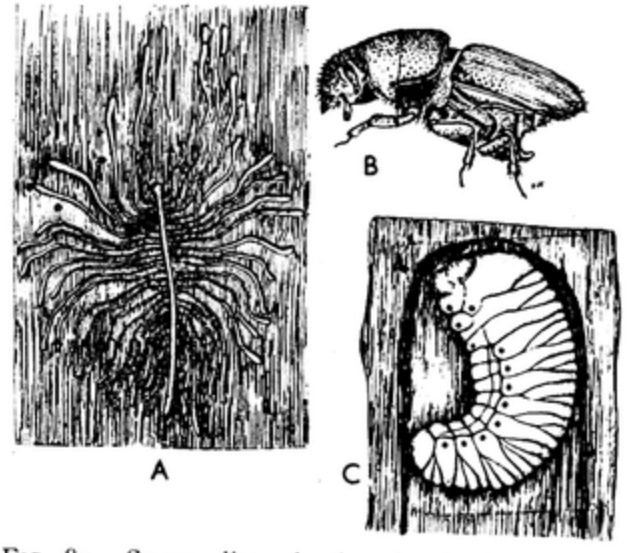


FIG. 80.—Spore dissemination by boring insects. Dutch elm disease (*Ceratostomella ulmi*). *A*, egg galleries and larval mines of the smaller elm bark beetle *Scolytus multistriatus* on the surface of the wood. *B*, the adult beetle (×8). *C*, a pupa (adapted, after Welch, *Cornell Ext. Bull.* 290)



FIG. 81.—Dutch elm disease. Emergence holes made by adults of the smaller elm bark beetle, natural size (after Jones, Lft. 185, U.S. Dept. Agric.)

blight form of infection, and the discovery that the organism is able at times to persist in the hive was followed by an embargo being placed on honey from infected countries in some places still free from the disease. Some of the insects concerned are able to inoculate healthy trees by puncturing the young growth. The organism that causes bacterial wilt or Stewart's disease of maize, *Xanthomonas stewarti*, has been found to over-winter in hibernating *Chaetocnema pulicaria* flea beetles, the chief vectors of the disease in the United States, and to be inoculated into the growing crop by these insects in the spring. Cotton stainers of the genus *Dysdercus*, and some other insects, are responsible for the introduction of the minute fungi *Nematospora* and its allies into young cotton bolls, where they induce internal boll disease; the insects live in between successive cotton crops on various (mostly wild) plants and trees, of which the baobab is one of the most important in Africa, but it is not yet known how or where they harbour the fungus, though the evidence suggests that the association is close.

In many cases insects that cause wounds, such as those that bore into stems or gnaw the softer parts, carry parasitic bacteria or fungi on their bodies and serve to spread infection. The Dutch elm disease (*Ceratostomella ulmi*) (Figs. 80, 81) is mainly disseminated by bark-boring beetles of the genus *Scolytus*, and the water-mark disease of willows (Part II, p. 888) seems to be spread by several different kinds of insects. The black rot, and soft rot of cruciferous plants, due to *Xanthomonas campestris* and *Bacterium carotovorum* (Part II, p. 553), respectively, are also carried passively by insects and other small animals such as slugs. It is easy to demonstrate how readily some of these organisms are picked up by allowing an insect from a heavily diseased plant to pass across the solid surface of some suitable nutrient preparation and observing the colonies of micro-organisms that subsequently develop along the insect's track.

The Nutrition of Fungi

The spore, on germination, gives rise to a germ-tube, which at first is nourished by reserve protein, carbohydrate, and fat stored up in advance. In many cases the fat reserves are more prominent than those of a protein or carbohydrate nature, being often very noticeable as oil drops in the ripe spore. Very soon the young hyphae begin to feed for themselves. The organic food they require must be dissolved before it can be taken in, because solid particles cannot pass through the cell membranes. This food in solution reaches the interior of the hyphae by a process of absorption. Unlike the green plants, fungi cannot build up their carbohydrate nutrients by photosynthesis from the carbon dioxide of the air, for they lack the chlorophyll which makes this possible. In this respect the chlorophyll-free plants are like animals requiring their carbohydrates to be ready prepared by having been built up to form the bodies of other plants or animals. They are usually capable, however, of modifying carbohydrates and other organic compounds by enzyme action so as to make them more readily assimilable.

Inorganic substances — mineral salts and elements like nitrogen — are as necessary in the nutrition of fungi as in that of higher plants and animals. Most of these, however, occur in combination in the organic substances on which fungi feed. As with the higher plants nitrogen, phosphorus, and potassium are amongst the most important of these elements, others required in lesser degree being sulphur, magnesium, calcium, and a little iron.

It is nowadays a matter of common knowledge that animals and man require certain accessory food substances — vitamins and the like — in addition to those which satisfy their needs in proteins, carbohydrates, fats, and salts. Fungi seem to have similar requirements, though knowledge of these is not yet far advanced [1a]. Many fungi and bacteria are able themselves to synthesise these substances and even to produce them in excess and excrete them into the surrounding medium [29]. Various examples of the growth- or development-promoting action of one fungus on another have been known for a long time, and most of these can be traced to the liberation of auxins or vitamins from the mycelium of the first of the two organisms concerned. Filtrates of the staled media in which certain bacteria and fungi have been cultivated have the same effect.

Aneurin (vitamin B_1) has been found especially active in this respect. Many fungi are able to manufacture sufficient for their needs or even to produce an excess, but others require an external source of supply. On the whole, this part of the vitamin B complex appears to be the most generally valuable accessory substance required by fungi.

Yeasts grown in pure synthetic media develop slowly until they synthesise a substance originally named ' bios ' but now known in purified form and commercially available under the name ' biotin '; when biotin or various fungal or bacterial filtrates or organic substances (e.g. yolk of egg) containing it are added, growth is considerably stimulated. A good many other fungi are now known to respond by increased growth when biotin is made available to them; some samples of the agar used for solidifying culture media contain it in considerable quantity.

Sometimes two fungi can be grown successfully in mixed culture in synthetic

media when either alone makes little progress [18]. Thus, *Polyporus adustus* and *Nematospora gossypii* do well together, the former supplying biotin which is essential to *N. gossypii* and the latter aneurin without which *P. adustus* virtually fails to grow. Similarly the red yeast *Rhodotorula rubra* can grow together with *Mucor ramannianus* in a synthetic medium in which neither alone succeeds. Both biotin and aneurin can be synthesised by certain fungi from pure dextrose, amino-acids, and inorganic salts [33].

According to the manner in which fungi obtain their organic food, they are divided into two great classes : Saprophytes and Parasites.

Saprophytes obtain their organic food from the dead tissues of animals or plants, or of substances derived from them. Hence they are found in large numbers in the soil, pervading every bit of rotten leaf or twig or root or every particle of manure. They grow as moulds on foodstuffs, and in the tropics on boots and the backs of books in the rainy season. Every bit of old timber in the forests is liable to be permeated all through with their hyphae. As already mentioned, they share with the bacteria the rôle of nature's scavengers, often having a specified part to play in preparing refractory material, such as the skeletal parts of plants, for bacterial decomposition. In breaking down the walls and contents of plant cells, they liberate carbon dioxide which becomes available in the air for the carbohydrate nutrition of green plants, hydrogen which forms water, ammonia, and so on.

Parasites obtain their food from the living tissues of animals and plants. While, therefore, many saprophytic fungi play a useful part in nature as scavengers, the parasites are almost entirely hurtful. The few cases of beneficial parasites, from the point of view of man, are those which attack noxious insects and occasionally destroy large numbers of them. The great majority of parasitic fungi feed on living plants, those useful to man as much as, or more than, the weeds or useless kinds. A living plant on which a fungus feeds is often termed a ' host plant ' for that fungus. There is scarcely a plant of our fields, gardens, and forests which does not serve as a host plant for one or more species of fungi.

Several different classes of parasites may be distinguished. *Obligate parasites*, for instance, pass through the whole of their life-history on living plants, and cannot be grown on dead or artificial food material. Such are the rusts, the powdery mildews, some Ascomycetes, and other fungi. *Facultative saprophytes* are those that feed usually on living tissues but can, at need, pass through a part of their lives as saprophytes. Often their full development is only reached on the living plants and they cannot be got by artificial cultivation to complete the whole of their life-cycle. The smuts furnish examples of this, as they can often live by budding out little conidia for a considerable time in soil, but usually only grow to maturity and produce their perfect spores on a living plant. *Facultative parasites*, on the other hand, are those which usually grow on dead or decaying matter and are capable of passing through the whole of their development as saprophytes, but have at the same time the faculty of attacking living tissues under certain conditions.

Within these main divisions, parasitic fungi may show every degree of adaptation to the parasitic mode of nutrition. At the lowest end of the scale are certain

facultative parasites, which may be called ' weak parasites ', as they become parasitic only occasionally, when the host plant has been weakened in its vitality by some harmful agency. Plants not yet acclimatised in a new locality, or suffering from insect attack or mineral deficiency, or grown in insanitary conditions such as overcrowding, too dense shade, unsuitable or badly drained soil, and the like, are particularly liable to become available as food to these fungi that are normally saprophytic. Cases of the sort have been recorded from time to time, though few have been adequately investigated; for instance, *Diaporthe perniciosa* and other fungi to which the die-back and bark canker of fruit trees in the south of England were at one time attributed are now believed to attack only trees weakened from other causes. It is, perhaps, as profitless as it is impossible to draw a hard-and-fast line between saprophytism and parasitism in some of the fungi; even in the commonest of all species, *Cladosporium herbarum* (Part II, p. 387) on fading green plant parts, there are too many records of injury from its action to deny it all possibility of behaving as a weak parasite under certain conditions.

In many cases, particular tissues of a plant are normally of low vitality and are readily available as food for weak parasites. Such are the gorged cells of many pulpy fruits, oranges, cherries, apples, and the like, and also the older living wood of trees.

It is probable that the fungi termed ' wound parasites ' are limited in their aggressiveness by some nutritional failing. At first these live only on the dead and rotting tissues caused by a wound or injury to the plant. Later on they begin to spread to the undamaged cells farther in, and they may extend through a large area of the living tissues, eventually, perhaps, killing the whole plant. It may be that during the period of saprophytic life the enzymes of the fungus are produced in quantity and concentration sufficient to enable them to attack living tissues exposed to their action, and transform the walls and cell-contents into nutrient material which the fungus can utilise. If this is so, an analogy can be drawn with the action of decaying petals in promoting infection by *Botrytis cinerea*. That enzyme production can be greatly modified by nutrition has been fully established in this and other fungi (p. 130).

The distinction between parasites and saprophytes cannot be maintained in a few fungi which are able to attack perfectly sound tissues of healthy plants should they come in contact with a suitable host, but in the absence of the latter can live and develop freely on dead materials. Such a case is the ' damping off ' fungus *Pythium de baryanum* (Part II, p. 579), which is present as a saprophyte in almost every garden in some parts of Europe, and at once attacks and destroys seedlings of cress and certain other plants, should they be grown in the soil which contains the fungus. Neither neglect of cultivation nor unsuitability of soil predisposes to this disease; wounds are not necessary for the entry of the parasite; and, if anything, damping-off is worse on rich soil than elsewhere. Several parasites of trees, e.g. the common bracket fungus on elms, *Polyporus squamosus* (Fig. 34), can continue to live on the dead wood as saprophytes; these, however, are usually wound parasites of a less aggressive type than the damping-off fungi. On the Aleppo pine in France, *Trametes pini* (Fig. 434 H, I) dies with the tree, but *Fomes pinicola* continues to rot the fallen timber.

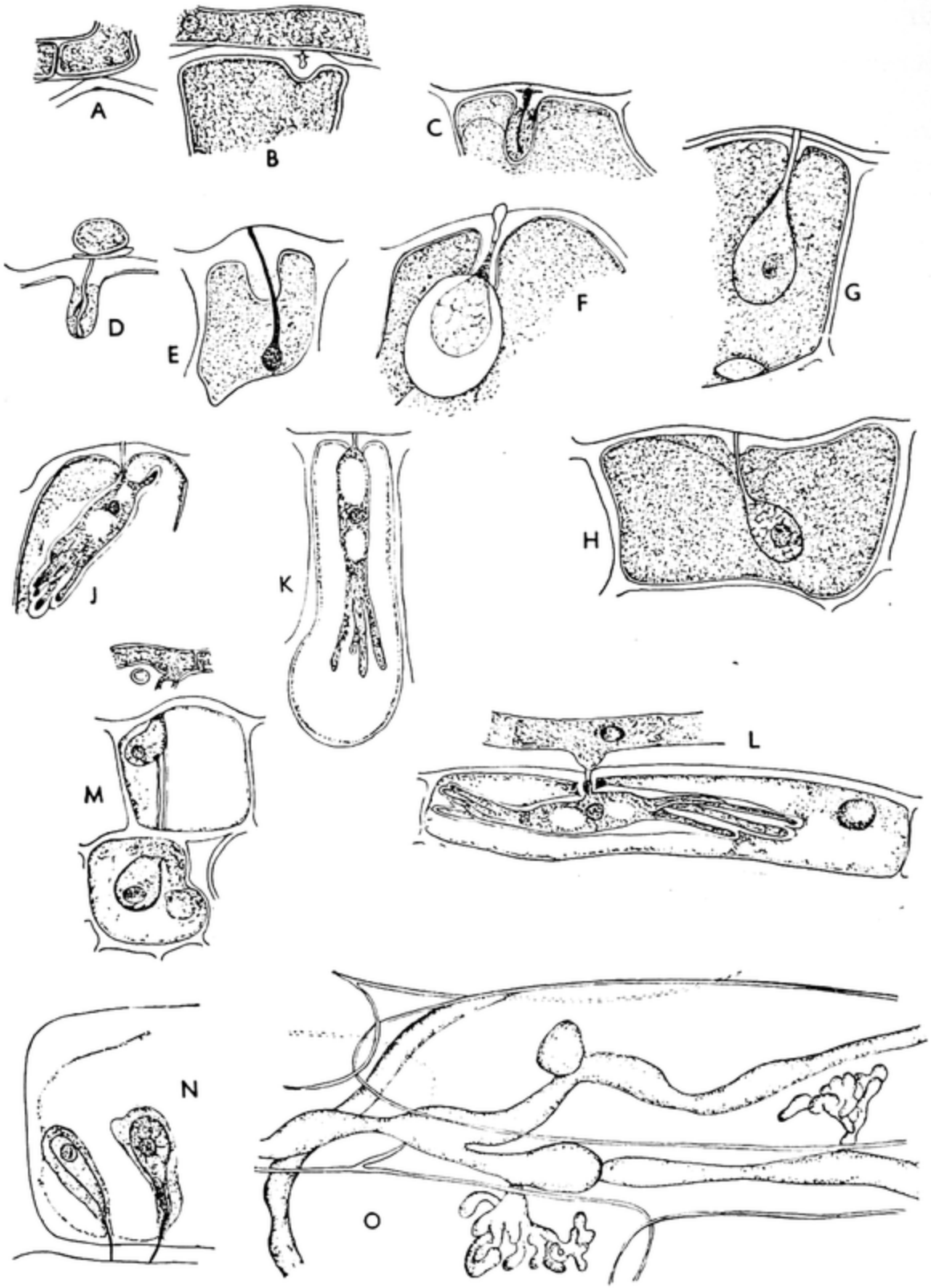


FIG. 82.—Types of haustoria. *A–L, Erysiphe graminis. A*, first stage of penetration, marked by a spot on inner side of host cell wall. *B*, penetration tube making its way through epidermal wall. *C*, further stage in penetration; cellulose papilla on inner wall elongates and the tube grows. *D*, distal end of penetration tube enlarging. *E*, papilla bored through, the haustorium developing without a sheath. *F*, stage before entrance of nucleus of haustorium; stretched and intruded plasmic membrane of host cell forms a boundary-membrane or sheath which, in this case, is devoid of contents. *G*, haustorium without a sheath. *H*, haustorium without sheath, but protoplasm of host cell adheres to side of neck. *J, K, L*, types of the lobed haustoria on *Poa*. *M*, *Uncinula salicis*, on *Salix discolor*. The epidermal cell contains one haustorium,

The Mechanism of Feeding by Parasites

Parasites reach their food either by sending their mycelium into the interior of the host plant or by remaining for the most part superficial and sending only suckers or a limited growth of feeding hyphae into the surface tissues. Usually in the internal parasites (' endophytes '), e.g. *Rhytisma acerinum* (Fig. 5), the feeding hyphae penetrate directly into the cells of the host, the cell sap and more solid contents being taken up by absorption after the latter have been brought into solution by the enzymes of the fungus. Often, however, the earlier stages of feeding are marked by the dissolution of the cell walls of the host by enzymes such as pectinase and cellulase ; these walls can provide many fungi with much of the carbohydrate food that they require. In a few fungi, such as the *Exoascaceae* (Fig. 143), the mycelium is wholly intercellular, without any intracellular parts, so that absorption of nutrient solutes must occur through the membranes of the host and parasite cells ; this is probably a common occurrence, for even when the cells are entered much of the mycelium may remain intercellular and probably receives its food directly from the surrounding cells. It was at one time believed that some of the superficial parasites (' ectoparasites ') of leaves and twigs could similarly feed by absorption from the epidermal cells across the outer wall of the cell and the cuticle, but in most of these fungi, feeding hyphae or suckers have now been found to enter the cell cavities ; this occurs, for instance, in the light-coloured thread blights (mostly species of *Corticium* or *Marasmius*) and in the dark *Meliolaceae* or *Asterineae*. In the ectoparasite of some European pine trees, *Acanthostigma parasiticum*, it is stated that the white mycelium which forms cushion-like layers on the under surface of the needles obtains its food through tiny rod-shaped processes which penetrate through only the cuticle and not through the inner (mainly cellulose) parts of the outer wall of the epidermal cells. As in the subcuticular parasites such as *Taphrina rhomboidalis*, absorption of nutrient solutes must occur through the subcuticular membrane of the needle leaf.

Haustoria

Many intercellular parasites that apparently find difficulty in getting adequate nutrition by absorption of solutes into their hyphae from the cavities of the host cells send into the cells specialised hyphae which act as feeding organs. These are known as ' haustoria '. Haustoria occur in all the main groups of fungi, usually but not exclusively in obligate parasites with an ectoparasitic or an intercellular mycelium. They vary in shape from the knob or sac-like type found in *Cystopus* (Fig. 75) to simple or lobed or branched or coiled hyphae in many *Erysiphaceae* Phycomycetes and rusts, or coralloid types as in *Peronospora schachtii* on beet (Figs. 82, 84) [1]. Some of the complex forms almost fill the cells they occupy and can hardly be distinguished from an intracellular mycelium, as in

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while from the same infection-hypha another penetration tube passes through the epidermal cell into a cell below (the sheath is not visible) (× 600). *N*, *Erysiphe cichoracearum*, on *Eupatorium perfoliatum*. Part of cell of a hair showing two haustoria (× 1300, all other figures above, × 1200) (after Grant Smith, *Bot. Gaz.*). *O*, *Peronospora schachtii*, on beet, showing mycelium and lobed, coralloid haustoria (× 500) (after Singalovsky, *Ann. Epiphyt.*)

Peronospora parasitica (Fig. 83). Their shape is not very constant, as it may vary to some extent according to the tissue invaded.

The haustorium usually enters the cell by an exceedingly fine hypha resembling the infection hypha arising from an appressorium by means of which, as will be seen later, many parasites enter the outer membrane of the host (Fig. 82). After entry the haustorium often grows towards the nucleus of the host cell (Fig. 84), but while, at first, it may not enter the ' primordial utricle ' or peripheral plasmatic membrane of the protoplast, only pushing it in and invaginating it, the utricle may finally be pierced by it [52a]. In the early stages the haustorium is bounded

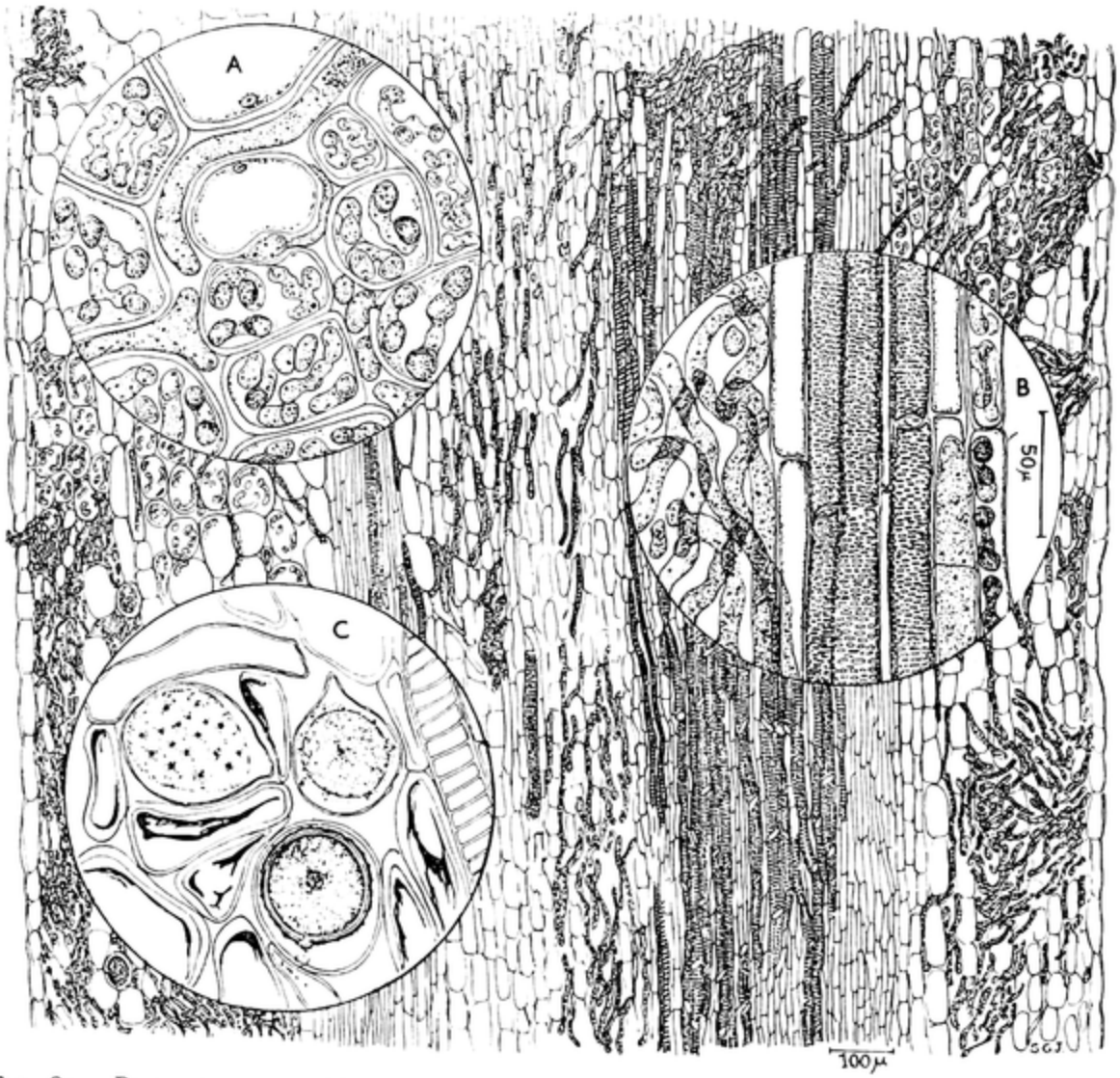


FIG. 83.—*Peronospora parasitica* in the floral axis of shepherd's purse (*Capsella bursa-pastoris*). Longitudinal section showing the intercellular mycelium, chiefly in pith and cortex. Lobed haustoria are frequent in the cortical cells, amounting almost to an intracellular mycelium. The right half of the section has passed largely through a vascular bundle; the left half between the bundles. Insets: *A*, the coiled haustoria in the cortical cells; note also intercellular hyphae. *B*, the intercellular mycelium in the vicinity of the pith, where there is much destruction of the tissues; the xylem vessels are not entered by the fungus; on the right, coiled haustoria in the xylem parenchyma. *C*, the sexual organs in the intercellular spaces of the cortex; multinucleate oogonium on the left; on the right, all the nuclei of the oogonium, except the egg nucleus, are at the periphery of the ooplasm; below, a ripe oospore showing remains of a fertilisation-tube and an empty antheridium beneath it

by a thin limiting membrane, and this sometimes persists. Frequently, however, various thickenings occur around the point of entry or deeper in. When developed around the narrow style-like stalk of the haustoria of *Erysiphe graminis* [12a], or many rusts, the thickening is continuous with the inner wall of the cell (Figs. 82 C, 111) and is closely similar to the infection pegs (Chapter IV) found where a parasitic hypha enters a cell. It is sometimes effective in checking penetration. When

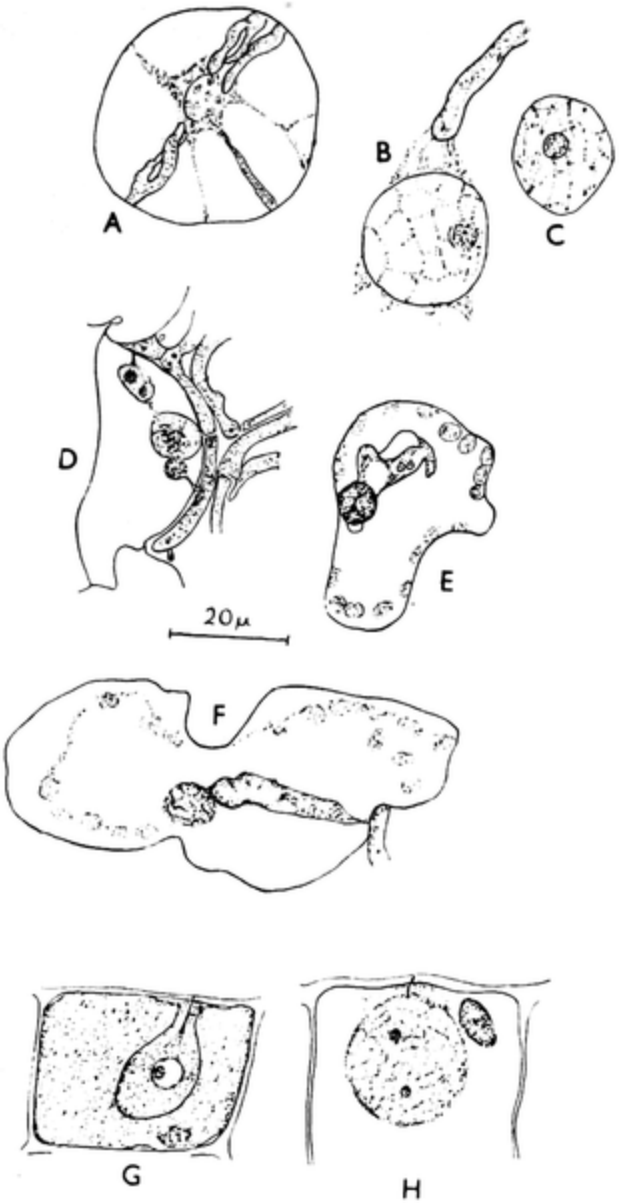


FIG. 84.—Apparent relation between host nuclei and fungal haustoria in penetrated cells. *A*, a living cell from the cortical parenchyma of the petiole of hollyhock attacked by *Puccinia malvacearum*; haustoria are seen lying in the protoplasmic strands, and the disorganising chloroplasts are aggregated around the enlarged nucleus (×650). *B*, the same, showing the connection between the haustorium and the host nucleus by fine strands of protoplasm; the nucleus has lost its chromatin granules (×1800). *C*, the nucleus of an unaffected cell, corresponding to that in *B* (after Robinson, *Manch. Mem.* 57). *D–F*, haustoria of *Puccinia anomala* on barley. *D*, epidermal cell with its nucleus pulled in two directions by two haustoria. *E*, formation of a vacuole at the point of contact between haustorium and nucleus. *F*, haustorium in a mesophyll cell in a nine-day-old infection, in contact with nucleus (after D'Oliveira, *Revista Agron.*). *G*, *H*, *Podosphaera leucotricha* on apple. *G*, uninucleate haustorium. *H*, binucleate haustorium with host nucleus close by (×750) (after Woodward, *Trans. Brit. Myc. Soc.*)

found around the body of the haustorium it is often later in appearing and may, in old haustoria, be a means of walling off the intruder, or a consequence of excessive drain on the cell contents, as in tubers attacked by potato blight (p. 516); as injury to the cell from over-virulent action of the fungus increases, so the haustorial cover thickens. There is little doubt that these thickenings are products of the host cell, and indeed it is sometimes possible to distinguish two layers in the sheath, one being the membrane of the haustorium and the other a covering deposited by the host. The latter seems to be usually cellulose, but there may be some lignification of the older sheaths. Callose is stated to occur sometimes between the two layers or in the exterior one. The haustorium itself may have from one to many nuclei. In some smuts the haustoria are typically binucleate, but those of the downy mildews may sometimes have only one nucleus though others have several, as in the coenocytic hyphae from which they arise.

Most of the haustorial fungi are obligate parasites, but some, such as the Phytophthoras and *Actinonema*, can be grown in artificial culture. It is probable that many of the intercellular hyphae are not wholly dependent for their nutrition on the haustorial apparatus, but, like other fungi occupying a similar position, can get some nutrition by absorption from the surrounding cells. While, therefore, the haustorial habit is not a strict criterion of status as an obligate parasite, it suggests a high degree of specialisation in some (presumably nutritional) requirement which ordinarily it has not proved possible to supply in culture. It marks increased adaptation to the parasitic life over that shown by the destructive action of parasites such as *Pythium* and *Botrytis*, for one of the characteristic features of many of the haustorial fungi, especially the rusts and smuts, is the absence of any gross indication of injury to the protoplast of the host cell for a period which may sometimes be almost equal to the lifetime of the plant. This aspect of their parasitism will be referred to again in a later chapter.

The Enzymes of Fungi

In the preparation of their food for absorption fungi, like animals and man, rely on enzymes or ferments, of which they may possess a formidable array. Among the hydrolytic enzymes detected in *Polystictus abietinus* are cellulase, pectinase, ligninase, diastase, sucrase, inulase, tanninase, pepsin, trypsin, emulsin, and lipase, while the oxidising enzymes of this fungus include catalase, peroxidase, and laccase [20]. In *Aspergillus oryzae* diastase, invertase, maltase, lactase, protease, rennet, and lecithinase have also been recorded. Many of these enzymes are excreted into the surrounding medium, sometimes in considerable quantity and capable of utilisation on a commercial scale [47]. Apart from the use of yeasts in brewing, distilling and baking, diastase from fungi is used medicinally and in various industrial processes; acetone can be formed by *Mucor rouxii* and other fungi from sugar-yielding plant products; citric acid is now mainly a fermentation product of *Aspergillus niger*, for which one American firm some years ago kept nine acres of mycelium in constant commission; yeasts, Mucors, Aspergilli, and bacteria are all employed singly or in various combinations in the East and in Africa for the preparation of various ' beers ', fermented milks, non-alcoholic

drinks, and seasonings; while gallic acid, which is used in dyeing and as a constituent of inks, has been commercially produced by the fermentation of tannins. The proteolytic enzymes of *Aspergillus oryzae* and its allies are used in the Far East in the preparation of readily digestible substances from the protein-rich soy bean. Not less important are the processes of fermentation carried out during metabolism within the hyphae. During the period of stringency of fats in Germany about 1918 more than a million kilograms of glycerine were being produced monthly from fungi, and in Russia *Endomycopsis vernalis* was used experimentally for the same purpose some years later. Molasses, beer-wort, potatoes, hydrolysed wood pulp, or the like, together with salts of nitrogen and phosphorus, provide the culture medium for the fungus. Some moulds are even more effective in synthesising fat as a reserve nutrient in their mycelium than the yeast-like *Endomycopsis*; *Penicillium javanicum* can store up to 41·5 per cent. of fat in its hyphae when grown on glucose and salts. Yeasts and their allies are also able to manufacture high-quality proteins, with vitamins, from the same raw materials, and some 20,000 tons of dried yeast, containing about half its weight of protein, were annually produced in Germany during the first great war. Probably the best species for this purpose hitherto tested is *Torula utilis*. These examples, taken from only a few amongst many, indicate that both by means of the enzymes excreted from the mycelium and by those concerned in metabolic processes within the hyphae fungi can synthesise a remarkably wide range of substances from organic materials. Many have been identified and they represent almost every type of compound known to organic chemistry.

The enzymes are organic catalysts which, unlike the inorganic ones, are destroyed by heating. Hydrolysing enzymes split up disaccharides such as sucrose, maltose, lactose, and the like into two similar or dissimilar molecules of monosaccharides —dextrose (glucose), levulose (fructose), mannose, galactose, etc. Cane sugar, for instance, is readily ' inverted ' by invertase from fungi, giving one molecule of glucose and one of levulose, while maltase gives two molecules of glucose from maltose. The hexose monosaccharides, especially glucose, appear to be readily available to fungi; on condensation (the reverse of hydrolysis) they give hexosan polysaccharides (starch, cellulose, etc.). The pentose monosaccharides, such as xylose and arabinose, condense with other sugars to form pentosans — polysaccharides common in pectic membranes, gums, mucilages, and the like; acted on by the pectinase of fungi, they give galactose and other bodies. On splitting up, the polysaccharides give more than two molecules of monosaccharides. The proteolytic enzymes of fungi, pepsin, trypsin, erepsin, etc., change the less soluble proteins into the more soluble and readily diffusible peptones and amino acids. Tannase forms glucose from tannin and lipase, glycerol and higher fatty acids from fats. Certain moulds can grow in the waste liquor from the manufacture of sulphate wood pulp, decomposing the lignin present as chemically combined lignosulphonates [32].

The oxidative processes of certain bacteria provide energy enabling them to synthesise carbohydrates when carbon dioxide is their sole source of carbon, although they have no chlorophyll and light is not needed for the purpose. Such are the nitrifying bacteria, *Nitrosomonas* and *Nitrococcus*, the sulphur bacteria

Beggiatoa and *Thiothrix*, and the iron bacillus *Spirophyllum*. The oxidase of strains of the common mould *Aspergillus niger*, can oxidise glucose to gluconic acid directly. Similarly some yeasts can directly oxidise maltose, giving off carbon dioxide. Yeasts will grow with the organic salt ammonium tartrate as their only source of carbon. Such examples are sufficient to illustrate the remarkable versatility of the enzymes of bacteria and fungi.

The enzyme armament varies considerably in different fungi. Some can attack chiefly the cell membranes, others the cell contents. The *Peronosporaceae*, *Erysiphaceae*, and rusts, for instance, have little or no action on the membranes, using chiefly some of the cell contents, while those wood-decaying fungi (see p. 211) that affect tissues with scanty cell contents must get much of their food from the walls ; this accounts for the cavities in the older wood caused by certain species. Enzyme production may vary according to the strain of the fungus concerned, as is fully recognised in brewing and other fermentation industries. The conditions under which the fungus grows, especially its nutrition and aeration, also have a marked effect. Thus a strain of *Aspergillus niger* was found to form diastase copiously when grown in media containing starch, glucose, or maltose but not at all when grown on 5 per cent. glycerine. An acid reaction was necessary for optimum production on the enzyme in this case, but pectinase may sometimes be most active in acid, sometimes in alkaline plant juices, and this seems to depend on the presence of other substances which are adsorbed from the medium. It is believed that the pectinase is the same in all of a number of fungi tested (including *Botrytis cinerea*, *Pythium de baryanum*, *Phytophthora erythroseptica*, *Sclerotinia fructigena*, etc.), but its properties depend on its exposure to the action of other substances [39], while whether it is produced at all and the quantity produced, depend on the medium in which the fungus is grown (see also below, p. 129).

Some fungi can cause notable rises in temperature in decomposing plant material. Thus *Aspergillus fumigatus* has been found to raise the temperature of oat straw inoculated with it from 25° to nearly 55° C., in 38 hours. The most extensive decomposition of straw has been obtained by a mixture of soil Actinomycetes and fungi. Most of the common soil moulds *Aspergillus*, *Penicillium*, *Trichoderma*, *Rhizopus*, and so forth utilise the carbohydrates cellulose, hemicellulose, pentosans, and mannan readily and rapidly in decomposing plant material but not lignin ; certain Basidiomycetes, however, such as the common edible mushroom, do not make use of hemicelluloses but deplete the lignin and protein, while causing also a loss of cellulose. Some of these fungi can decompose humus [11].

Effect of Nutrition on the Structure and Life-History of Fungi

All saprophytes and many parasites can be grown on artificially prepared food, or ' culture media '. The kind of food supplied may have a profound effect on the structure and reproduction of the fungus. With such plastic organisms, however, generalisations are difficult and the most that can be said is that a particular effect is obtained with a given strain of the fungus grown under a certain set of conditions. Other strains and other conditions may give different results.

In some fungi, as in higher plants, it is possible to enhance vegetative vigour at the expense of reproductive activity by nutritional treatment. Thus, the aquatic fungus *Saprolegnia mixta* tends to produce a vegetative mycelium only, without any spores, when supplied with abundant food regularly renewed. If such sterile, strongly growing cultures be transferred to water, or to a weaker food solution, sporangia with zoospores are formed in quantity. Transferred to a solid substance of little nutritive value, such as plain agar, sexual oospores are alone produced [27]. In early studies of *Pythium ultimum* (p. 507) only oospores were produced on cabbage leaves whereas on other substances sporangia were formed [55]. The mycelium of various common moulds (*Mucor*, *Penicillium*, *Aspergillus*, *Alternaria*, etc.) is quite different in structure with different food supplies; in some of the Mucors it may be changed wholly or in part from the filamentous to the gemmate condition. Nutrition may determine the proportion of aerial to submerged mycelium or the formation of coremia (mycelial strands) in place of single hyphae, or even of sporodochial erect clustered hyphae instead of a recumbent growth. In some genera (e.g. *Fusarium*, *Actinomyces*) the colour of the mycelium may vary according to the composition of the medium on which it grows and also according to the *p*H of the latter [58]. Such effects are the commonplace of all mycological laboratories and account for the caution shown in attempting to identify fungi from mycelial characters alone. As already mentioned, the morphology of the spores and their method of attachment to the sporophore are much more reliable specific characters and are those mostly used. Even here, however, there are difficulties, linked largely with vigour or luxuriance of development. To surmount them, many futile attempts have been made to devise a standard or an ideal culture medium on which fungi would give consistently satisfactory growths. That these must fail in, at least, the considerable number of facultative parasites which show the specialisation of parasitism described in the next chapter is evident, if specialisation is dependent on nutritional qualities of the host, and there is overwhelming experience available to show that even pure saprophytes do not all use the same food and cannot all be given a single satisfactory diet.

Symbiosis

The parasites mentioned so far obtain their food at the expense of the host plant and are not known to give anything in return. The food-supplying cells are usually killed eventually by the excessive demands of the invader, but, as already mentioned, certain obligate parasites, especially those that feed by haustoria and those that form galls, may long defer this critical period and kill only when the production of spores makes a culminating call on the nutritional resources of the host. In many cereal and grass smuts this does not happen until the ear or individual caryopses are developing at the end of the vegetative life of the host. The Ascomycete *Epichloe typhina* (Part II, p. 473) may go to a stage further than this, for it sometimes fails to fructify even when its systemically infected grass host has grown to maturity. The mycelium may be found in the anthers and around the embryo of the grass seed without necessarily destroying the pollen or preventing the seed from germinating. Latent infection may persist for several seasons, the

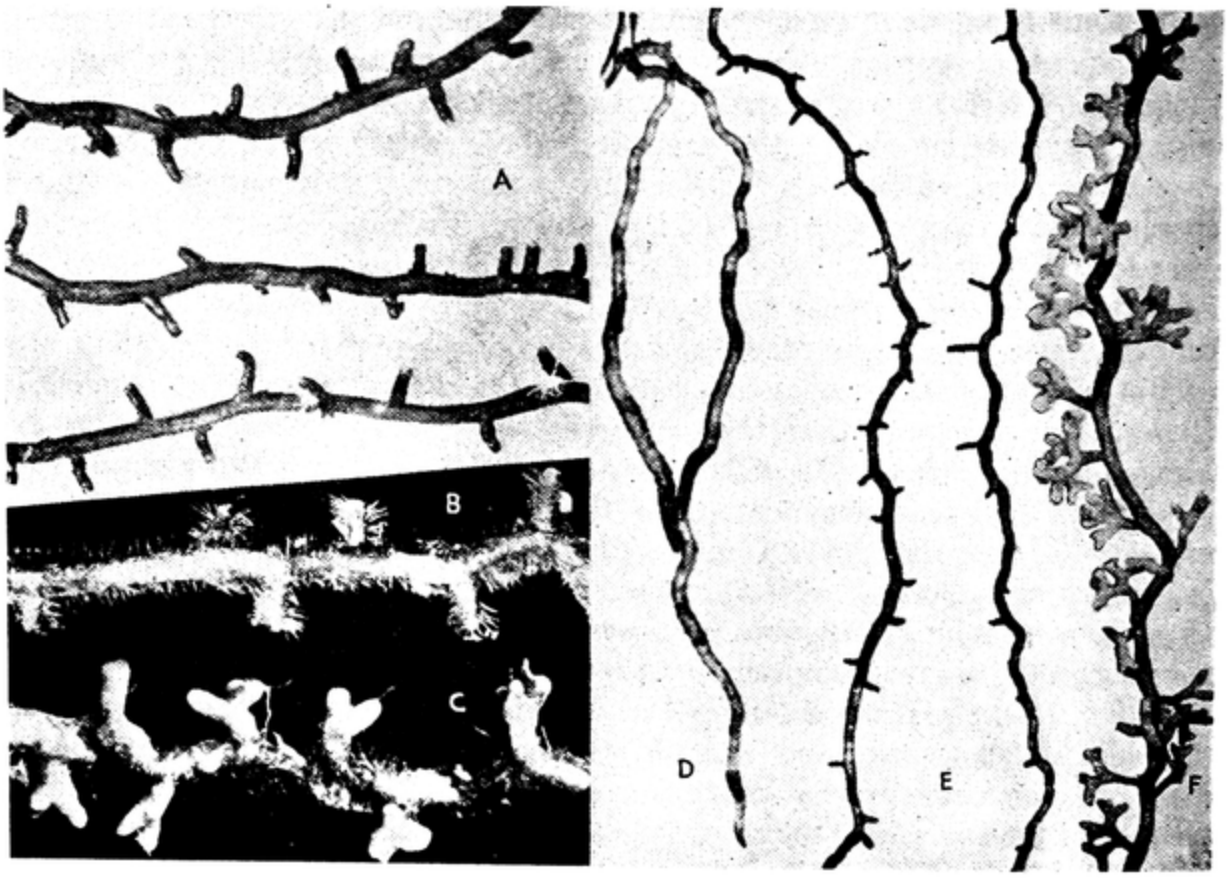


FIG. 85.—Mycorrhiza on roots of pine. *A*, roots without mycorrhiza. *B*, root covered with mycorrhiza (in pure culture). *C*, root covered with mycorrhiza (natural). *D*, *E*, *F*, showing the relation between the rooting environment and the development of short roots in raised seedlings (after Hatch & Doak)

plants sometimes remaining barren, sometimes producing viable seed. Ultimately the conidial stromata appear during a flowering period, which may be in the second or third year of growth ; up to this time no apparent injury is suffered by the host [49]. Perhaps the end point in this progress towards a regulated parasitism is marked by the endophytes which occur at times in various species of *Lolium* (rye grasses, darnel, etc.). Two of these have been found to cause systemic infection of the above-ground parts of the hosts, one difficult to isolate and remaining sterile in artificial media while the second grows readily in culture and produces microconidia : the former develops a dense mycelial stratum between the seed coat and the aleurone layer, the latter is sparser and more difficult to detect. Both are transmitted in the seed, from which they pass to the growing point of the seedling. Neither appears to cause any injury, and their presence in or absence from a particular plant cannot be detected without isolation or microscopical methods [50]. Their identity is unknown, and there is no real clue to the physiological relationships between the host and the parasite (if such terms are justified in what appears to be a perfect state of equilibrium between the two organisms), though there is some experimental evidence which has been considered to indicate that the endophyte assists in the supply of nitrogen to the host by fixing the nitrogen of the air [6].

So far all the advantages seem to lie with the parasites ; the host at best may

be able to escape injury to a greater or lesser degree, or perhaps, in *Lolium*, gain a little nitrogenous food by the aid of the fungus. Some higher plants, however, have progressed beyond this stage and have established a symbiotic partnership with fungi in which a mutualistic relationship is evident. As the association between the fungus and higher plant is chiefly evident in roots, the term ' mycorrhiza ' is applied to it (42).

The best-known mycorrhizas are those of forest trees (Fig. 85) and orchids : these represent the two main types of fungus-root association, the ' ectotrophic ' and the ' endotrophic '. In the ectotrophic mycorrhizas of trees many of the fine lateral rootlets are enveloped in a mantle of fungal tissue which prevents growth in length of the rootlet, causing it to assume a swollen or coralloid form. Some of the hyphae usually pass down between the cells of the outer layers of the root, forming the so-called Hartig net (Fig. 86). The fungi concerned chiefly belong to

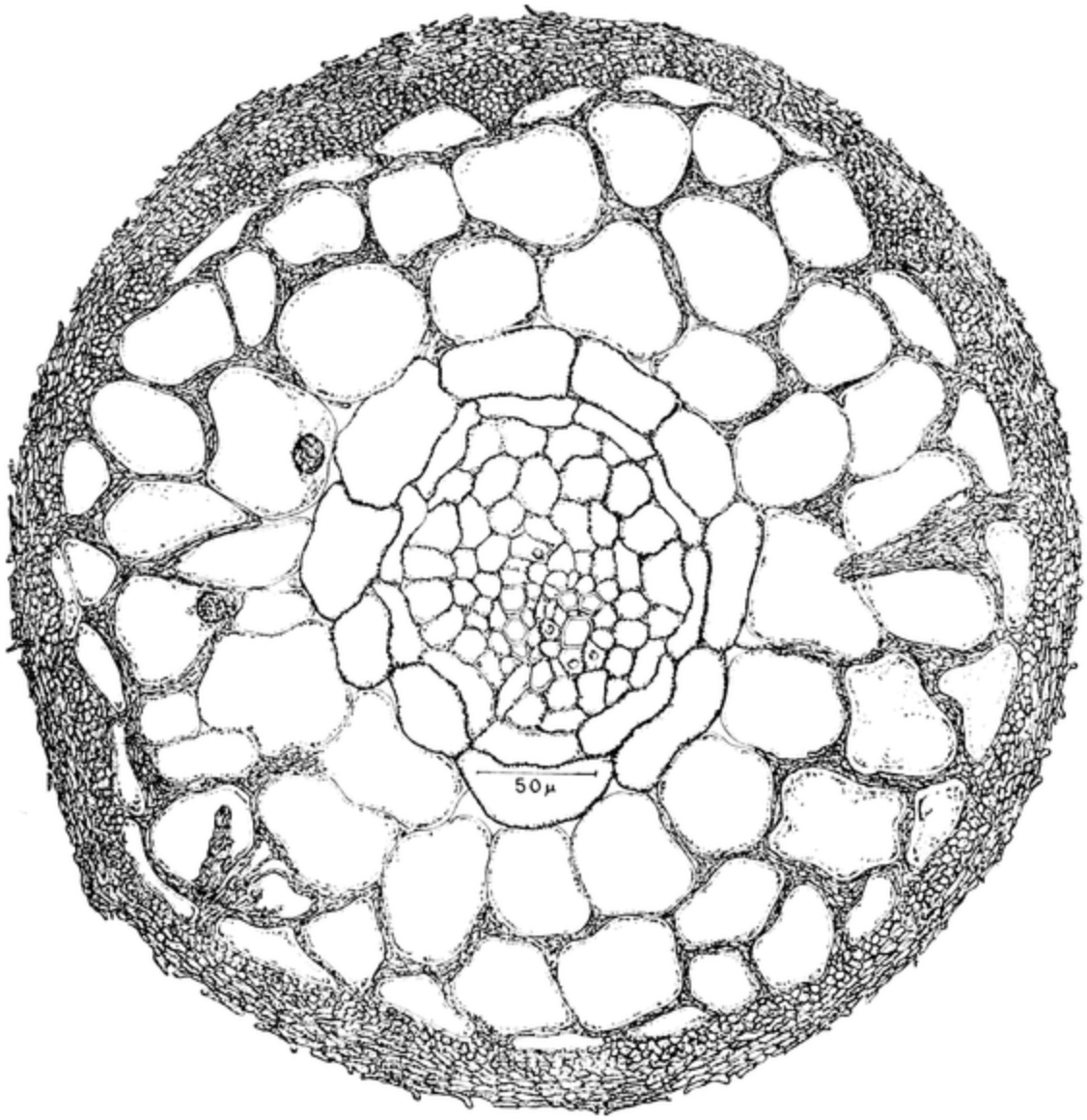


FIG. 86.—Transverse section of a young root of Scots pine showing ectotrophic mycorrhiza forming a compact mantle covering the root, with the fungus penetrating between the cells of the cortical tissue (the ' Hartig net ') (from a slide by Bond)

the Basidiomycetes and include many gill and pore bearing toadstools (*Boletus*, *Russula*, *Cortinarius*, *Lactarius*, *Clitocybe*, *Tricholoma*, *Amanita*, *Paxillus*, and the like), but truffles (Ascomycetes) grow in mycorrhizal association with oaks and other trees and the systematic position of some mycorrhiza-forming fungi is unknown. Field mycologists are well aware of the common occurrence of particular species of fungi under certain trees of which this is, perhaps, one explanation. The mycorrhizal association in the orchids differs widely from that of trees (Fig. 87). The fungi concerned are endophytes, mostly sterile *Rhizoctonia* stages of the genus *Corticium*, though the honey agaric, *Armillaria mellea*, forms mycorrhiza with the tuberous orchid *Gastrodia elata*, in the East. In the *Rhizoctonia* type, the hyphae enter the cells of the cortex of the root, where they form dense coils which eventually break down into an amorphous mass. It has long been known that orchid seeds are difficult to germinate, and the surmounting of this difficulty by providing the seeds with cultures of a suitable strain of *Rhizoctonia* is a good example of the application of science to practice by many orchid specialists, who not infrequently have kept their methods as a closely guarded

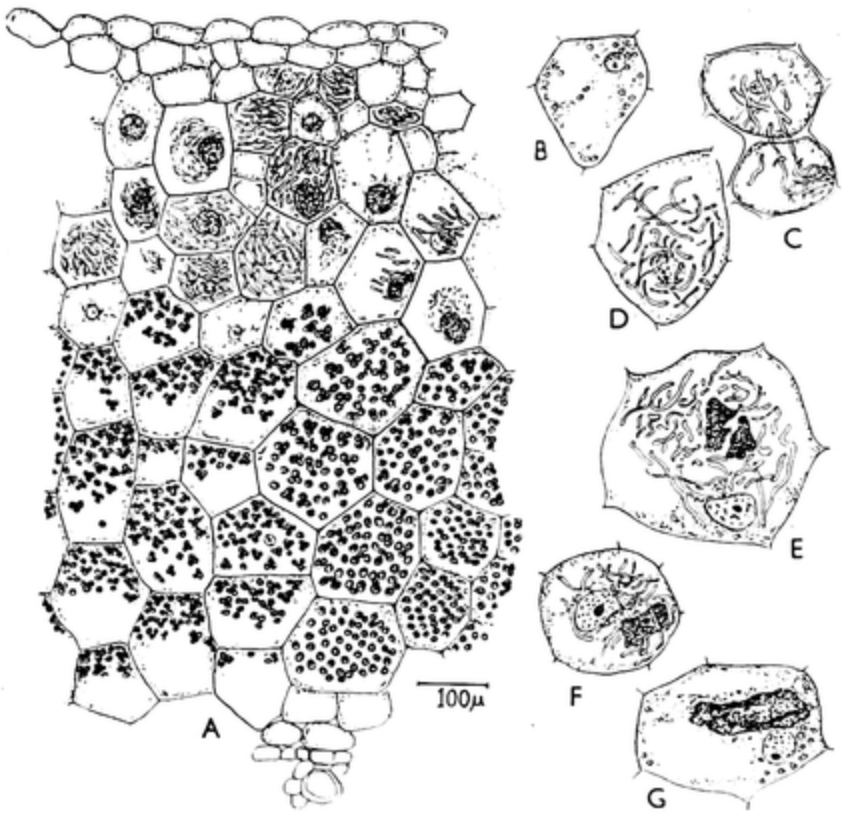


FIG. 87.—Endotrophic mycorrhiza. *A*, portion of transverse section of root of a saprophytic orchid (*Neottia nidus-avis*). The fungus occupies the peripheral tissues, about three or four cells deep; the inner cells contain starch grains. *B–G*, cells of a saprophytic orchid (*Dipodium punctatum*) showing stages in digestion of the mycorrhiza-fungus. *B*, a normal cortical cell with nucleus, cytoplasm, and spherical starch grains (× 200). *C*, two cells showing early infection; the hyphae are clearly associated with the host nuclei (× 83). *D*, aggregation of the hyphae around the nucleus (× 250). *E*, partial digestion of the central hyphae, and the slight enlargement of others around; the nucleus enlarges and stains deeply (× 250). *F*, further stage in the disorganisation of the fungus, nucleus enlarged (× 250). *G*, almost complete digestion of the fungal hyphae; starch grains are reappearing in the cell (× 225) (after McLuckie, *Proc. Linn. Soc., N.S.W.*)

secret. The heaths, in general, resemble the orchids, except that the endophytes belong to the genus *Phoma* and in some cases (e.g. in the common ling, *Calluna vulgaris*) occur as a sparse mycelium throughout the plant as far as the fruit capsules, whence the seed on germination may become naturally infected. In these two families and a few others similarly dependent on fungal infection for satisfactory germination, the seed is small and lacking in food reserves, so that it would appear that the endophytes supply some nutritional requirement. What this is is not definitely known, but the fact that satisfactory germination of orchid seeds occurs without the fungus in certain sugar solutions suggests that carbohydrates are involved. In the heaths a fixation of atmospheric nitrogen has been attributed to the endophyte (as in *Lolium*), but the evidence for this has been questioned.

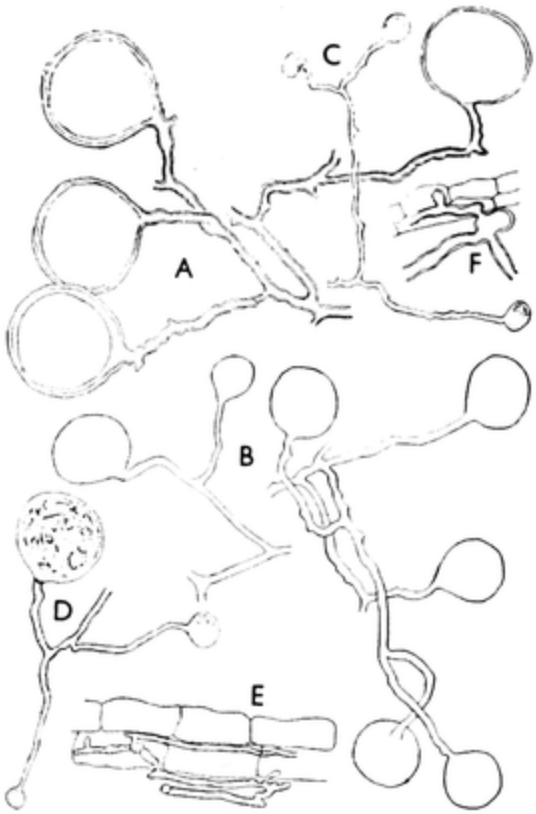


FIG. 88.—Vesicular-arbuscular type of mycorrhiza. *A*, mycelium of *Rhizophagus* sp., bearing four large terminal thick-walled vesicles, from soil around cotton roots (× 240). *B*, two hyphae of same bearing, respectively, five and two, less mature vesicles with thin walls and no basal septum (× 240). *C*, early stage in formation of extramatrical vesicles of same (× 240). *D*, a hypha of same, bearing one older vesicle with a basal septum, and others in earlier stages (× 240). *E*, penetration of cotton root by a hypha from the external mycelium of same (× 240). *F*, old hypha, with thickened, rigid walls, showing mode of entry, with appressorium-like swelling, into *Abutilon* root (after Butler, *Trans. Brit. Myc. Soc.*)

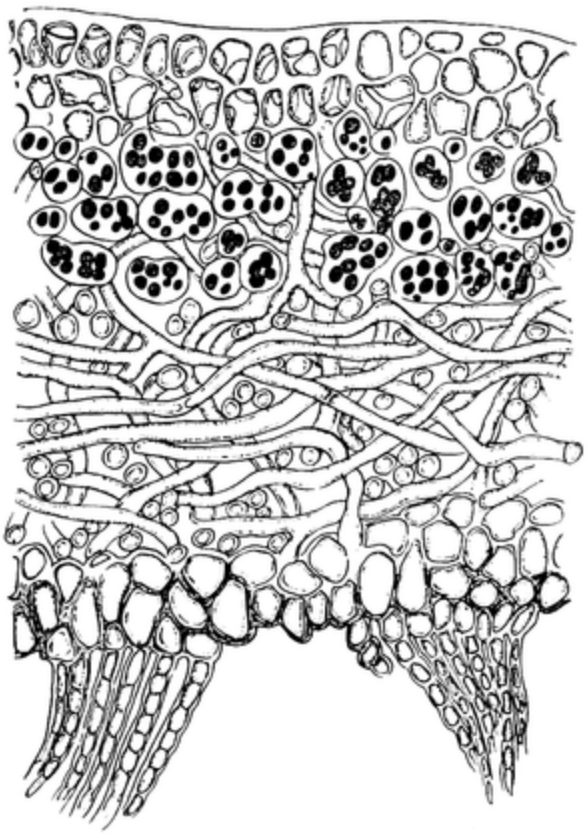


FIG. 89.—Structure of a lichen. *Sticta fuliginosa*. Transverse section of the thallus showing a zone (dark cells) of the blue-green alga *Chroococcus*, covered by a fungal cortex; below, a medulla of loose hyphae, with a second cortex, and fungal rhizoids (rhizines) on the under side (× 614) (after Sachs)

There is much difficulty in accounting for the physiological relationships between the partners in a mycorrhizal association [43-46]. Several converging lines of evidence are strongly indicative

that it began as an attack by a parasite on the roots of a higher plant — the prevalence of infection pegs and other reactions of the host, the occasional tendency (well known to orchid growers) for the fungus to develop destructive capabilities, the peculiar features of the mycorrhizal function of *Armillaria mellea*, are some of these. The eventual reduction of the invading hyphae to a condition in which they are not only made harmless but actually in large part digested by the host cells is easily reconcilable with the view that the higher of the two partners gains nutritional benefits in the process. But even this view, which is far from easy to prove, has been the subject of adverse criticism, with the result that, so far at least as concerns forest trees and orchids, the practical man was for a time more universally ready to accept it than his scientific fellows. Gradually, however, the beneficial effects of mycorrhiza have gained almost general recognition, though there is still a lack of agreement as to their nature. The fungal partner in endotrophic mycorrhiza gains food, often using up all the starch in invaded cells, but beyond escaping the competition of ordinary soil saprophytes, seems to profit little from its colonisation of the roots. The ectotrophic forms seem to have even less chance of benefiting from the association, though it may be presumed that those of their hyphae that grow down between the host cells can absorb nutrient material from the host. There is some evidence that *Boletus elegans* gets a growth-promoting substance from the larch roots with which it forms mycorrhiza [25]. Aneurin stimulates the growth of several mycorrhizal fungi.

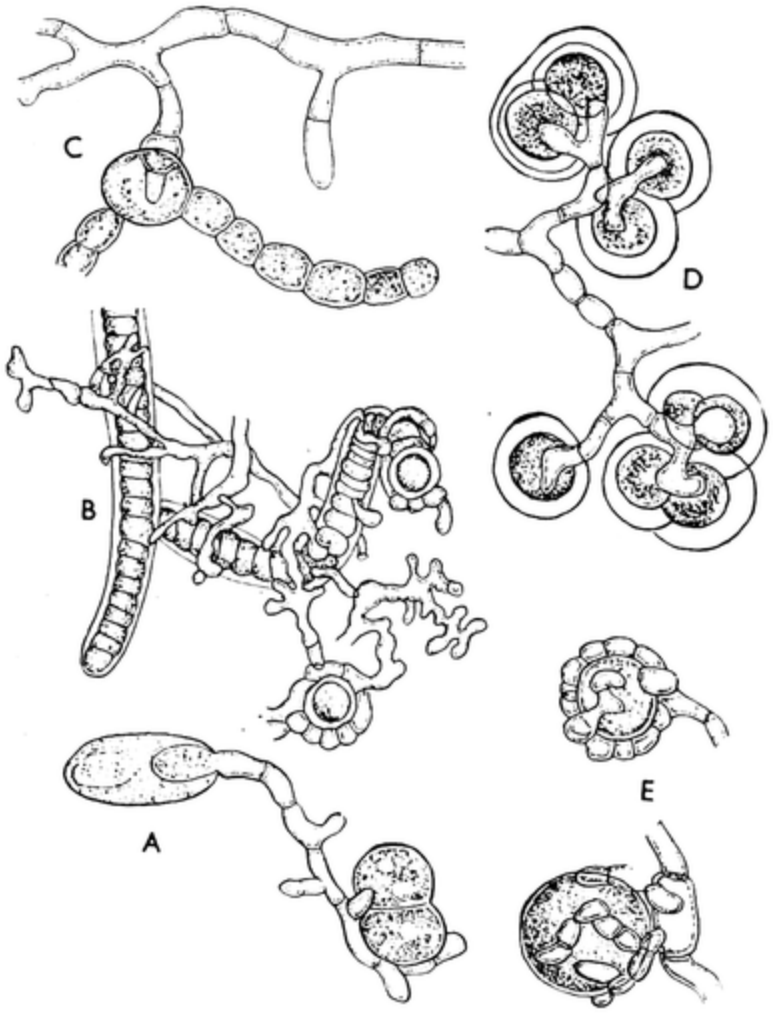


FIG. 90.—Association of alga and fungus in the lichen. *A*, two cells of the alga *Protococcus viridis* invested by a germ-tube from a spore produced by the lichen *Xanthoria parietina* (×960). *B*, two filaments of the alga *Scytonema* invested with fungal hyphae, from the lichen *Stereocaulon ramulatum* (×730). *C*, a chain of the alga *Nostoc*, one cell being penetrated by a hyphal branch, from the lichen *Physma chalazanum* (×960). *D*, the spherical cells of the alga *Gloeocapsa* penetrated by fungal hyphae, from the lichen thallus of *Synalissa symphorea* (×960). *E*, two cells of the alga *Protococcus* sp., the fungus merely investing the cells, from the lichen *Cladonia furcata* (×960) (after Bornet)

An entirely different type of mycorrhizal association is found in the

majority of annual and perennial plants, in which the cortex of the root is invaded, usually in limited areas behind the apical meristematic zone, by a sterile fungus characterised by special organs known as ' vesicles ' and ' arbuscules ' (Fig. 88). The vesicles are large thick-walled diverticula or terminal swellings on an inter- or intracellular mycelium, while the arbuscules are haustoria-like, finely branched bodies formed often in particular cortical cell layers. Fat is stored in quantity in the vesicles and larger hyphae and is liberated into the host cells through the arbuscules, which collapse into formless clumps of indigestible fungal elements. The fungi concerned are regarded as probably members of the Zygomycete family *Endogonaceae* and have been assigned to an imperfect genus, *Rhizophagus*, of this family [8]. It has been suggested that they may supply, with the fat, some accessory nutrient or growth promoting substance to the host. The last-mentioned type of mycorrhiza is often casual in its formation and there is little evidence that it meets an obligate need of the host. But, in general, there is little doubt that mycorrhizas are beneficial and in certain cases are of great value in securing a satisfactory growth.

The lichens form the most prominent case of symbiosis in which fungi are concerned, though of lesser interest to the plant pathologist than those mentioned above [53]. They consist of a fungus and an alga living in such perfect association that they appear to form quite definite plants (Figs. 89, 90). The fungus hyphae surround and grow between the algal cells, into which they sometimes send haustoria; they feed in part on the organic material which the alga builds up by photosynthesis from the carbon dioxide of the air. The fungus in return appears to supply water, minerals and possibly peptone; it seems to have the best of matters, for it alone forms spores in most cases.

The best-known symbiotic association in which bacteria take part is that of the root nodule organism of the Leguminosae, *Rhizobium* or *Pseudomonas radicicola* (Figs. 91, 92). This is of considerable importance in agriculture, for many pulse crops will not grow well unless furnished with a suitable strain of the bacillus. The latter has the power of fixing atmospheric nitrogen and making it available to the higher plant and also indirectly to the soil when the crop

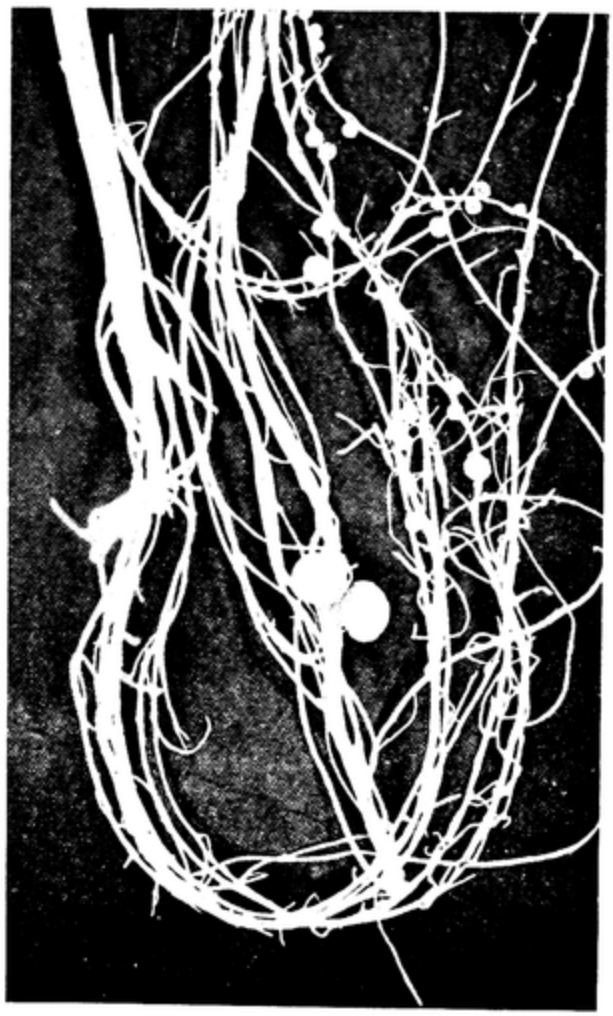


FIG. 91.—Bacterial nodules on roots of soya bean

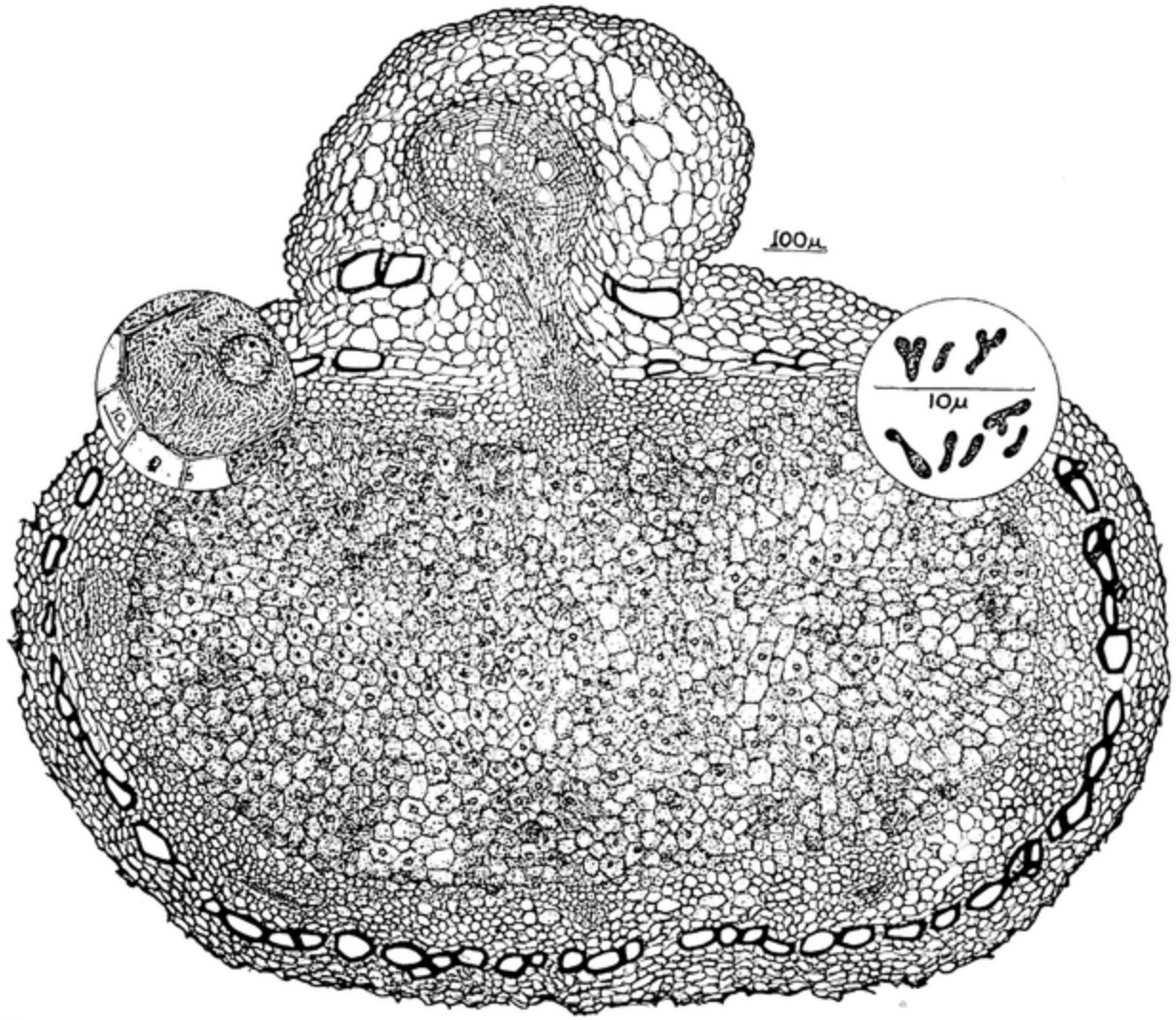


FIG. 92.—Transverse section of root nodule on soya bean showing vascular connection with the root. The central part of the nodule consists of larger cells infected with bacteria (dark-shaded), with smaller uninfected cells amongst them; the zone of small cells surrounding the infected tissue is the meristematic zone to which is due the continued growth of the nodule; vascular strands are seen at various points outside this zone; the prominent thick-walled elements are fibrous cells. Insets: left, portion of an infected cell showing nucleus, and adjacent uninfected cells; right, bacteroids ("involution forms") from root nodules on broad bean (*Vicia faba*) (from slides by Bond)

decays or is ploughed under as green manure. Strains vary in their power to do this; some have been found in Great Britain that are poor nodule formers yet can outgrow and overcome the better types. It may well be that the association started in this case also as a parasitic attack by the bacillus on the root, for under certain conditions *Pseudomonas radicicola* can become parasitic (see p. 156). The bacillus profits by the same freedom from competition by soil saprophytes that is enjoyed by root parasites.

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Chapter II

THE LIFE-HISTORY OF PARASITIC FUNGI

A PARASITE of the roots or stems of perennial plants or the leaves of evergreens has always available the food supplies requisite for its development. It may be, and often is, dependent on seasonal variations of activity, but is rarely put to such straits to tide over periods unfavourable to its existence as the forms which live on annuals, or on the leaves of deciduous plants. Most of the common crop pests, and many other parasites, have to face a period of the year when their ordinary supports are not available. The fungi which attack agricultural and horticultural crops have usually to pass through a period of about half the year in some other way than as active parasites of the crop concerned. The different methods by which this problem is solved are of considerable interest.

THE PERSISTENCE OF PARASITES FROM YEAR TO YEAR

In the powdery mildews on annual leaves, the greater part of the fungus dies off when the leaf withers. Later in the season, however, the perithecia often develop, sometimes abundantly. At this period they are immature, and with the ascospores undeveloped. After a time they become detached from the mycelium and fall to the ground, or they reach the soil with the leaves. Here internal development proceeds, and after some months, particularly if exposed to wet, as during showers, the asci mature and ripe ascospores are formed; dew is usually inadequate to promote this. The spores are ultimately set free by the rupture of the perithecium, and infect the growing plants. In the cereal mildew, *Erysiphe graminis*, a temperature of about 21° C. has been found to conduce to the rapid maturation of the ascopores [10]. The apple scab parasite, *Venturia inaequalis*, behaves in much the same fashion. Its perithecia can be found on fallen leaves in the winter, but ascospores do not appear until about March in the south of England and are not ready for liberation until April or May, when rain will cause their discharge. Apple leaves kept in moist chambers mature their perithecia comparatively slowly at 4° and 7° C. but the rapidity of development increases with rise in temperature up to about 20° C. [12]. In America, it has been found that the date of maturation of the spores may vary from year to year by as much as a month, no doubt in dependence on weather conditions during the winter, but in Kent it is seldom that danger from the ascospores threatens before the third week of April, and the date of their first discharge falls, almost always, within the second half of this month. *Venturia*, however, is able to persist over winter in the mycelial condition on the shoots and bud scales of the host (Fig. 93), and while in the south of England the ascospores are held to be the main source of the



FIG. 93.—Bud-scale infection. Apple scab (*Venturia inaequalis*). *A*, *B*, portions of cross-sections of buds of variety 'Bismarck' showing the fructifications of scab on the internal scales and on the axis (×17) (after McKay, *J. Dept. Agric., Eire*)



FIG. 94.—Six-year-old branch of pear showing pustules of pear scab (*Venturia pirina*). On the old wood the pustules are present at the points indicated by the pins; usually the pustules occur only on the one-year-old wood (photo by Dillon Weston)

first infections, in other parts of the country the new infections from conidia in these situations are stated to be usually well established before ascospore discharge occurs [15]. Perennial mycelium also exists in branches of pear trees affected with scab (*Venturia pirina*) (Fig. 94), in pine trees suffering from blister rust (*Cronartium ribicola*) (Fig. 95), and in numerous trees or shrubs attacked by a wide range of fungi.

Many other Ascomycetes mature their ascospores soon after the perithecia are formed, but these spores, aided often by the protection afforded by the wall of the perithecium, preserve their powers of germination for many months, even if exposed to considerable variations of temperature and humidity, as when cast into the soil. In deciduous perennials, the perithecia may lodge in crevices in the bark, and the spores be liberated in the following season in time to infect the new leaves. Many rusts and smuts, and most *Peronosporaceae*, are carried over by the prolonged germinative capacity of their perfect or other durable spore forms. In the onion mildew (*Peronospora destructor*) (Part II, p. 697) the oospores usually do not germinate until several years after their formation [14]. In spinach mildew (*Peronospora effusa*), (Part II, p. 691) oosporal contamination of commercial

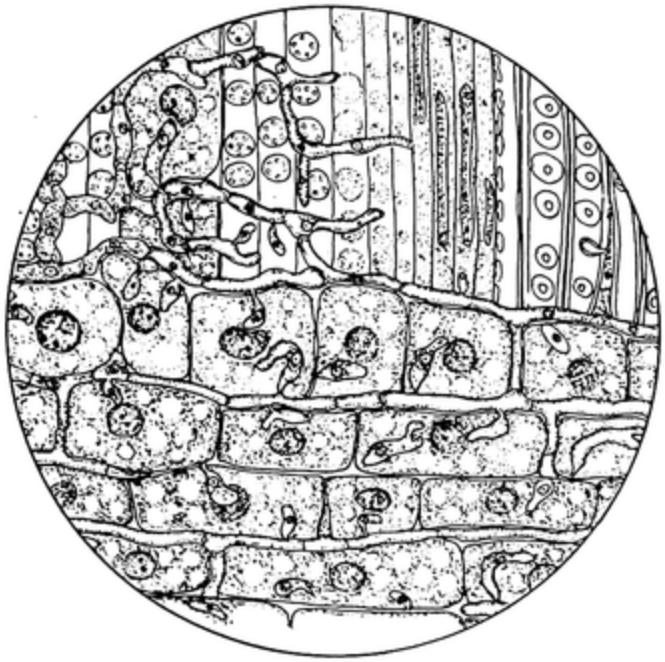


FIG. 95.—Perennial mycelium of 'blister rust' (*Cronartium ribicola*) in stem of pine. The fungus passes between the cells of the medullary rays into which it sends haustoria, and penetrates also into the cambium and phloem (× 330) (after Colley, *J. Agric. Res.*)

seed has been found to an extent of one oospore to 8½ seeds, and heavily contaminated seed gave a severely mildewed crop in the next season, as well as after two years (3). Sclerotia, chlamydospores, resting mycelium and even the conidia of some imperfect fungi (e.g. *Alternaria brassicae* and species of *Fusarium*), Ascomycetes, and smuts, can remain viable for prolonged periods.

In bunt (*Tilletia caries*) the spores, which are somewhat greasy and can remain viable for several years, remain adherent to the seed grain, which they reach during harvest, threshing, or storing, and are sown with the seed for the following season. Both parasite and host germinate together, and infection takes place in the seedling stage. Sometimes a second method is found (Fig. 63). Under certain conditions bunt spores survive in the soil for a considerable time and then germinate to form sporidia and secondary sporidia during a period of saprophytic life. If this coincides with the sowing of the new wheat, or if the field be manured with contaminated manure containing active sporidia, infection may occur. Many facultative parasites and facultative saprophytes owe their persistence largely to their power of passing a more or less prolonged period as saprophytes in the soil, or plant debris, or manure, until a new food crop is available. Indeed, were it not for the effect of the antagonistic competition of the normal soil saprophytes (see p. 171), the growing of healthy crops might be rendered difficult on account of this power possessed by many parasites of maintaining their life on non-living soil or organic nutrients.

The loose smuts of cereals differ widely from the above fungi in that they can persist from one season to the next in the mycelial condition in the grain (caryopses) of the host plants. In some of them the spores fall on the recently opened flowers, germinate, and the germ-tube grows into a mycelium which remains in or on the grain in a dormant condition until it is sown, when it resumes activity (Figs. 121, C, F; 206 E). In wheat and barley the embryo can be penetrated and the fungus passes from it, up within the seedling; in oats the seedling is infected as, or after, it emerges from the caryopsis. Other similar methods of carrying over until the following season are known in several families of the fungi. In the bean anthracnose (*Colletotrichum lindemuthianum*) (Part II, p. 603) the beans become infected through the pod but, if the attack be mild, are not much injured and will germinate in the ordinary course when sown; the fungus grows into

the young seedling and reproduces the disease in the new crop. Some bacterial diseases, such as bean blight (*Pseudomonas phaseolicola*) (Part II, p. 596), and black arm of cotton (*Xanthomonas malvacearum*), can also persist through internal infection of the seed. It has been established also that mycelium of *Epichloe typhina* is present in seed of *Festuca rubra* (Figs. 96, 239 B), and probably occurs also in *F. ovina* and *Poa bulbosa*; it is reported that *Poa bulbosa* may also carry mycelium in all the bulbils [21]. In potato blight the fungus which remains dormant in the tubers can sometimes give rise to diseased shoots when they sprout (Fig. 97). Several other members of the *Peronosporaceae* persist as perennial mycelium within the host; in the onion mildew (Fig. 329), the primary infection is frequently due to internal mycelium from the bulbs. The downy mildew of the hop (*Pseudoperonospora humuli*) (Part II, p. 879) survives in the root-stock from which internally infected spike-like shoots are produced in the spring and bear the spores which disseminate the disease each year (Fig. 98). In woody plants the fungi which produce witches' brooms (Fig. 99) possess perennial mycelium. In peach leaf curl, the parasite *Taphrina deformans* (Part II, p. 770) is said to have perennial mycelium in the branches, which occasionally serves to infect the newly opened leaves in the spring. The majority of the new infections, however, come from conidia which have over-wintered on the bud scales and twigs, and the most recent work (based largely on evidence from spraying experiments) goes to show that perennial mycelium does not usually occur and that either the ascospores or their derived conidia are responsible for the continuance of this disease [32]. Many other instances of the occurrence of perennial mycelium are known, as in horse-radish, *Helleborus*, *Ranunculus*, *Vicia*, for *Peronosporaceae*; *Euphorbia*, *Anemone*, for rusts; *Scilla*, *Lychnis*, for smuts. Among ectophytes several of the powdery mildews, e.g. those of apple (Fig. 337), *Euonymus* and vine, persist as dormant mycelium in the bud scales and on perennial parts of the plant. Where internal mycelium is

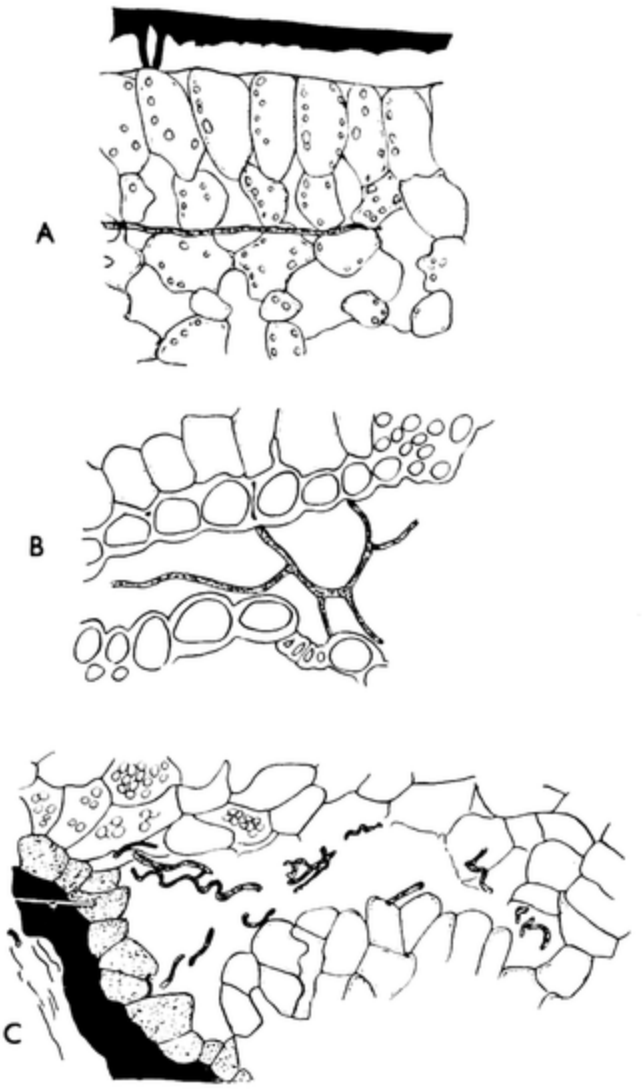


FIG. 96.—*Epichloe typhina*. *A*, the fungus in the leaf of *Festuca rubra*. *B*, part of shoot of *Alopecurus pratensis* showing the mycelium emerging from the leaf. *C*, the fungus in the ovary of *F. rubra*; the aleurone layer on the left is stippled (× 330 (after K. Sampson, *Trans. Brit. Myc. Soc.*)

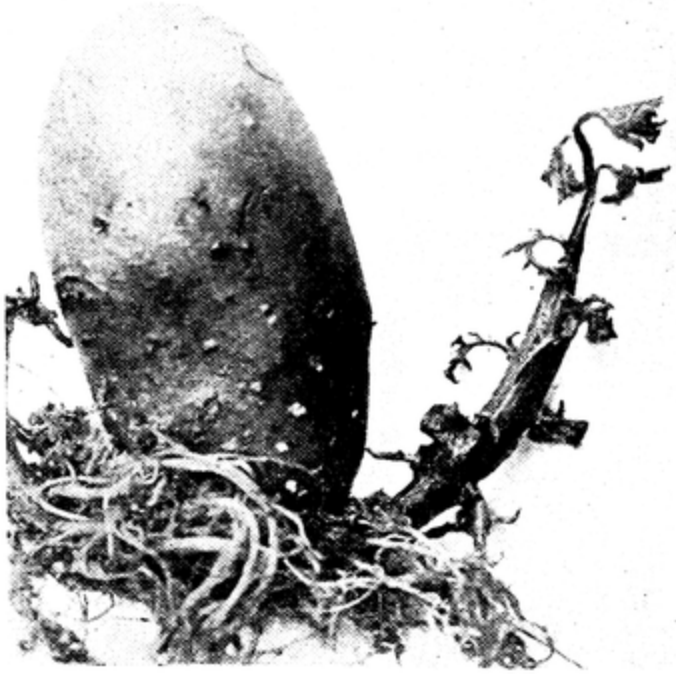


FIG. 97.—Potato tuber in which the fungus of potato blight (*Phytophthora infestans*) has apparently remained dormant, giving rise to a blighted sprout (after Alcock & McIntosh, *Ann. App. Biol.*)

present in shoots or branches it is obvious that propagation by cuttings offers another means for continuance of disease; this applies in several cases and *Epichloe typhina* may be cited again, as an example of transmission by divided propagants of its grass hosts.

Plants harbouring a perennial internal mycelium, as well as those which are infected in the seedling stage and retain the parasite within their tissues during their lifetime (as many of the smuts), are said to be 'systemically' infected.

Another interesting group of cases is that in which the normal 'incubation' period of the parasite, between infection and spore formation, is prolonged as a result of unfavourable conditions of temperature, nutrition and so forth. The mycelium passes into a condition of suspended activity but is not killed, as its subsequent development, when conditions become more favourable, proves (rusts of cereals, hollyhock, etc.). The term 'hibernating mycelium' is sometimes used to describe this, but is obviously a misnomer in the tropics where the unfavourable conditions are more usually in the hot season. An exceptional form of this type of delayed activity occurs in some parasites, especially parasites of fruit belonging to the closely allied genera *Gloeosporium* and *Colletotrichum*, where the infection hypha from an appressorium penetrates the cuticle of the young fruit but its further passage is delayed until the fruit ripens. In the banana anthracnose caused by *Gloeosporium musarum*, the penetrating thread forms a small hyphal knot between the cuticle and the outer wall of the underlying cell, and since these knots have been produced by inoculation of the young fruit, whereas the anthracnose spots do not develop until the fruit is nearly ripe, it may be taken as highly probable that delayed infection results from them. A similar course of events has been postulated in infection of papaw and mango by allied fungi, and in all these cases the control of infection by spraying has proved impracticable [24]. Even when the fungus is one that enters through stomata, as in the infection of young fruit of Avocado pear by *Dothiorella gregaria*, a latent period may follow before the hyphae invade the underlying tissues, as these begin to soften [11].

Many parasites are capable of living on more than one host plant, sometimes on a large number belonging to several different natural orders (e.g. *Sclerotinia sclerotiorum*, *Botrytis cinerea*, and species of *Pythium*, *Rhizoctonia*, etc.). This,

naturally, may enable them to remain active throughout the year. A special type of this aptitude is known as heteroecism.

Heteroecism

In some of the parasites which attack different plants, especially those that are widely separated in botanical classification, the fungus assumes a different form on the separate hosts, and passes one phase of its full life-cycle on the first plant attacked and another phase on the second; it thus requires both hosts for the completion of its full development. To this the term 'heteroecism' is applied. Heteroecious fungi occur almost entirely among the rusts, the only other known instances being two members of the Ascomycete genus *Sclerotinia*, *S. heteroica* and *S. rhododendri*, and in these the two alternate host plants involved are botanically related [8].

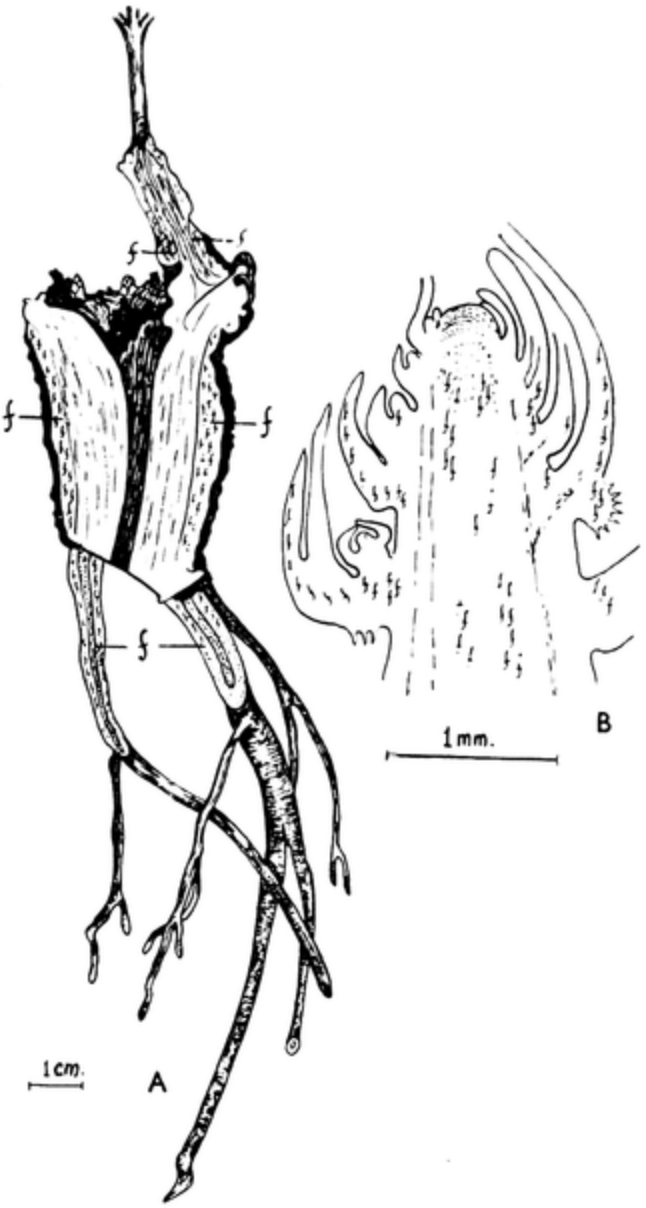


FIG. 98.—Perennial mycelium of downy mildew of the hop. *A*, section of a nursery set from a cutting planted in 1923, and examined on Nov. 11, 1925. The mycelium (*Pseudoperonospora humuli*) is shown by the small letters *f* in the bast and cortex of the older parts, and also in the swollen base of the 1925 growth; in addition, it was present in the roots. *B*, diagrammatic outline of a longitudinal section of apical bud of a shoot (bine), with a terminal 'spike', at about 7 ft. from the ground; mycelium again marked *f*; from here, hyphae were traced downwards to a distance of 39 cm. in the bine (slightly adapted, by permission of Ware, *Trans. Brit. Myc. Soc.*)

The most familiar instance of heteroecism occurs in the black rust of wheat. The destructive parasite passes the first part of its life on certain barberries, the second on wheat and other cereals and grasses. It never progresses on the barberry beyond the aecidial stage and the aecidiospores borne on the barberry cannot infect the same host. About seventy-five years ago, however, it was discovered that these aecidiospores could infect wheat and other Gramineae, and on these hosts produced the long-known black rust, the uredo-teleuto stages of *Puccinia graminis*. On germinating the teleutospores it was found that the resulting sporidia were unable to attack the cereal or grass host on which they had been formed, but infected barberry leaves readily, giving the spermagonia and



FIG. 99.—Cacao (Trinidad, B.W.I.). *A*, branches showing witches' broom caused by *Marasmius perniciosus*. *B*, showing the small sporophores of the fungus (photos by permission of Wiltshire)

aecidia of barberry rust. The cycle was therefore complete; two of the spore forms, one of which represents the haploid gametophytic fructification (see p. 344), are borne on the barberry; two on the wheat or other grass; and one — the sporidium — is developed at the expense of the reserve food stored in the teleutospore.

In *Sclerotinia heteroica* the cycle is somewhat simpler. On one host, *Ledum palustre*, sclerotia are found in the fruit and develop, after a period of rest, into the apothecia of the ascigerous stage. The ascospores, however, cannot infect *Ledum*, but for their further development must reach the shoots of a different plant, *Vaccinium uliginosum*. On this they produce a conidial stage, differing widely from the ascigerous form. The conidia are again unable to infect the host on which they arise, but if sown on the stigma of a *Ledum* flower, germinate, pass down to the ovary and reproduce the sclerotia; there are only the two spore forms, each on a different host.

Heteroecism in the rusts always follows in regular sequence; the aecidial stage is passed on the one host and the uredo-teleuto stage (or sometimes the teleuto only, the uredo being dropped) on the other. It is probable that originally the fungus lived its whole life on one plant and later took to the habit of spending part of its life on a second. How this arose, however, is obscure, though attempts to account for it are not wanting.

If the heteroecious fungi were always obliged to go through their whole cycle of development, it is clear that they would only continue to exist as long as both hosts were available; it would then be possible, for instance, to eliminate the cereal black rust by destroying the barberry. Unfortunately, this is not the case, for it has been found that the alternation of hosts is by no means essential to the life of the fungus. Black rust has been prevalent in Australia for over 140 years, though the aecidial stage was only seen for the first time in nature there in 1933 [33]. So also in India the aecidial stage, found only in the Himalayas, cannot be con-

nected with the commonly prevalent black rust of wheat in the central part of the country. Nevertheless, there are many areas where barberry eradication has greatly reduced the injury caused by black rust (see below, p. 225).

Outside the fungi, this peculiar habit of changing hosts is of still greater interest, owing to its connection with certain human diseases. Malaria, for instance, is due to a heteroecious animal parasite which passes one stage of its life in some mosquitoes and another in man.

Specialisation of Parasitism

Amongst those fungi which are capable of attacking several different species of plants many have developed into distinct races, each of which, though outwardly similar to the others, is restricted to one, or a few only, of the host plants. A special species of fungus, such as *Puccinia graminis*, may include a number of these races, similar in structure and not to be distinguished from one another in any other way than by their capacity for living on certain hosts [27]. Furthermore, since a single species of host plant may comprise a number of varieties, especially if it be an economically important plant that has been subjected to breeding and selection, there may be several, or even many, different races of the parasite on one species of host, differentiated by their capacity to attack particular varieties more or less severely than others. The difference between them is in physiological characters, not in characters usually recognised in botanical classification, so that there may be several or many physiological varieties or forms within the one botanical (morphological) species. To this splitting of a parasite into specialised races on different host plants the term ' specialisation of parasitism ' is applied. The races are now known as ' physiologic races '. In certain rusts, mildews, and other fungi, the specialised races may be grouped into a smaller number of groups possessing slight but constant morphological differences from one another. With some of these it has been found convenient to regard each of these groups as a sub-species of the fungus. A trinomial nomenclature has become a common usage with some economically important parasites in order to define the sub-species concerned, while within the sub-species the physiologic forms of which it is composed are given numbers. Thus *Puccinia graminis tritici* ' 56 ' refers to physiologic race No. 56 of sub-species *tritici* (so named because wheat is its most important host) of *Puccinia graminis*.

These various races of rust are not morphologically distinct but physiologically they are, because they produce different effects on certain cereal hosts. They are distinguished by the degree of spotting which they produce when severally inoculated into seedling leaves of twelve selected standard wheat varieties; five oat varieties are used for differentiating between the races of *P. graminis* on oat, and also five for the rust on rye; these cereal hosts are known as ' differential hosts ' (Fig. 100). The inoculations are carried out with uredospores, sprayed on to the foliage leaves. According to the degree of spotting produced, each type of infection is indicated by one of six symbols, thus :

Type 0—very faint, light flecks; host therefore considered immune.
,, 1—uredosori very minute; host strongly resistant.

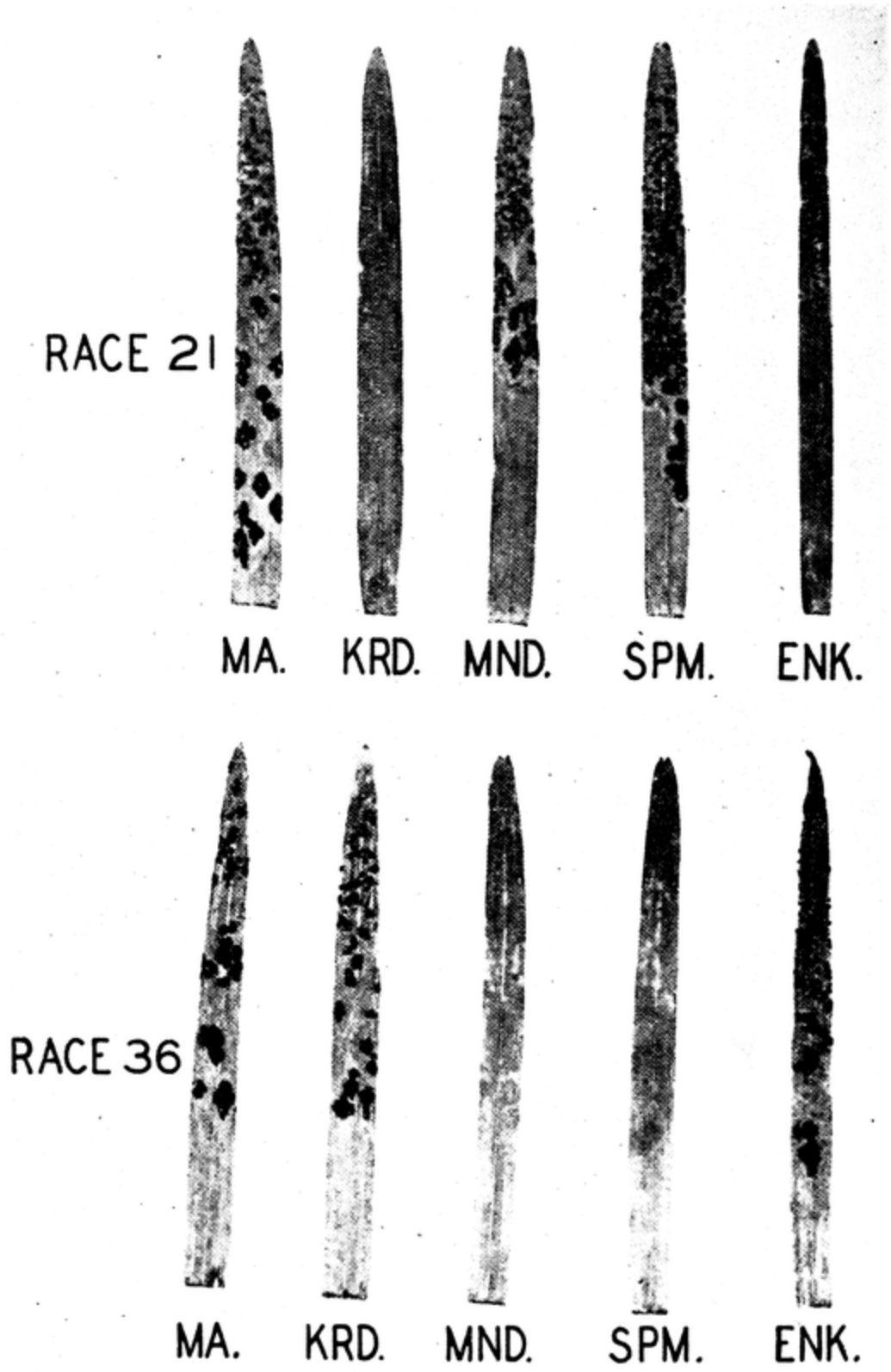


FIG. 100.—Physiologic races. Infection types produced by two different races of *Puccinia graminis tritici* on the same wheat varieties. *Ma.*, Marquis; *Krd.*, Kanred; *Mnd.*, Mindum; *Spm.* Spelmar; *Enk.*, Einkorn (by permission of Craigie, *Dom. Can. Agric. Frmrs'. Bull.* 84)

Type 2—uredosori small to medium ; host resistant.
,, 3—uredosori medium size ; host moderately susceptible.
,, 4—uredosori large and confluent ; host very susceptible.
,, X—a ' mixed ' reaction ; uredosori of variable size, mostly those of 1 and 4.

In addition, a ' plus ' or a ' minus ' sign, or two pluses or two minuses, are included to indicate variations of a slightly greater or slightly less susceptibility, respectively, than that represented by the infection type shown in the table. A double plus or a double minus denotes a slightly greater departure than the single plus or minus. The following table shows some actual results, the names indicated being those commonly applied to the twelve differential strains of standard wheat specially chosen for this purpose [4] :

Physiologic Race	Little Club	Marquis	Kanred	Kota	Arnautka	Mindum	Spelmar	Kubanka	Acme	Einkorn	Vernal	Khapli
19	4	2 −	0	3 −	4 =	4 =	4 =	3 + +	3 + +	3	0	1 =
56	4	3 +	3 +	3 +	1 =	1 =	1 =	3 +	3 +	1 =	1 =	1 −

Every degree of transition may be found between those parasites which freely infect all their hosts, through those that attack other hosts less readily than the one from which they are taken (e.g. *Rhizoctonia*), to those which are narrowly specialised on a single host (e.g. *Urophlyctis alfalfae* (Part II, p. 452) on lucerne). Evidently the adaptability of the parasites to their hosts varies greatly in different fungi and is sometimes exceedingly close. Whole genera of fungi are known that are confined to a single family of plants, as the *Phragmidium* rusts to the *Rosaceae*. Less frequently a single species of fungus is restricted to a single genus of host (as the uredo-teleuto stage of *Puccinia anomala* (Part II, p. 430) on *Hordeum*), or to a few allied genera, as the potato-blight fungus (*Phytophthora infestans*). There is nothing surprising, therefore, in finding a tendency amongst those fungi that attack many hosts to split into races, each specially adapted to life on a single one or a few of them. Chemical heterogeneity of the hosts is probably the reason for this.

Specialisation of parasitism is strongly marked in many rusts and mildews (both *Peronosporaceae* and *Erysiphaceae*). It is also very evident in the *Chytridiaceae*, Ascomycetes such as *Protomyces*, *Exoascus*, *Claviceps* and *Epichloe*, smuts, Basidiomycetes (*Corticium*) and Deuteromycetes (*Colletotrichum*, and several other genera).

Specialisation has been very fully investigated in the rust and mildew of the commoner cereals [4, 17]. Wheat, barley, and oats have each their distinct races of rust and mildew. The black rust of wheat will not attack oats, nor that from oats, wheat, under natural conditions ; successful infection of oats has been effected however, when spores from wheat were applied to young, rapidly growing tissues

in the laboratory. The races which occur on the cereals may, in certain cases, attack various grasses ; *Puccinia graminis tritici* has been found on some 25 species of grasses and *P. graminis avenae* on nearly as many, some species being susceptible to both sub-species of the rust. Grass hosts can sometimes be implicated in the persistence of a cereal rust from one season to the next.

The cereal rusts are heteroecious. In none is the aecidial stage produced on the crop attacked. So long as the fungus reproduces itself by means of the uredospores, it breeds true except for the occasional appearance of a mutation or saltation, about which more will be said below. On the alternate hosts (barberry, buckthorn, and so forth) the sexual stage of the fungus is developed, and this gives an opportunity for the mixing of the nectar, bearing spermatia, produced by spermagonia arising from different sporidial infections. In this way, different physiologic races can hybridise, and the result will be a ' form ' which is heterozygous in respect of the character of physiological specialisation. If a heterozygous physiologic race on a cereal or grass passes again on to its alternate host, an opportunity for segregation is given, and forms that are new to the locality may arise. A case of the sort is reported from Australia, where the relatively small number of physiologic races of *Puccinia graminis* that were known there up to 1934 had probably been, for the most part, introduced from other countries. In 1933 the heterozygous *P. graminis tritici* ' 34 ' was found on barberries, and evidently had reached them from the grass *Agropyron scabrum*, which was infected with race ' 34 ' of the rust, in close proximity. In the following year, race ' 11,' not previously known in Australia, was found in the neighbourhood, and it has subsequently been reported on the wheat in several districts. Race ' 11 ' is known to be one of the segregates from ' 34,' and it can scarcely be doubted that it arose as a result of the passage of ' 34 ' from *Agropyron* to barberry [34]. In the barley rust, *P. anomala*, two of the races, ' 12 ' and ' 13,' occurring naturally at Cambridge, are heterozygous ; self-fertilised material of the aecidial stage on the alternate host *Ornithogalum umbellatum*, gave, from ' 12,' four races not previously recorded, and from ' 13,' two. Both also produced some already known races [6]. This faculty of producing, by segregation, new physiologic races in an area is an additional and very strong argument in favour of the eradication of the alternate host of destructive heteroecious rusts. It is noticeable that in countries like Australia, India, and Kenya, where barberries play little or no part in the life-history of *Puccinia graminis*, the number of physiologic races is much less than in Europe or North America where barberries are, or have been, numerous. There are over 200 known physiologic races of *P. graminis tritici*, but up to 1939 only nine of these had been found in Australia, and still fewer in India (six) and Kenya (five). A sixth was added in Kenya in 1940.

In the other cereal rusts, the number of physiologic races hitherto distinguished is less than in *P. graminis*. There are about 90 of *P. triticina*, about 40 of *P. coronata*, and about 30 each of *P. glumarum* and *P. anomala*. These enumerations, however, are a little unreal, for they are based on the reaction to infection by the rust in question of an arbitrarily selected standard collection of varieties of its host, as above mentioned for wheat, and it has sometimes been found possible to split a particular race into two by adding a new host variety to the collection. The

tests, too, are not always made under standard conditions of environment, and it is now known that differences in temperature and light may produce changes in the reaction of a test variety of the host to a particular race of the rust.

The cereal mildew, *Erysiphe graminis*, is rather more strongly specialised than *Puccinia graminis*, for the physiologic races on wheat (of which several are known) will ordinarily only infect species of *Triticum*, those on barley, species of *Hordeum*, and those on oats, species of *Avena*. An exception to this is the susceptibility of young plants of *Hordeum sylvaticum* to mildew from wheat, while resisting infection from barley. As usual with specialised parasites, the physiologic races differ from one another in their powers of attack, and the different species and varieties of the host are not all equally attacked by any one form of the fungus. In several of the *Erysiphaceae* a reduced type of infection occurs on some of the susceptible hosts which are not readily attacked, and may show a prolonged incubation period and yield few spores. A distinction has also been drawn between full infections and sub-infections, the latter term being applied to infections on hosts not habitually attacked by the fungus and also to infections artificially produced on otherwise immune plants after wounding, or scorching, or the application of narcotics. By the latter treatment the reaction of potato tubers resistant to blight could be retarded so that a susceptible reaction was obtained, this reaction being one of speed of production of a substance toxic to the fungus. It has been found that *E. graminis* from wheat will infect barley leaves that have been touched by a hot knife, or near where a piece of leaf has been removed or gnawed by slugs, or where bruised by hail or in rolling the crop. Attack by green fly may have a similar effect. If exposed to the vapour of ether, chloroform, or alcohol, the whole leaf may become susceptible [20]. In such cases the defensive mechanism, which acts by preventing full penetration of the epidermal cells by the haustoria of the fungus, is unable to function and successful entry by some haustoria is effected. A sub-infection has been observed to change into a full infection towards the end of the vegetative period of the host, presumably because the defences of the plant are too much weakened to arrest the activity of the haustoria, or chemical changes have occurred in the host cells which make them more receptive to the parasite. The total infection by *Ustilago zeae* in place of its usual localised attacks in maize plants exposed to the vapour of ether, or heated to 70° C., may be due to changes in the host cell walls, or cell contents.

Specialisation of parasitism is almost as strongly marked in some smuts of cereals as in the rusts and mildews, but in others, such as *Ustilago zeae*, it is less evident. In this group, as in the rusts, the sexual process may play a part in the production of new races. Thus at Halle, in Germany, crosses between sporidia of physiologic races of *Ustilago avenae* gave segregated forms that had very different powers of parasitic attack on particular varieties of oats from those possessed by the parents [18].

As a general rule, the infective powers of a specialised race are identical in all its spore forms. Thus in physiologic races of *Puccinia graminis* that are homozygous for their parasitic character, the aecidial form on the barberry has the same specialisation as the uredo-teleuto stage on cereals, unless crossing with another race, or saltation (which will be referred to below) occurs. In *Erysiphe graminis* also,

the conidia and ascospores have the same host range and this is true likewise for the five races of *Claviceps purpurea*.

In a general way the physiologic races of a specialised parasite are quite stable. It is true that the range and degree of their parasitism may fluctuate under temporary conditions of environment or nutrition, but such fluctuations do not become permanent. In most cases they appear to be due to factors which act by influencing the receptivity of the host rather than the parasitic powers or aggressiveness of the fungus. Thus, in the extension of the host range of forms of the *Erysiphaceae* mentioned above as having been caused by wounding or narcotising an ordinarily unsuitable host plant, it is the host and not the parasite that is modified. A leaf partly eaten by slugs sometimes becomes susceptible near the bitten edges to a fungus which it resists when undamaged. Frost, hail, and the like may act in the same way, just as they can enhance the injury caused by weak parasites like *Botrytis*, which is often most destructive after frost, or *Coniothyrium diplodiella* which may do so much damage to grapes after hail storms that it is sometimes known as the ' hail disease '. Even the agricultural operation of rolling the crop may temporarily alter the resistance of the bruised leaves. High fertilisation has also been found to affect the susceptibility of cereal varieties to physiologic races of rust. It was shown in Germany that extensive modifications of the reaction of the standard test collection of wheat varieties — used to identify physiologic race ' 14 ' of *Puccinia triticina* and races ' 4 ' and ' 7 ' of *P. glumarum* — could be induced by drastic modifications in the nitrogen and potash supplied [9].

In these examples the resistance of the host is modified, but the spores borne, for instance, on an injured part remain incapable of infecting sound tissue on the plant. It has not hitherto been found possible to ' educate ' a true parasite by any artificial means into attacking the perfectly sound tissues of a host that is normally immune from it. Prolonged experiments have shown that the propagation of wheat bunt or rust on varieties that have considerable resistance to the physiologic forms tested, does not alter their pathogenicity in any way. Similarly, *Erysiphe graminis tritici* has been cultivated in wounds on the leaves of barley for 128 clone generations, without showing the least sign of adaptation to this host. The cases to the contrary that have been reported are better explained by automatic selection, in a mixed population of the parasite placed on a refractory host, of those units of the population to which the plant is susceptible. The possibility also that saltation may account for some of these reports cannot be excluded. It has even been suggested that the now abandoned view — that certain host plants can act as ' bridging species ' in that, by growing the parasite on them for a time, it then becomes capable of infecting plants normally immune — may have a certain basis of truth if it is assumed that the ' bridging species ' has the faculty of inducing saltations capable of extended parasitism [22].

Saltation

The term saltation is applied to an abrupt change in the morphological characters of the whole, or more usually of a part, of the thallus of a fungus which up to then had followed the general rule that like gives rise to like in the successive

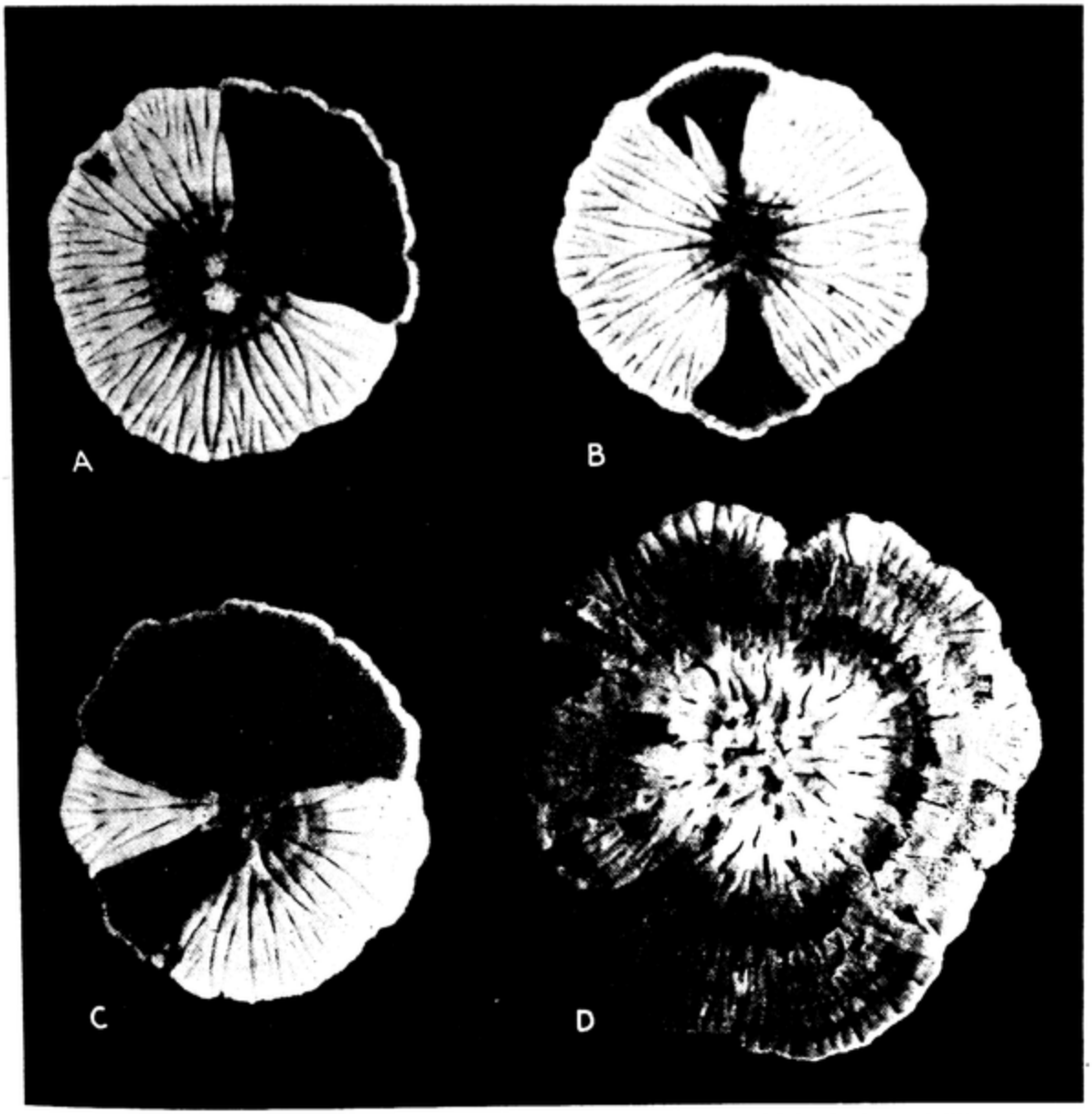


FIG. 101.—Saltation in colonies of *Penicillium notatum* (the fungus which yields penicillin) showing natural variation in culture. *A*, *B*, *C*, show light, mycelial sectors and dark, sporulating sectors of various shapes. *D*, a normal colony showing sectors of varied appearance (photo by Hutchinson)

vegetative or asexual spore generations of a species. As will be seen, there is reason to believe that saltation may similarly produce abrupt changes in physiological characters, but these are usually much more difficult to observe. Saltation is generally visible in artificial cultures of fungi (Fig. 101), not because there is any bar to its occurrence in their natural life, but because it is much less likely to be noticed when the fungus is growing in its natural site, usually accompanied by others, than when it is in pure culture in a test tube or petri dish. In culture, the new modification generally appears as a sector in an otherwise homogeneous growth, and if the culture has been kept free from contamination and is the product of a single spore or piece of hypha, the change is likely to be quickly noticed.

Saltants may differ from the parent strain in such characters as rate of growth ; extent of sporing and sometimes of chlamydospore and sclerotium formation ;

length, breadth, shape, and septation of the conidia ; density, colour, and zonation in the mycelium. Some of these characters may be linked or antagonistic, albinism with sterility, or copious formation of aerial mycelium with scanty sporing.

The change that occurs is often permanent in successive vegetative or conidial generations. It has, thus, a certain similarity to mutation in higher plants. It is, indeed, quite likely that some saltations are mutations ; others, however, are probably not connected with chromosomal or gene changes, and the term saltation has the advantage that it is non-committal as to the origin of the new strain. Sometimes saltation may be the result of segregation in hybrid strains ; it has indeed been suggested that most saltations can be ascribed to a heterozygous constitution of the parent mycelium and that, even where there have been no nuclear fusions, vegetative anastomoses may lead to a condition of heterocaryosis due to the presence of nuclei of different origins in the one cell. It is known that the presence of the paired nuclei in the dikaryophytic mycelium of the Basidiomycetes can affect such characters as the rate of growth of the hyphae and the like. But there are many cases of saltants first appearing after many successive culture generations of the fungus have been grown, and of their production as a result of irradiation (X-ray and radium) (Fig. 102), chemical treatment (zinc sulphate, the sodium salt of salicylanilide), high temperature, and so forth, that are difficult to ascribe

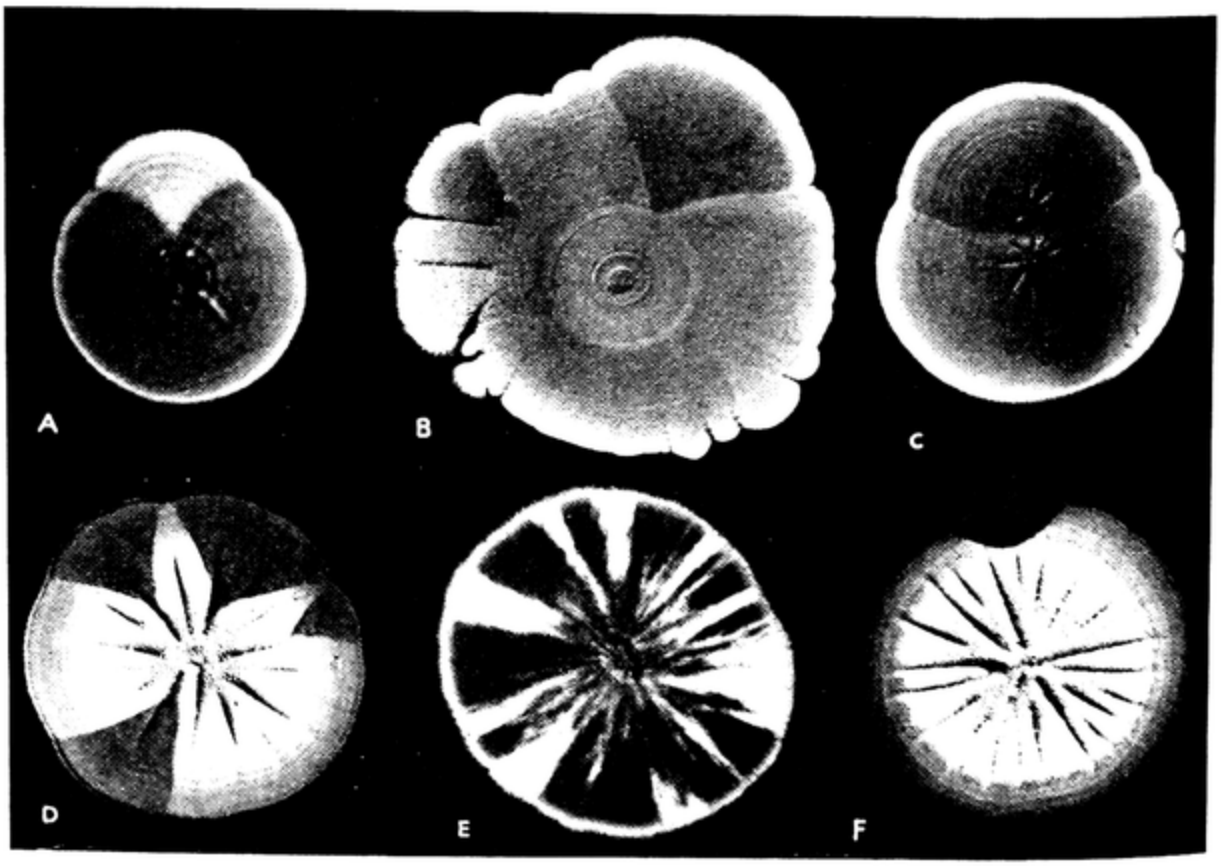


FIG. 102.—Saltation and irradiation. Colonies of *Penicillium notatum* showing sectors. *A*, a spontaneous sector. *B*, irradiated colony showing mutant sectors of different shapes. *C*, two separate colonies with different growth-rates meet and compete for the same territory. *D–F*, imitation of various types of sectors, by inocula of two strains, with equal (*E*, *F*) and different (*D*) growth-rates (after Pontecorvo & Gemmell, *Nature*, 3913, 1944)

to heterozygosity [29, 30]. The frequently observed fact that a young mycelium is more likely to show saltation than one that is older, is also not in accordance with expectation on this view. At the same time, it is known in some cases that the parent characters may mask the presence of those of a saltant in the mycelium. In a strain of *Diaporthe perniciosa* (which always developed a masked saltant revealed on sub-culturing, at a certain stage of its growth) small pieces of the hyphae were removed and separately cultured; for the first few days of growth the pieces all gave the parent form, but those removed later grew into the saltant form; as the colony extended, the part yielding the saltant remained at a fixed distance behind the growing margin, until growth ceased when the whole colony produced the saltant. Furthermore, when the saltant was brought into contact with the parent strain, anastomoses occurred and the saltant was reconverted into the dominant parent type. So also when the fusion cells, produced when hyphae of two saltant strains of species of *Fusarium*, and of *Helminthosporium* anastomosed, were isolated and grown, they yielded forms that were neither new nor intermediate, but resembled one or other of the parents, or were a mixture of the two. When no mixture resulted, it was suggested that some form of dominance of one or other of the parents in each fusion product was involved. Such cases show a certain resemblance to the phenomenon of dominance in a hybrid strain, and if dominance can occur, some apparent saltations may be the result of segregation.

Saltation is much influenced by nutrition, so much so that it has been considered to be a function of the medium in which the fungus is grown. It is favoured by media rich in nutrients (e.g. Richards' solution), by high concentrations of salts, and by absence of staling products. Several cases have been reported in which two saltants, each of which grown separately is either infertile or sparingly fertile, when grown so that their colonies meet, produce spores copiously along the margin of contact. When these spores are asexual, as in the pycnidia of *Cytospora ludibunda*, there can be no question of the union of different sexes being involved, and the suggestion has been made that nutritive heterothallism may explain the result, each strain being able to absorb a part only of the nutrients, required for the formation of the fruit bodies.

Sometimes saltation is progressive, a first saltant giving rise to a second, and so on. Occasionally, one strain regularly gives rise to the same saltant in each culture generation of the parent. This has been observed, for instance, in *Coniothyrium pirinum*, in which the saltant remained stable in succeeding generations, and in *Diaporthe perniciosa* in which the saltant sometimes gave the parent form again. The last condition has been called cyclic saltation, and has been reported several times. Some strains of *Monotospora lanuginosa* give regularly cyclic saltants, while in others the saltant seems to be permanent [5]. A saltant in a species of *Alternaria* differed so much from the parent as to be referable to the genus *Stemphylium*, and the *Stemphylium*-form again gave the *Alternaria*. In such cases the process which leads to saltation is evidently reversible.

Saltation may be detectable immediately on the germination of the spore. Whether, when this occurs, it is due to a change in the spore, or just before its formation, or after the germ-tube grows out, is difficult to establish. Different

spores from a single pycnidium may give rise to different strains, some permanent, others not. Sub-cultures from each of the eight ascospores in an ascus of *Gibberella zeae* gave variants which occurred haphazard and did not appear to be due to orderly segregations in the ascus. The variants differed in their rate and type of growth and in pathogenicity [31]. Similarly, a culture made from a single ascospore of *Diaporthe perniciosa* gave four sectors which appeared to start from the neighbourhood of the spore; three of these again gave the parent form, the other persisted as a new form. Cultures made from different hyphal tips of the same germinating conidium of *Helminthosporium gramineum* differed in cultural characters and pathogenicity. It appears, therefore, that even an asexual spore may carry factors producing different types of progeny [23].

It appears probable that under the general term saltation, in fungi, several distinct processes are included. Some may be genetic and resemble mutations in higher plants, or be merely the result of segregation in hybrid strains. Others appear to be somatic and to be analogous to bud variation in higher plants. Others again may be primarily nutritional or due to the action of high temperature, chemicals or radiation on the nuclear constitution, or on the cytoplasm. This action must be deep-seated and it seems difficult to find any single explanation that will fit all the cases of permanent modifications that have been described.

The importance of saltation in plant pathology is connected with the evidence that the differences between saltant and parent in physiological characters, such as rate of growth and pigment formation, may extend to their parasitic properties. This has been fully established on several occasions [30]. Thus, of thirteen saltants of *Helminthosporium sativum* the pathogenicity of which was tested on wheat and barley, two proved to be more virulent than the parent strain, while three were less so [2]. So also in a single unisexual monosporidial line of *Ustilago zeae*, several saltants occurring as sectors in the colonies differed from the parent and from one another in pathogenicity, and these differences were maintained in successive sub-cultures for several years [28]. It is not infrequent to find colour variations due to saltation in the uredosori of rusts, and these are sometimes linked with new pathogenic properties. Thus, orange pustules appeared among the brown in some inoculations on barley of physiologic race '14' of *Puccinia anomala*, found in Cumberland. These bred true in successive uredo generations, and when their pathogenicity was tried on a series of test barleys, they proved to belong to a hitherto undescribed physiologic race; on some barley varieties severely attacked by the parent form, the new form produced no sori, and on one or two it was somewhat more virulent than the parent [6]. Similarly, physiologic race '33' of *Puccinia coronata* on oats apparently arose as a saltant from race '1' [16]. Extensive variations in pathogenicity have been found in some of the weakly parasitic fungi that attacked stored apples; in numerous saltants of *Cytospora ludibunda* the most aggressive form, as judged by the rate of advance of the mycelium in apple tissues, was more than four times more virulent than the parent, and more than thirty times more so than the weakest saltant. In similar work with *Fusarium fructigenum* no saltant was more virulent than the parent, but several were weaker; of two strongly contrasting saltants, one with a copious aerial mycelium and few spores, and the other a low-growing, sporing type, the action of the former in

rotting apples was much stronger than that of the latter. In some of these fruit parasites the saltant was either equal to, or more or less virulent than the parent according to the variety of the host on which it was tried. Age of the fruit also affected the relative virulence of the strains.

The significance of these facts in the occurrence of disease — in nature, and in the efforts to control it by breeding resistant varieties of crops — is obvious. Saltation may produce a strain of an ordinarily weak parasite with such enhanced virulence that an epidemic outbreak follows. If this occurs, it is probably rare, but it is a possibility that cannot be discarded in seeking to explain such outbreaks as that of the withertip disease of limes which destroyed the lime fruit industry in the island of Dominica in 1922, though the fungus responsible was morphologically indistinguishable from *Colletotrichum gloeosporioides*, a common but rarely serious parasite of citrus trees in most countries where they are grown.

Still more serious, however, may be the production of a new physiologic race of a fungus such as one of the cereal rusts, against which resistance has been secured by breeding and selection. The new form may render this work nugatory and compel a further effort to secure resistance against it. Fortunately, here again, such a contingency appears to be rare except where the new form is merely the result of segregation in a hybrid strain, but the collapse of the resistance to the wilt caused by *Fusarium niveum* of certain improved watermelon varieties in the United States has been ascribed to it [26]. What is much more frequent is an increase in the prevalence of a previously uncommon race ; the effect may be the same, and indeed may be a consequence of the increased cultivation of host varieties resistant to the older races.

Bacterial variants have also been described, though there is no certainty that the processes involved are analogous to those that cause saltation in fungi. The most common change is from a smooth to a rough type of growth or vice versa, and this morphological change may be accompanied by changes in pathogenicity. Thus the smooth colony strain of *Pseudomonas phaseolicola* was more virulent to beans and also more sensitive to the action of a bacteriophage than the rough colony form, in studies of the two forms carried out in Australia [1]. Sectorial variants differing from the parent colony in being white instead of yellow, are known in single-cell cultures of *Xanthomonas stewarti* and may remain permanent on sub-culturing, or in the host, or they may revert to the yellow type [7]. In this organism saltation or mutation affecting colony characters, colour, and virulence seems to be common. Passage through susceptible varieties of maize increases the number of colonies of low virulence obtained by subsequent sub-culturing, while virulent types predominate after passage through resistant varieties. The variants obtained in cultures from single cells may be more or less virulent than the parent, and the evidence suggests that when resistant seedlings are inoculated the proportion of virulent bacteria increases by selection within the plant [13].

Parasitism of Different Stages in the Life-History

In very many fungi active parasitism is confined to the earlier part of the life-history ; the later stages, particularly the development of the ' perfect ' spore

form, occur only after the tissues of the host plant have been killed. Thus, in many *Peronosporaceae* and Ascomycetes the oospores, or the perithecia, only appear in or on dead parts and are nourished saprophytically either from the dead organic matter of the host cells or, more probably, from reserves stored up in the mycelium during its period of active parasitism. The perfect stage, *Corticium solani*, of the ubiquitous *Rhizoctonia solani*, differs from these in that it is only found on healthy green parts of plants; but still it behaves as a saprophyte, causing no injury to the parts on which it grows, using them only for support. In marked contrast to these are the cases where the gametophytic stage is not parasitic. In the peach leaf curl fungus *Taphrina deformans* (Fig. 367) the yeast-like haploid generation may be prolonged as a saprophyte on the buds and other superficial parts of the tree until a cool wet spell occurs in the spring, when the parasitic sporophytic stage develops [32]. In some of the *Ustilaginaceae* also it is only the dikaryophytic stage, developed as a result of the fusion of two haploid sporidia germ-tubes, or mycelia, that has the power of entering the tissues of the host. In wheat bunt, for instance, the hyphae arising from the haploid secondary sporidia ('secondary basidiospores') are slender and show no parasitic ability, but when two of these hyphae from sporidia of complementary sex fuse, a stouter fusion hypha results, and it is from an appressorium developed from the fusion hypha on the coleoptile of the host that the infection hypha arises (Fig. 63). There is evidence that the germ-tube (promycelium) arising directly from the smut spore can penetrate into the host and establish a parasitic mycelium, bearing nuclei of complementary sexuality; this has been reported in *Ustilago zeae* and *U. avenae* (Fig. 206), and no doubt occurs commonly in other species under natural conditions.

Nutritional Stimulation by Parasites

In Chapter VI reference will be made to the galls produced by fungi in many plants. The development of these is due to some stimulant action of the parasite which provokes hypertrophy or hyperplasy, or both, in the tissues of the host. The gall tissue is sometimes remarkably rich in reserve food. Thus the 'cedar apples' caused by *Gymnosporangium juniperi-virginianae* on *Juniperus virginiana* are due to the stimulation of the host to prodigious activity in the multiplication of its cells and the storage of starch. In Formosa the swollen shoots annually produced from plants of *Zizania aquatica* infected by *Ustilago esculenta*, which is perennial in the rhizomes, are marketed for food, the plants being sometimes specially grown for the purpose by Chinese. Similarly the hillmen in the Himalayas relish the hypertrophied shoots of *Urtica parviflora* infected with *Aecidium urticae*, a rust which has its uredo-teleuto stage on *Carex*. The young heads of sorghum transformed into sori of the head smut *Sorosporium reilianum* are sometimes eaten in India. In these and similar cases the parasite provokes a flow of nutriments from other parts of the plant, some of which are built up into protein matter and reserves in the mycelium, a dense accumulation of which may be found in the gall, some deposited as starch in the host cells. The gall cells may thus be regarded as constituting a storage tissue; but they differ from other

storage tissues in serving the needs of an alien invader rather than those of the parent organism, for they are largely depleted during the fructification of the fungus. The preservative, or even apparently restorative, action of certain parasites on the chlorophyll apparatus of leaf tissues (see below, p. 209), is a further example of nutritional stimulation evidently advantageous to the parasite rather than the host; it is marked by the formation of green islands of tissue surrounding infections by rusts and other fungi on leaves that have begun to fade from other causes, such as over-crowding, want of light, or the like, and is seldom seen in infections on fully healthy green tissues. Continuation of the photosynthetic activity of the cells has the effect of prolonging the parasitic life of the fungus. Chlorophyll is probably the most sensitive indicator in the cell of the degree to which a parasite has succeeded in establishing relations of equilibrium with its host [19].

Epidemics

For nearly a hundred years many of the most intensive studies of the life-history of parasitic fungi have centred around the species responsible for epidemic outbreaks of plant diseases. For other reasons also epidemiology (or the science of epidemics) has a particular appeal in plant pathology. The recognition of phytopathology as a distinct branch of biological science may be said to have had its foundation in a succession of epidemics (or 'epiphytotics' as they are sometimes termed in the United States), starting from the potato blight which swept through Europe in 1845 and 1846, and the vine oidium first seen in England in 1845 and France in 1848, and continuing with the coffee leaf disease which devastated the flourishing plantation industry of Ceylon and almost halted its development elsewhere in the East between 1869 and 1883, the vine mildew or 'peronospora' which, spreading from France through Southern Europe from 1878 onwards, brought panic and ruin to vine-growers everywhere in its track, to the rind disease which almost accomplished the destruction of the sugar-cane industry of the West Indies in its peak years about 1895. These and many similar outbreaks occurring with increasing frequency since the start of the present century have seriously affected the world distribution of crops of economic importance. Such are: coffee, of which Asia produced three-fifths of the South American production by 1879, before the industry was seen to be doomed from *Hemileia vastatrix*, as against a present-day production in the New World, free from this disease, some twelve times greater than that of the whole of the Old World; and rubber, in which attempts to establish a plantation industry in the western tropics, whence the tree originated, have hitherto been foiled by the South American leaf disease (*Dothidella ulei*). They have led to national disasters such as overtook Ireland when potato blight caused a loss of population of a million and a half from famine and emigration in the five years preceding 1850. They have had political consequences of the first magnitude, as this same disease in the repeal of the Corn Laws, of which Disraeli wrote in *Endymion*, "this mysterious but universal sickness of a single root changed the history of the world". They have been the probable cause of the extinction of species in particular areas, if one may draw conclusions from what is happening to the indigenous

chestnuts in the eastern states of the American Union, from *Endothia parasitica*, or to the elms in the Netherlands from elm disease. One may speculate on the possibility of a similar explanation of the disappearance of the British wine which the Romans enjoyed when one considers the loss of the Dominican lime-juice industry in recent years, from ' withertip ', or the fall in the export of Madeira wine from 7000 pipes in 1851 to 360 in 1861, said to have been due mainly to disease (presumably ' oidium ') : even though the species survived, it must be remembered that both blight and wart disease led to great changes in the varieties of potatoes grown in Great Britain, changes not always to the advantage of the consumer.

The epidemic diseases of plants can be divided into two main groups according as they occur in crops in which the disease is ordinarily endemic but assumes epidemic proportions from time to time, or as they are ' new '. Epidemics of the former group depend ordinarily on seasonal variations in the environment, especially those due to meteorological factors. An instance has already been cited to indicate that these factors can be operative at a distance both in time and space from the actual outbreak (see p. 59). In Great Britain, the chief diseases subject to seasonal influences which may raise infection to epidemic proportions include potato blight, cereal rusts, chocolate spot of field beans (*Vicia faba*) (p. 593), the powdery mildews of clovers (p. 456), brassicas, etc., and apple and pear scab (pp. 730, 749).

Chocolate spot is a good example of a disease dependent on several factors, for it only takes on the destructive epidemic form when a heavy inoculation with the spores of the causal organism, *Botrytis cinerea*, occurs coincidentally with favourable conditions for the development of the disease (Part II, p. 595).

Occasionally a parasite after years of mild attacks assumes and maintains epidemic severity. Such seems to have been the case with rubber oidium in the East, red rot of sugar cane (*Colletotrichum falcatum*) in India, and possibly ' withertip ' of limes in Dominica. The most plausible explanation of this is an accretion of virulence by saltation, but evidence for or against this suggestion would be difficult to find. The rust, *Puccinia psidii*, which developed with epidemic severity on pimento trees (*Pimenta officinalis*) in Jamaica in 1934, had not been previously recorded on this host, though a common parasite of *Eugenia malaccensis* in the island ; it has been considered to be almost certainly a recent mutant of the fungus.

The second group of epidemics of plant diseases, and that which has attracted the greatest public interest, is the result either of a new parasite having been introduced from another country, or of the passage of a parasite of some pre-existing plant of a region to a newly introduced host. The list of these (excluding virus diseases) that have been reported from all parts of the world during the past century is a long one : potato blight and wart disease ; vine oidium, ' peronospora ' and black rot ; coffee leaf disease or rust ; cacao witches' broom ; banana Panama disease and leaf spot ; palm bud rot ; sugar-cane *Sclerospora* or leaf stripe ; tobacco blue mould and wildfire ; onion smut ; hop downy mildew ; citrus canker and withertip ; American gooseberry mildew ; apple and pear fireblight ; chestnut ink disease and blight ; pine blister rust ; oak mildew ; elm die-back or

Dutch disease; hollyhock and antirrhinum rusts; and so on. Even such old and widespread diseases as apple scab, which did not reach Western Australia until long after the establishment of apple-growing in that State, and yellow rust of wheat, long unknown in North America and still not reported in Australia, have evidently not reached the limits of their spread. While the great majority of these diseases have assumed epidemic proportions on their introduction, this is not an inevitable consequence, as is apparent from the behaviour of yellow rust, which seems to have done comparatively little damage in North America.

Those who have seen the destruction caused by a bad attack of potato blight can best appreciate the vivid and harrowing descriptions on record of the outbreaks between 1845 and 1847 when it is remembered that none of the varieties then grown possessed any powers of resistance to the disease, while most of the present-day kinds probably have some. A group of scientific men reported that from a high point near Brussels the immense blackened patches that had been flourishing potato fields filled the vast horizon, and they were told that even one night had been sufficient to destroy the finest fields: this was soon after the first appearance of the disease in Flanders in July 1845. The following year a visitor to Ireland described "a magnificent field of potatoes, about twenty acres in extent, through which . . . one day during the last week in August, as we brushed through the dark-green foliage, earthy disagreeable odours . . . rose from the plants. On the following morning the entire crop looked as if it had been exposed during the night to the action of steam . . . in six and thirty hours a few sickly stems and discoloured leaves were all that remained." A few years later again, the famed temperance missionary, Father Matthew, wrote, "On July 27th, I passed from Cork to Dublin, and this doomed plant bloomed in all the luxuriance of an abundant harvest. Returning on August 3rd, I beheld . . . one wide waste of putrefying vegetation." In France and Italy references to the "veritably confounding" rapidity of the spread of vine downy mildew were equally common in the eighties of last century and were repeated as the malady reached new countries. A New Zealand vine-grower stated that when atmospheric conditions favour the development of the pest it "spreads like a prairie fire": "I have seen the foliage on ten thousand vines completely blasted by mildew within three days of its appearance".

Against these fulminant outbreaks may be set the slow, exterminating progress of diseases of forest trees. Chestnut blight (*Endothia parasitica*) was stated in 1940 not to have spared any existing tree of the species (*Castanea dentata*) indigenous in the east of the United States, but even after nearly forty years since its discovery in New York in 1904 it has not exterminated the tree, for individuals may continue to give repeated crops of basal shoots long after the trunk has been killed. In this and other cases, though recovery is unknown, it may be many years before the tree finally succumbs. It is well known that blister rust (*Cronartium ribicola*) of the Weymouth or white pine (*Pinus strobus*) and other five-needled pines has gradually caused the cessation of planting of these species except where control measures are rigorously enforced; *Pinus strobus* is gradually disappearing from Norway, its planting in Great Britain is now rare, and observations in New York State indicate that though some trees may survive attack they are generally so badly damaged as not to yield commercially valuable timber.

But the progress of the disease is slow; it may take three or more years for a sporidial infection from the teleutospores on *Ribes* into the pine needle to pass back to the branch and start a canker which will later bear aecidia, and the killing of a mature tree may perhaps take forty years or more. The allied *Peridermium pini* may take a hundred years to kill Scots pine in Bavaria. Actual infection by *Cronartium ribicola* may have occurred with epidemic intensity, for up to 5,000 cankers have been counted on the highly susceptible *Pinus monticola* in British Columbia, but this need not necessarily be reflected in the death rate. Time may be given for the application of control measures and, above all, for the introduction of disease-resistant species or varieties. Thus the ink disease of the sweet or Spanish chestnut, *Castanea sativa*, caused by *Phytophthora cambivora*, thought to have come into Europe (Portugal) possibly from the Azores, was seen in France in 1882. By 1922 it had destroyed 30,000 hect. of chestnuts in France, while in twenty-five years from 1910 it reduced the Italian nut crop by 100,000 tons. In Spain there are still (1942) about 5,000,000 trees in production, but in the relatively mildly infected province of Lugo there are only about half the number now that there were twenty-five years ago, and in Corunna province less than one-sixth of those recorded at the beginning of the century; in some areas destruction is almost complete. Meanwhile efforts are being made to cure early infections by exposing the roots and collar of the trees and applying antiseptics, and are meeting with considerable success, while both in France and Italy tests of resistant species from the Far East are proceeding on a large scale, the Japanese *Castanea crenata* showing a satisfactory degree of resistance in both countries (though susceptible to inoculation in England) and having some varieties that bear good fruit; both root-stock grafts with *Castanea sativa* scions, and direct producers, are being grown.

The best-known representative of this group of epidemic diseases in England is the Dutch elm disease, which has killed many fine trees in the east, south, and midlands, since it was first seen in 1927, but may have reached the limits of its extension in the north and west (Part II, p. 895). It is, however, still too early to predict to what extent it may reduce the total stand of elms in the affected areas. Hitherto it has not proved so virulent in England as in the countries in which it was first found (France in 1918 and the Low Countries the following year), whereas the hop downy mildew which reached Europe probably from Japan or North America about 1920 seems to cause greater injury in Bavaria than in England, which appears to have been the first European country affected.

These differences in virulence may depend on many causes, some of which, at bottom, are likely to be similar to those that have proved to be of ecological significance. The effect of these on the life-history of a parasite, however, is so complicated by pathogenic factors, such as are discussed in succeeding chapters, as almost to defy analysis. So far as climatic or seasonal factors are concerned, the evidence indicates that both the annual curve of fluctuations of temperature, humidity, and the like, and the maximal and minimal extremes, have to be taken into account, and that these may affect host or parasite, or both, while in diseases like the Dutch elm disease their effect on the insect vector may be at least equally important.

There is a rather general belief that epidemic outbreaks of 'new' diseases often reach their peak shortly after they are first introduced, and then undergo a gradual decline in virulence. In animals and man a partial explanation of this may be found in acquired immunity, but a similar explanation is rarely possible in plants. Perhaps some clue to a decline in virulence in plant epidemics may be provided by the evidence from bacterial wilt of maize that fully susceptible hosts tend to filter out the more virulent strains of the parasite. The records certainly suggest that hollyhock rust was as virulent when first introduced into Great Britain as antirrhinum rust (Part II, p. 846) has proved to be during recent years, but whether there has been any change in the hollyhock population in the direction of increased resistance is not known. In many cases it is not yet possible to account for the waning virulence of plant epidemics if such occurs. Among the factors tending to increase the degree of injury caused by parasites one of the most important is the length of time during which the crop remains exposed to attack. The reputation of the famous Marquis wheat in Canada and parts of the United States was largely due to its short period of growth, enabling it to yield its harvest before the main intensive invasion of black rust could reach it. In general, anything which tends to prolong the maturation of the whole crop (such as unfavourable weather) or of a part of it (such as insect injury) increases the prospect of heavy infection from parasites that are not confined to the early stages of growth. Even when the parasite is one that flourishes best on young leaves and the like, infection of the repeated flushes that follow defoliation, or the killing back of shoots from other causes, may bring about a great increase in the injury. Thus the killing of oaks from *Tortrix* infestation was not reported in England prior to the introduction of oak mildew (Part II, p. 900) in 1908, but since that time a number of deaths has resulted from the combined action of the caterpillars and mildew, since the latter tends to develop with great intensity on the young growth that follows defoliation by the insect. A similar lethal association has been reported from Canada on Scots pine (*Pinus sylvestris*) between the pine spittle bug, *Aphrophora parallela*, and *Diplodia pinea*, causing a crown blight.

The growth of large areas under a single crop assists in the spread of disease. In mixed vegetation an infected plant is often not a serious danger to its neighbours, as the majority of parasites are limited in the hosts that they can attack. On the other hand a disease centre in a large area under one crop may lead rapidly to infection of the whole field. Artificial inoculation in the United States with the potato blight fungus, where no blight was present naturally, proved that two plants near together were able to spread infection sufficiently to destroy the above-ground parts in a half-acre plot in 29 days. It has become a commonplace observation that disease sweeps through the large tropical and sub-tropical plantation crops — tea, rubber, cotton, tobacco, and so forth — with astonishing rapidity. Hence the mixing of field crops so common in countries like India has its advantages, for a field of, say, wheat, barley, gram, linseed, and peas mingled together will face less risk of the total loss of crop, which the Indian cultivator cannot afford, than five fields each carrying one of these crops. The same applies to the increasing tendency towards the cultivation of clonal lines of particular crops; should the

variety be one which is homozygous for susceptibility to a disease, every plant is liable to attack when the disease appears. In this connection there is a factor in epidemiology which is often not given due weight. So long as inoculum is light, few spores may hit a vital part and cause a casualty in a plant on which they fall. As the intensity of the spore shower increases, however, the chances that any single susceptible plant will fail to contract infection diminishes. Observation confirms the existence of epidemics, especially in permanent crops, which flare up into considerable intensity after a period during which the disease is sporadic, and these may perhaps be most readily accounted for by the parallel with the intensity of a field of fire in musketry here suggested. The intensity of an epidemic depends on the number of successful passages of the infective agent to a new susceptible host. When the hosts are fixed in space, and of approximately the same age and strain, as in the ordinary field and plantation crops, successful passage is a simple function of a number of spores in the air or other channel of infection at a time when the environment favours the establishment of the parasite in the host.

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Chapter III

PATHOGENESIS I: THE PARASITE IN RELATIONSHIP WITH ITS HOST

In order to obtain their food, parasites have to establish a close connection with their host plants; this implies that they have to enter into the tissues or to send haustoria into the surface cells. Entry is most frequently effected by the germ-tubes from spores, but can also often be accomplished by mycelial hyphae. In this process of 'infection' two stages may often be recognised and must be sharply distinguished, a stage of penetration and a stage of infection proper when the parasite is successful in establishing itself in the host. After infection is established there follows a period of incubation until visible signs of the presence of the parasite are seen. In many cases this is soon succeeded by the production of the sporing stage of the fungus, usually on the surface of the diseased parts. The normal progress of these stages may be modified by the influence of the host, as is shown in the next chapter, when, for instance, the host is one which is more or less resistant to the parasite concerned. It is also subject to much modification from influences of the environment; temperature, humidity, soil, and other conditions under which the host and parasite live have a marked effect on all stages of parasitism and will be discussed in a later chapter.

It is important to remember that the aggressiveness and virulence of a parasite are not constant qualities but may vary in different strains and different isolates of the parasite. The physiologic races of a parasitic fungus do not have identical powers of entering a particular host and spreading within it. We have seen that they are distinguished from one another by this character as brought out by testing them on a series of varieties of the host (Fig. 100). The process is capable of almost indefinite extension by increasing the number of test varieties of the host, and it is sometimes also made more difficult because of differences in the response of both host and parasite to different environmental conditions. For example, a spring wheat variety, Marquis, maintained its resistance to a physiologic race of bunt so long as it was grown in Minnesota, but became completely susceptible to the same race in Montana. Such a physiologic race consists of a collection of diploid zygotes which undergo segregation for various factors before the haploid sporidia that they produce fuse to constitute an infection hypha; a new series of related biotypes results which may not react to the environment like the original collection [12]. Genetical studies of the fungi concerned have shown that, in some cases at least, their pathogenic properties are inherited in accordance with the ordinary Mendelian laws. Races of *Puccinia graminis tritici* having different degrees of virulence on a particular variety of wheat have been crossed, with the result that the character of virulence has been found to depend

on a single factorial pair (or, apparently, sometimes on two pairs) present in the fungus. Tests on two or more varieties of the host indicate that the factors concerned may differ for different hosts. Perhaps the outstanding feature of the intensive breeding for disease resistance that followed the rediscovery of Mendel's laws has been the demonstration that neither host nor parasite is a single unit, but that both can be subdivided almost indefinitely (42).

Pre-penetration and Penetration Stages

When a spore rests on a suitable host plant it frequently requires free water, as drops or a dew film, for germination. The germination process has been shown in some cases to be stimulated by a diffusion of solutes from the plant through the surface covering into the water containing the spores. The spores of the conidial stage of *Sclerotinia fructicola* will not germinate in pure water but require an addition of electrolytes (salts), dextrose, or the like (47). Volatile substances given off by the underlying plant tissues have been found sometimes to have the same effect on spores not immersed in water. Perhaps the most frequently observed instance of the stimulating effects of external sources of nutrition on the infective vigour or aggressiveness of germ-tube infection is seen in *Botrytis cinerea* (10, 27, 57), where the spots caused by this fungus on leaves (primula, tobacco, etc.) are often found to begin where decaying petals have fallen and adhered to the leaf surface. In the allied grey mould of grapes it has been observed that, when the *Botrytis* conidia germinate on the surface of the grapes contaminated by juice from other grapes in the bunch that have been ruptured by insect punctures and the like, their germ-tubes show a special aggressiveness as compared with those of spores germinating in drops of water (59). Contact with withering cotyledons is also said to have a similar effect in some *Botrytis* attacks, as in grey mould of lettuce (Part II, p. 657). There are many indications that the aggressiveness of penetration of other parasites, both of roots and of the above-ground parts of plants, can be enhanced by nutritional treatment affecting the parasite. It is possible that the known effect of sterilising soil in increasing the damage caused by root parasites reintroduced from artificial cultures into such soil may be in part due to the same kind of effect, for sterilisation increases the nutrient value of the soil solution. This would not account, however, for the observed fact that the addition of very small quantities of unsterilised soil to that which had been sterilised was effective in suppressing the growth of the cereal parasite *Helminthosporium sativum* (Part II, p. 437) and in such cases the antagonistic action of soil saprophytes offers a better explanation (35).

Other conditions that may affect the parasite just prior to infection are moisture, temperature, and light. These will be more fully considered as influencing the incidence and course of disease in a later chapter, but some of their effects on spore germination should be mentioned here.

Moisture is necessary for the germination of fungus spores : some, such as most of the rust and downy mildew spores, require free water, as raindrops or dew films, for the process ; others, e.g. some of the powdery mildews, like *Erysiphe graminis* (Part II, p. 372), germinate better on a dry surface in moist air, while

complete immersion of the promycelium checks the development of the sporidia of the rusts. In *Cystopus candidus* (Part II, p. 635) it has been found that the sporangia germinate best when sown in water, if the tissues of the leaf on which they are borne have been dried so that the water content of the spores prior to sowing is reduced to about 70 per cent. of its maximum; those of *Phytophthora infestans* (Part II, p. 517) will germinate on immersion immediately after removal from the leaf only if their water content has been lowered, as by exposing the leaf to dry air for a short time; if picked while still saturated they will not germinate immediately [53]. A preliminary drying of uredospores is also sometimes favourable to their germination, and freezing, another way of drying out superfluous moisture, may have the same effect. Fresh spores of *Urocystis tritici* were found in Australia to be very difficult to germinate, but when dried over concentrated sulphuric acid for forty-eight hours germination was secured. Similarly, fungi that infect from the soil may be closely dependent on soil moisture. Club root of cabbage (Part II, p. 564) will not occur in, at least, certain soils, if the moisture content of the soil is much below 60 per cent. of its water-holding capacity, probably because a film of water around the spore is necessary for its germination. The intensity of this disease increases up to saturation. On the other hand, common scab of potatoes (Part II, p. 490) is checked by wet soil and is worst in dry seasons, perhaps because the parasite (*Actinomyces scabies*) is strictly aerobic and wet soils lose much of the air. Most smuts that infect from the soil are also favoured by moderate soil moisture, even when their spores germinate readily in water; it has been suggested that poor aeration in wet soils discourages infection in some way.

Temperature is often but little less important a factor in germination than moisture. There is, for most fungi, an optimum temperature at which germination is most free; and there are maximum and minimum temperatures, above or below which germination fails. Artificial inoculation with *Cystopus candidus* gave 95 per cent. of successes when the spores were chilled to about 10° C. (the optimum for germination), while, when the temperature was 24° C., only a few succeeded. In this fungus the maximum for germination seems to be about 25° C., and the minimum near the freezing point. On the other hand, some species of *Aspergillus* have optimum temperatures of 35° C. or over, and *Monotospora lanuginosa*, of 42° C. to 45° C., for spore germination, and 45° to 50° C., for growth [18], though these are not parasites on living plants. The optimum temperature for spore germination is not necessarily the optimum for the disease caused by the parasite. In potato blight, for instance, the germination of the sporangia of *Phytophthora infestans* (Part II, p. 520) is favoured by temperatures below those that favour the progress of the disease. Several of the wilt-inducing species of *Fusarium*, however, have an optimum temperature for germination which is higher than the optimum for the disease they induce, and the same is true for several of the cereal smuts.

Light sometimes has a considerable effect on spore formation and germination. Cultures of *Phytophthora infestans* kept in the dark tend to remain sterile, and darkened sporangia fail to form zoospores. This is not the case in the allied *Phytophthora colocasiae*. The common root parasite of tropical plantation crops, *Fomes lignosus*, can be induced to form sporophores by exposing infected roots

to light. Short exposures to ultra-violet radiation stimulate spore production in some fungi (*Fusarium eumartii*, *Glomerella cingulata*, etc.), but prolonged irradiation kills many, and has been suggested for the control of moulds on fruit, bread, and the like [64]. Exposure to ordinary visible light of high intensity has the same effect on cultures of *Helminthosporium avenae* and *Alternaria solani* [20]. It may be noted in passing that fungi are much more resistant to light than bacteria, many of which are readily killed in strong sunlight; this difference must affect the aerial dissemination in a viable condition of the two classes of micro-organisms. Very weak light suffices for the sporing of *Alternaria solani*, but complete darkness inhibits it [44]. Fairly strong natural light favours sporing in many powdery mildews, but intense continuous illumination may inhibit infection. Oxidation can replace the stimulus of light in favouring sporulation in *Plenodesmus fuscomaculans* [15]. The uredospores of rusts may fail to infect plants in darkness. In *Hemileia vastatrix* germination is best after a short exposure to strong light, but a long exposure hinders it; only the blue end of the spectrum is effective in this, the red being inert. In one experiment with this fungus an exposure of previously darkened spores for thirty minutes to the blue rays gave 27 to 36 per cent. germination, while, when exposed for an hour only three 3 to 6 per cent. germinated and, if unlighted, 9 to 17 per cent.

Sometimes the conditions prevailing at the surface of a leaf may be of critical importance in the process of infection, as in tomato-leaf mould (*Cladosporium fulvum*) (Part II, p. 675) where it depends largely on good spore germination and ease of penetration; moisture near 100 per cent. relative humidity, a temperature of 75° to 80° F., exclusion of strong direct light and abundance of stomata are favourable to germination and infection, and are found on the lower leaf surfaces at periods when outbreaks are commonest [31]. In assessing the significance of such factors as temperature, moisture, light, and so forth in the infection process it is necessary often to pay particular attention to the microclimate (the conditions prevailing at different levels within a growing crop), as opposed to the values indicated by the standard meteorological screen; the early recognition of this fact is due largely to Russian work [61].

Variations in any one factor may affect the action of the others. This can, perhaps, best be illustrated from recorded experiments on the cereal smuts [59 a]. With the oat smut *Ustilago kolleri* (Part II, p. 409) sand cultures of oats were grown at constant temperatures of 5°, 10°, 15°, 20°, 25°, and 30° C., with moistures of 15, 20, 25, 30, 35, 40, 50, and 60 per cent. of the water-holding capacity. When inoculated with the smut, infection on *Avena nuda* occurred at all the temperatures but was greatest at 15° to 25°. But, when the moisture was varied, the low moistures were much more favourable to infections than the high ones and the optimum temperature for infection was changed. At 15 per cent. moisture the highest infections were obtained at temperatures from 5° to 20°; at 20 per cent. moisture the highest infections were at 25° to 30°. With the oat variety Victor of *Avena sativa* the highest percentage of infections at 15 per cent. moisture was given by the series at 15° C., while 20° C. was best for moistures of 20 and 25 per cent. and 25° for higher moistures, at which infection was much reduced. Other experiments that will be mentioned further on show that once infection

has been successfully accomplished, the subsequent expression of the disease is not usually affected by nutritional factors influencing the rate of growth of the host plant, its height, its tillering capacity, or its rate of maturation, so that it seems safe to conclude that the effects of moisture and temperatures mentioned above were not complicated by nutritional variations. Similar experiments with the covered smut of barley, *Ustilago hordei* (Part II, p. 427) showed that soil reaction could also affect the action of moisture and temperature. With a 50 per cent. moisture, the highest percentage of infection (83·3) at 20° C. was given at a reaction of pH_5, and at 15° C. the highest (71·2) occurred at pH_6 [24].

The entry of the parasite into the host may be effected through such natural openings in the surface of the latter as stomata, water pores, nectaries, lenticels, and the like, or directly through the cuticle and outer wall of the surface cells (including root hairs) or through wounds. Even in the one parasite, different spore-forms may infect in different ways; through the stomata for uredosporal infections in the rusts; across the cuticle and epidermis for the sporidial infections in the same fungi. The first passive resistance to attack that a fungus parasite meets may be encountered here. In order to attack a leaf, a spore must remain long enough on it to germinate and produce an infection hypha, and as already mentioned, it usually requires high moisture or free water for the purpose; waxy leaves may allow the water to run off too freely for this to be possible. If it has to enter through stomata there may be few or none on the most exposed surface, and many spores may fail to reach the vicinity of one. If it penetrates through the cuticle, the latter may be too thick, or too tough, to allow free passage to the infection hypha.

Entry through Stomata and Other Natural Openings

In parasites that penetrate through stomata it is still a matter of controversy whether the size of the stomata and their behaviour in the times of opening and closing are factors in surface resistance; on the whole, the most modern evidence is against the view, which was once widely held, that stomatal behaviour has much influence on penetration by fungi, though it is of importance in bacterial infection [19]. Entry through stomata is a curiously passive process. Very many cases have been described in which fungi readily enter the stomata of plants which they are unable properly to infect. The older view that parasites were attracted by some substance of a chemical nature within the tissues has had largely to be abandoned, for even if a positive chemotropism attracted the fungus to enter, it would have to act across the stomatal cavity which is generally filled with air; the attracting substance, therefore, should be volatile and there is no evidence of a positive tropism to gases other than water vapour. Earlier work suggests that, in some cases at least, a positive tropism towards water vapour may be the cause of penetration in rusts, and this would account for entry into uncongenial hosts. There are, however, difficulties in accepting this explanation to cover all cases; experiments with the vine mildew (*Plasmopara viticola*), (Part II, p. 833) showed that the swimming zoospores of the fungus tended to congregate around open stomata, whether of the vine or many other plants, and as these spores were immersed in water, water vapour cannot have been the inducement [1]. Though

in this and a few other cases there is evidence that the stomata exert some form of attraction, it is often found that germ-tubes pass by or grow across stomata without any apparent directive influence from the latter, and it is evident that there is still much to be learned of the reasons for the predilection shown by so many obligate parasites for entry through stomata.

The actual entry is usually preceded by the formation of an appressorium (Fig. 103) over the mouth of the stoma. This may be a swelling of some size, or a mere thickening of a short length of the tip of the germ-tube. In either case it becomes fixed to the surface of the guard cells by some kind of sticky secretion, and the actual entry through the stoma is effected by a slender infection hypha arising as a branch on the under side of the appressorium. Occasionally, as in *Cladosporium fulvum*, the tips of the germ-tubes enter the stomata of the tomato leaf directly without forming an appressorium.

The fate of the infection hypha in the sub-stomatal cavity immediately after entry varies according as the fungus is able to establish itself as a parasite or not. In the rusts, the infection hypha usually swells up into a kind of a vesicle in the sub-stomatal cavity, and branches arise from this. In a susceptible host, one of these branches early sends out a haustorium into a host cell in the neighbourhood (Fig. 103 H). Food is obtained in this way (it is not at all surely known what the food is, or how it is taken in) and further growth to reach new cells into which other

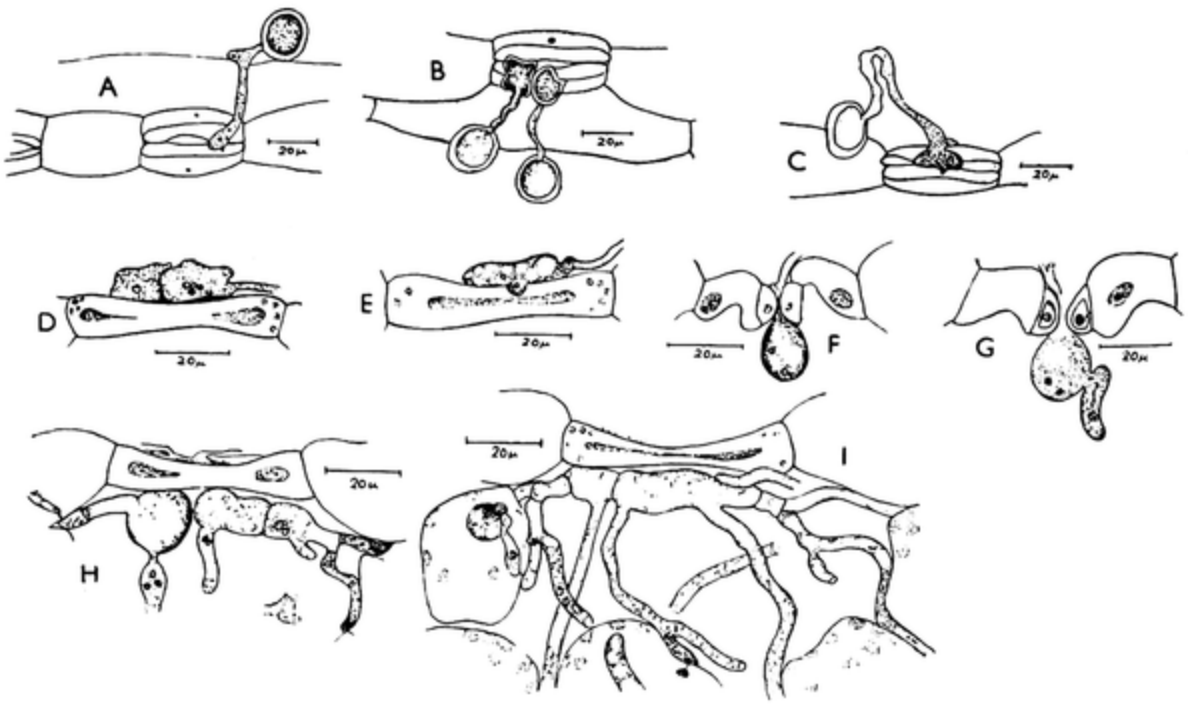


FIG. 103.—Stomatal penetration. *A–I*, germination of uredospores of *Puccinia anomala* on barley. *A*, the spore showing germ-tube, at end of which is a young appressorium (swelling), the latter producing a fine penetration peg, as if to enter a guard cell of a stoma. *B*, appressoria from two spores close to a stomatal aperture. *C*, an appressorium with a penetration peg formed over a closed stoma. *D*, section of a stoma showing two appressoria resting on a guard cell. *E*, an appressorium putting forth its first infection hypha. *F*, *G*, sub-stomatal vesicles. *H*, a two-day-old infection from two vesicles in the same sub-stomatal cavity; note haustorium sent into epidermal cell on left. *I*, a three-day-old infection; note two haustoria, the one on left in contact with host nucleus (after D'Oliveira, *Revista Agron.*)

haustoria can be sent is rendered possible. In an uncongenial host the first steps are the same, but the haustorium either fails to develop or, if formed, is unable to obtain food or absorbs something deleterious, and infection is checked after a variable amount of progress [55]. At the same time, the fungus often exerts a damaging effect on the penetrated cells or neighbouring ones, and this may cause their death and the formation of small necrotic groups of cells around the seat of infection. In some cases, as in many rust attacks on resistant varieties of cereals, the failure of the infecting mycelium to develop is ascribed to ' hypersensitivity ' of the host tissues, which are killed by the overactive parasitism of the fungus, so that the latter, requiring as it does living cells to feed on, is obstructed by a

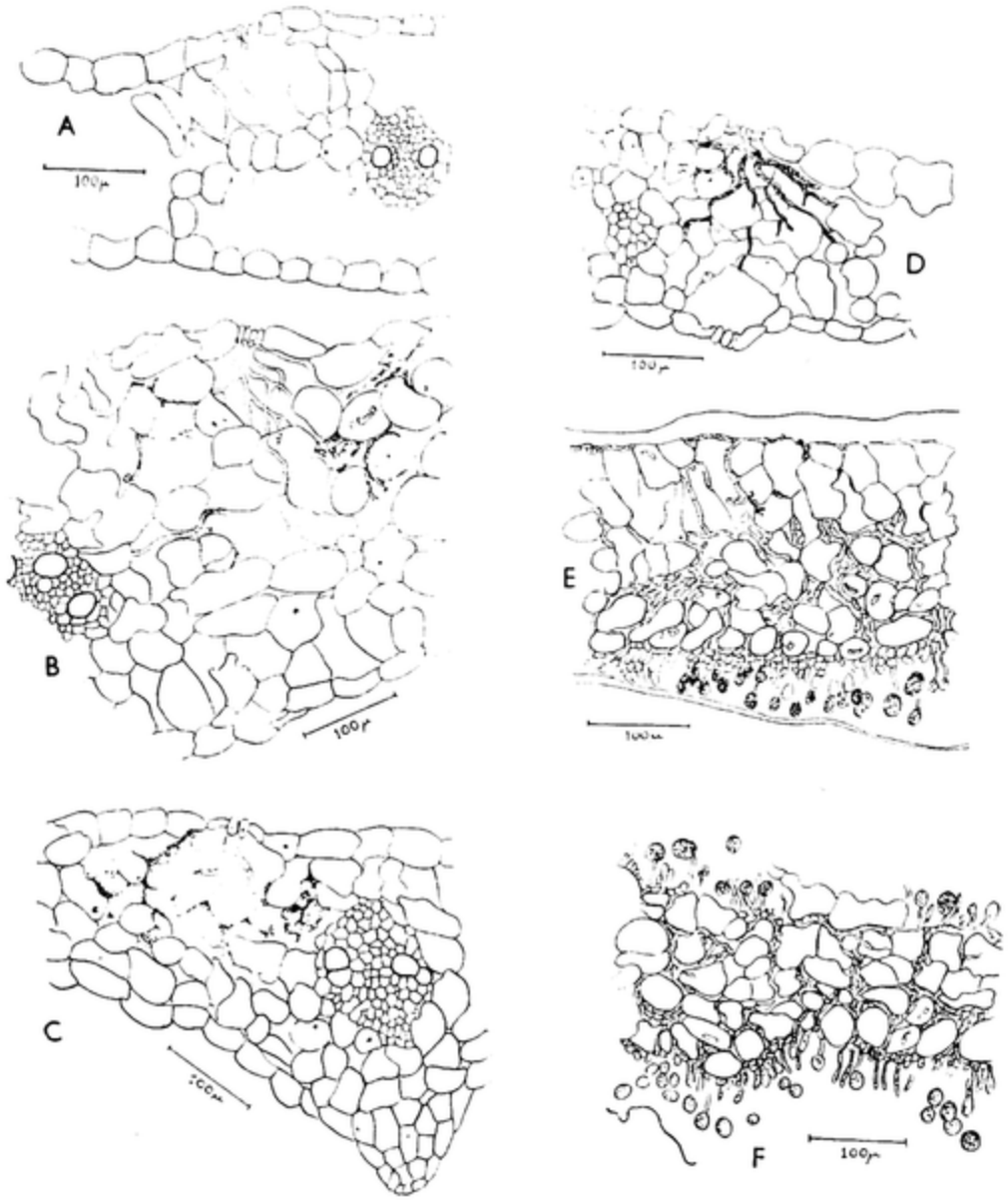


FIG. 104.—Course of infection in resistant and susceptible barley, infected with ' race 17 ' of *Puccinia anomala*. *A*, *B*, *C*, on resistant Quinn barley. Early infection in *A*, showing the sub-stomatal vesicle. *B*, rapid necrosis of host cells in contact with the mycelium. *C*, complete destruction of the mycelium, and further infection checked. *D*, *E*, *F*, on the susceptible *Hordeum vulgare pallidum*, showing congenial relations between host and parasite, total absence of necrosis, and formation of uredospores (after D' Oliveira, *Revista Agron.*)



FIG. 105.—Section of leaf of Scots pine showing penetration hypha of *Lophodermium pinastri* on the surface, and a branch hypha entering a guard cell, with the formation of a sub-stomatal vesicle from which infection hyphae are developing (after Jones, *Ann. Bot.*)

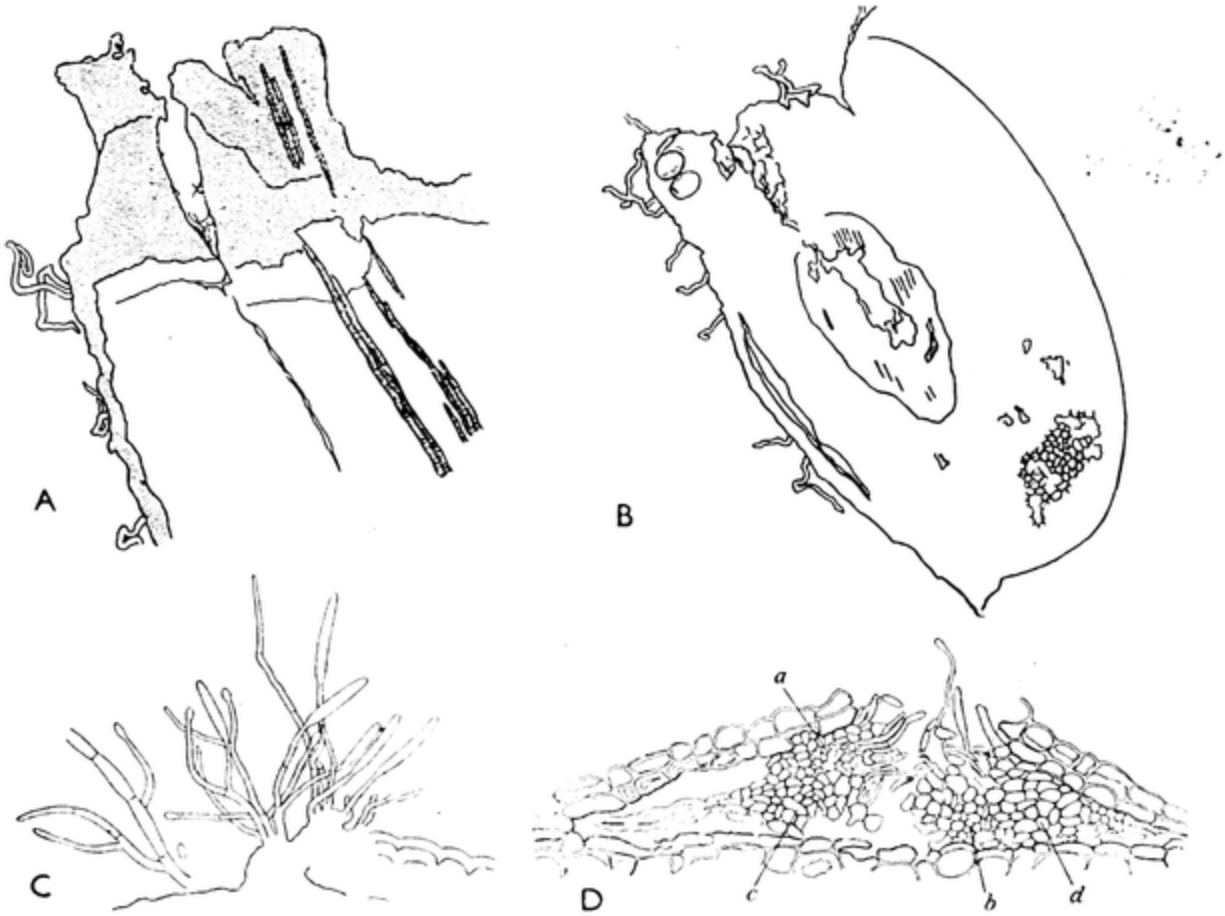


FIG. 106.—Leaf-scar infection, in apple canker (*Nectria galligena*). *A*, longitudinal section of leaf base showing crack in leaf scar and mycelium about to enter; the intercellular space in the tissue below affords an unimpeded entrance to the fungus. *B*, leaf base showing entry of fungus; there is no sign of phellogen formation and the mycelium has penetrated deeply; an infection by the scab fungus (*Venturia inaequalis*) can be seen on the outside of stem. *C*, young canker pustule on the outside of a scab infection. *D*, mycelium of *Nectria galligena*, *a*, *b*, growing on the mycelium of *Venturia inaequalis*, *c*, *d* (× 220) (after Wiltshire, *Ann. App. Biol.*)

barrier of dead tissue (Fig. 104). Similarly the tomato leaf mould, *Cladosporium fulvum*, will enter the stomata of many unrelated plants, but in unsuitable ones will either die from starvation after forming a small mycelium which appears to have no effect on the host cells around it, or will kill a few cells and produce a local necrotic patch beyond which it does not spread [8]. *Lophodermium pinastri* (Fig. 105) both sends fine feeding hyphae into the guard cells of the stomata of pine needles as well as forming a sub-stomatal vesicular structure from which branches are given off to disintegrate the mesophyll [43].

Turning to the parasites that enter their hosts through other natural openings, several plant pathogenic bacteria make use of water pores as well as stomata to gain a footing in the tissues. *Bacterium amylovorum*, which causes a destructive fireblight of apples, pears, and other hosts in North America, New Zealand, and elsewhere, enters through stomata (especially in the flowering parts), through hydathodes, and through nectaries in the receptacle. It can also enter through non-cutinised surfaces, such as those of the stigma and the locules of the anthers, and through wounds. The common green mould of citrus fruits (*Penicillium digitatum*), one of the chief causes of decay in oranges and grape fruit, can effect an entry through wounded oil vesicles in the rind of the fruit even under dry conditions when the rest of the rind is impenetrable. In the rotting of apples caused by *Penicillium expansum* (Part II, p. 721) and other fungi, penetration commonly occurs through lenticels in the fruit [39]. Infection through lenticels has been reported in the apple canker-inducing fungus *Nectria galligena* (Part II, p. 724), which can also penetrate through leaf scars (Fig 106 A), through 'woolly aphis' galls, and through wounds caused by shoot attacks of the apple scab fungus, *Venturia inaequalis* (Part II, p. 730) (Fig. 106 B). Lenticel infection also occurs in common scab of potatoes, caused by *Actinomyces scabies* (Part II, p. 488).

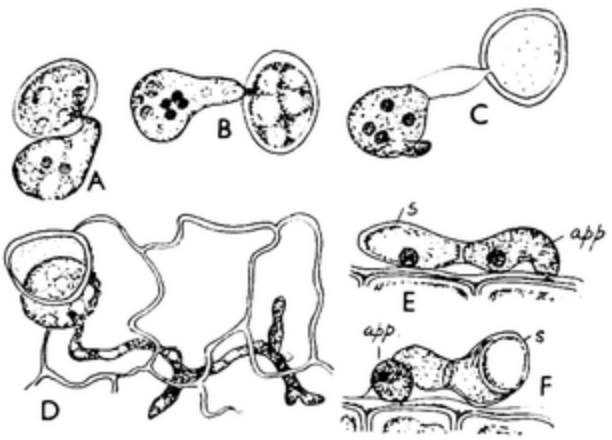


FIG. 107.—Appressoria. *A–D*, germination of aecidiospore of *Gymnoconia interstitialis*. *A*, the almost immediate expansion of the germ-tube to form an appressorium, with two nuclei. *B*, later stage showing four nuclei. *C*, development of a penetration hypha from the appressorium. *D*, growth and branching of the penetration hypha in the host (× 375) (after Pady, *Phytopathology*). *E*, *F*, penetration of apple leaf by the apple-scab fungus (*Venturia inaequalis*), showing spore *s*, appressorium *app*, produced at end of germ-tube; note the extremely narrow penetration peg entering the cuticle, and the mucilage between appressorium and cuticle (× 810) (after Nusbaum & Keitt, *J. Agric. Res.*)

ENTRY THROUGH SURFACE CELLS

Most parasitic fungi penetrate into their hosts by boring through the outer wall of the surface layer of cells, whether these be epidermal cells (or the hairs arising from them) or the secondary surface layer left in the roots and shoots after the epidermis disappears (Fig. 107). In some cases, as in the cereal diseases caused by species of *Septoria*, in 'shot-hole' of almond (Fig. 108), and in wheat bunt, the germ-tube passes down between

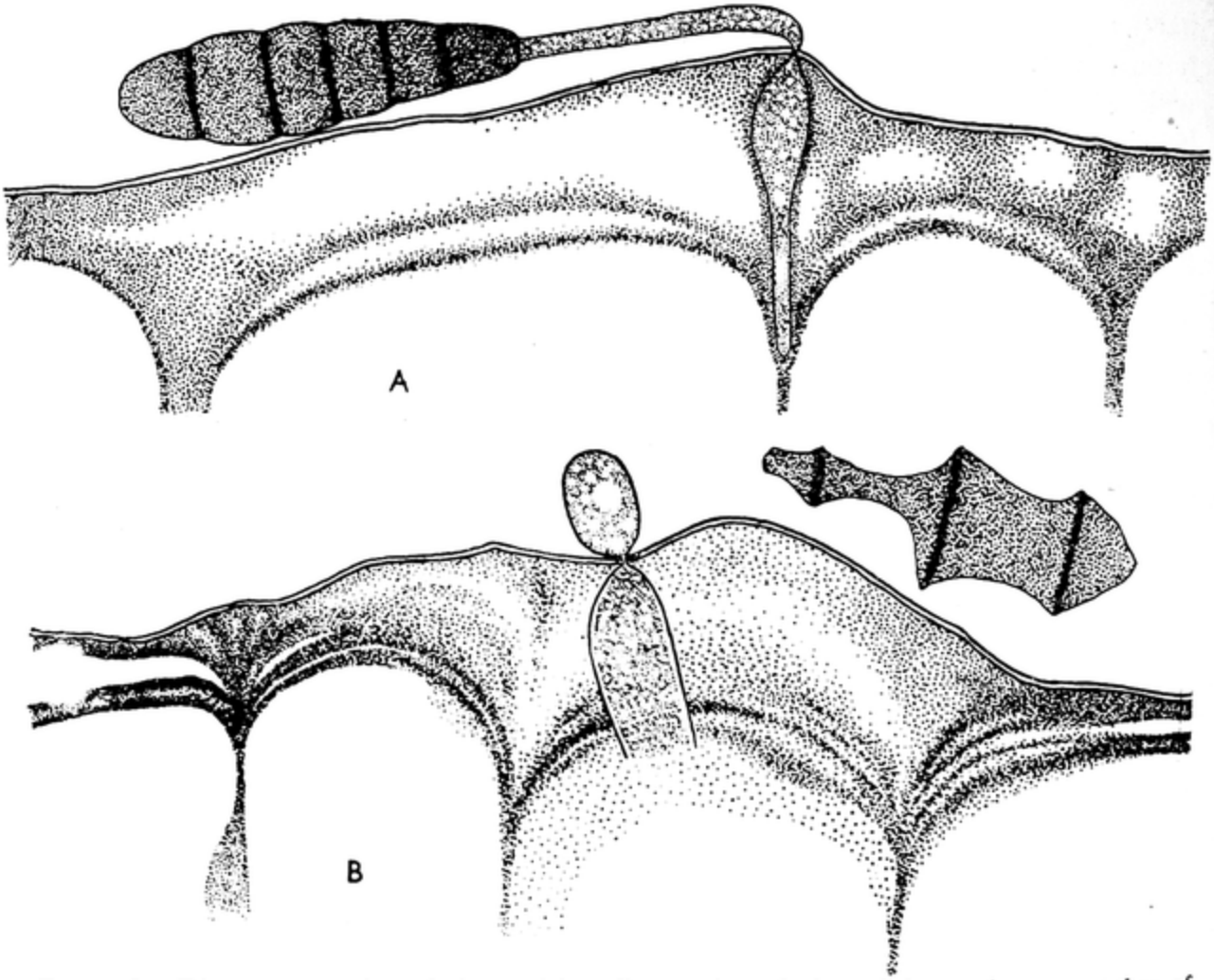


FIG. 108.—Direct penetration of the cuticle. Penetration of almond leaves by germ-tubes of *Clasterosporium carpophilum*. *A*, the penetration hyphae passing between epidermal cells. *B*, passing into the epidermal cell (× 1100 and 900 respectively) (after Samuel, *Ann. Bot.*)

the radial walls of two adjoining epidermal cells without, as a rule, actually entering these cells. Much work has been done on this type of penetration and the essential features are well established. An infection hypha of extremely small diameter arises either directly from the spore or from near the tip of the germ-tube, usually after this has been anchored to the host surface by an appressorium or equivalent holding organ. As in stomatal penetration, the directive stimulus towards the interior of the tissues is not surely known. The formation of adhesive organs may well be a contact reaction, and this is supported by the fact that they are sometimes inhibited by too moist conditions and may develop more freely as the surface dries, so as to bring them into close contact with it ; it is suggested that the stimulus to penetration may also be a contact one (haptotropic), experimental work with strips of epidermis or membranes of wax or formalised gelatine giving no support to the view once held that it is chemotropic. In the simplest case known, that of the entry of certain Chytrideaceous parasites into the hyphae of fungi such as *Pythium*, the zoospore comes to rest on the surface of the hypha, surrounds itself with a wall, and after a short time puts out an extraordinarily slender tubular process (often not a micron in diameter) which bores through the hyphal wall into the interior

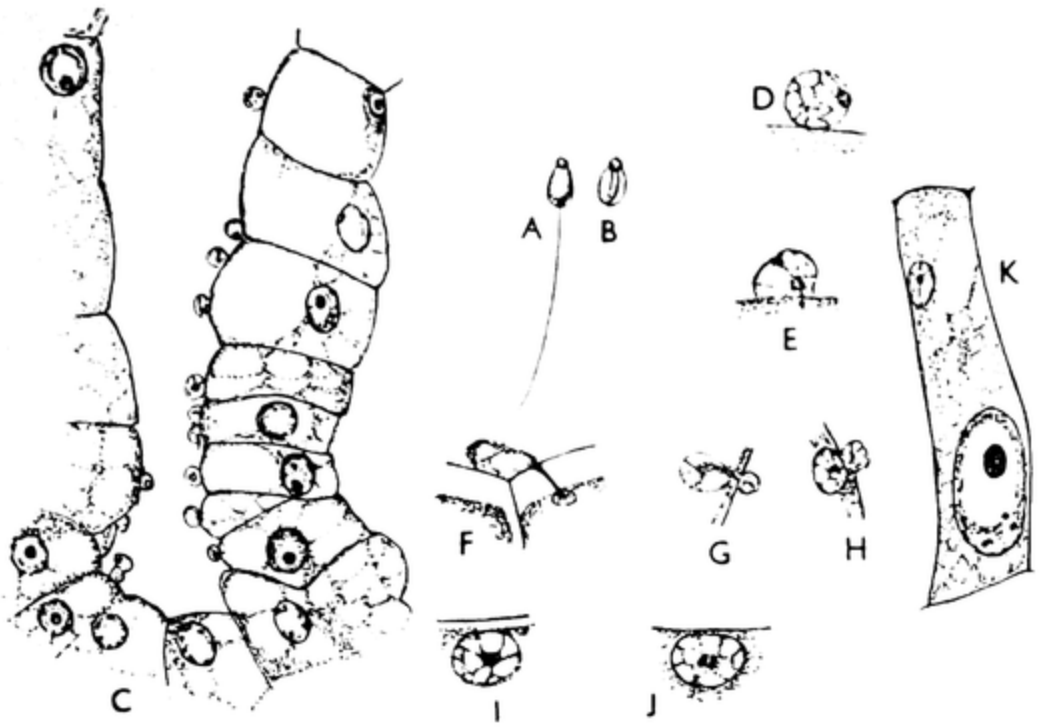


FIG. 109.—Penetration of potato tuber by zoospores of *Synchytrium endobioticum*, causing wart disease. *A*, the zoospore with nucleus and flagellum (× 780). *B*, zoospore with retracted flagellum. *C*, a number of zoospores in a cleft on the surface of the tuber (× 800). *D*, zoospore at rest on epidermal cell before entry, showing nucleus and cytoplasm. *E*, elongation of a nuclear projection towards the surface of contact. *F*, later stage of same. *G*, *H*, *I*, *J*, stages of entry into the epidermal cell. *K*, entry completed, showing the young ' prosorus ' against the cell wall, with the large host nucleus below. (*D*–*K*, × 2000) (after Curtis, *Phil. Trans. Roy. Soc., Lond.*)

and allows the contents of the zoospore to flow into the host cell, leaving an empty shell behind. In the potato wart fungus *Synchytrium endobioticum*, (Part II, p. 500), the whole of the zoospore passes into a surface cell of the host through a small pore in the cell wall (Fig. 109) [17]. The zoospores are said to be attracted by the host, especially towards cells in which nuclear division is proceeding. The opening is formed by a projection from the nucleus of the zoospore which reaches the surface of the latter where it is in contact with the epidermis. It then continues to grow, presumably accompanied by some of the cytoplasm of the spore and pierces the host cell wall. The whole of the cytoplasm and nucleus then pass through, the latter being much drawn out in the process, and on reaching the inside again rounds up. No cell wall appears on the zoospore at any stage, there being only plasmatic membranes until the ' prosorus ', or the ' resting sporangium ' stages in the development of the parasite are formed within the cell.

In all the cases that have been fully investigated penetration of the external cutinised membrane of the host is effected by mechanical pressure (Fig. 110) [10]. No good evidence, free from objection, has been given that the infection hypha dissolves a passage through the cuticle by enzyme action, as was at one time believed. Cuticle-dissolving enzymes have not been found and in experiments on the penetration of artificial membranes of graded thickness or hardness it was demonstrated that when a certain grade was reached, penetration could not be secured no matter how long this membrane was exposed to the action of the

fungus. Enzyme action can, however, come into force as soon as the cutin layer and the heavily cutin-impregnated outer layers of the membrane are passed, and there are many evidences of it in the deeper layers of the epidermal wall where the cellulose is less impregnated with cutin. Variations in the thickness and composition of the cuticularised epidermal wall are of much importance in the resistance of the host plant to parasitic invasion through the unbroken surface. In many cases resistance to this kind of attack increases with the age of the plant part concerned ; this can be linked with the fact that the outer wall of the epidermis becomes more deeply cutinised with age, and cutin deposits may then occur in the radial and even in the inner walls of the cells. Actual tests of resistance to mechanical puncture as measured by a fine weighted needle or the like have shown a correlation between this resistance and that to surface-penetrating parasites in several cases, such as barberries of varying resistance to sporidial infection from *Puccinia graminis* (51), and tomato fruits of different ages to *Alternaria tomato*.

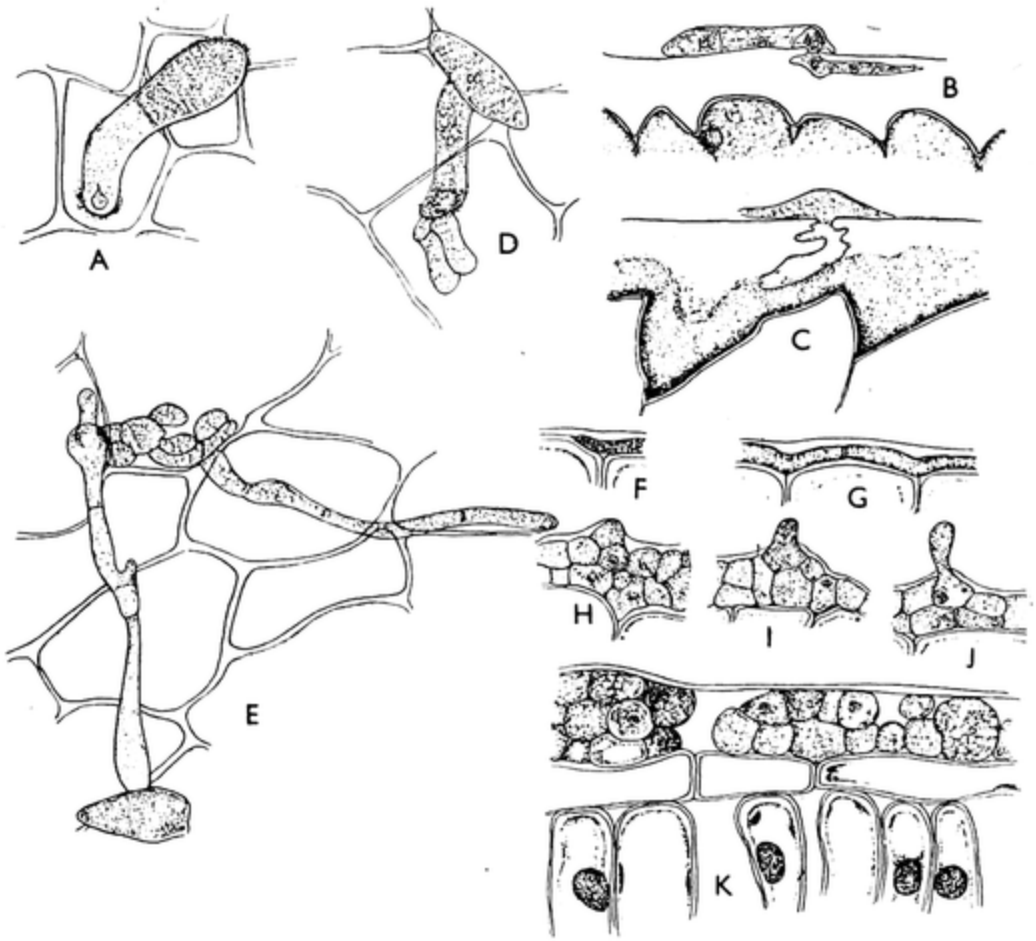


FIG. 110.—Penetration, infection, and early formation of a stroma by the apple scab fungus *Venturia inaequalis*. *A*, spore germinating on apple fruit. *B*, entrance of infection hypha under cuticle of apple fruit. *C*, the same, showing the mycelium roughly following the layer of the cuticle. *D*, *E*, spore of the pear scab fungus *Venturia pirina* germinating on leaf of pear, showing branching of infection hypha. *E*, the same, later stage, showing early formation of a primary stroma under cuticle, and of a stolon hypha (× 1600) (after Wiltshire, *Ann. App. Biol.*). *F–K*, early formation of stroma (sub-cuticular) by *Venturia inaequalis* in leaf of apple 30 days after inoculation ; note formation of the first conidiophore, and impoverishment of palisade cells and plastids (× 1000) (after Nusbaum & Keitt, *J. Agric. Res.*)

In other cases paring off the outer layers of cuticle has rendered a leaf susceptible when, if unthinned, it was immune; older barley leaves with cuticles of 2·5 to 5 μ thick, as compared with 0·4 to 1·5 μ in young ones, were found to resist attack by *Erysiphe graminis* unless thus treated, while the increased resistance of apple leaves to mildew as they age is partly due to the same cause, as it can be reduced by abrading with carborundum. The *Ascochyta* foot rot of peas (Part II, p. 619) attacks chiefly the base of the epicotyl, where there is normally a thin cuticle; in varieties resistant to the disease this thinning of the cuticle is less marked [26]. So also, since the thickness of the cuticle may depend on the intake of mineral ions from the soil solution, fertilisers may exercise an effect on the penetration of some parasites into their hosts. The fatty acids which are its base combine with these minerals to form soaps, and the readiness of their movements towards the surface, where they are oxidised to cutin, depends on the relative solubility of these soaps. The cuticle formed when calcium ions are in excess is thin, as compared with that when potassium, the soap formed by which is much more soluble, is abundant [28, 46].

The ' natural ' softening of the cuticle that occurs in etiolated plants, or those grown under too moist conditions, may increase susceptibility. A correlation has also been reported between the thickness of the outer epidermal wall of the rice stem corresponding to the accumulation of silica in it under high moisture and nitrogen nutrition, and resistance to the rice blast fungus, *Piricularia oryzae*; in dry-land rice, and on certain parts of the leaf blade infection is enhanced parallel to a reduction in the silicification of the epidermis [65, 72]. Furthermore, there is good evidence that the penetration of the epidermal wall by parasitic fungi may be affected by alterations in the wall induced in response to the threatened attack. In the *Erysiphaceae* that feed by sending haustoria from surface hyphae into the epidermal cells through the outer cutinised wall, it has been found that a germ-tube from a spore sown on a leaf of a plant which the mildew concerned is unable to parasitise, will begin to form an infection hypha quite similar to that on which the haustorium is formed in the proper host of the fungus [16, 32]. This penetrates the cuticle and induces the formation of a local internal swelling of the underlying cellulose wall, so that a small papilla projects from the under side of the wall into the cell, just as has long been known to occur when infection is successful. In successful infections, however, this papilla is penetrated by the haustorial tube and the latter enters the cell and forms a haustorium (Fig. 111). On the unsuitable host the infection tube is checked without penetrating the papilla and fails to enter the cell. This and other similar cases are, properly, examples of the reaction of the host to surface attack, and will be again referred to in the next chapter.

Mycelial Infection

Infection can be caused by the mycelial hyphae of many fungi as readily as by the germ-tubes from spores. Perhaps its most extreme example is found in fungi that infect by means of rhizomorphs, such as *Armillaria mellea* (Part II, p. 907), a root parasite of many temperate and tropical trees. Infection in this fungus is restricted, so far as is known, to the rhizomorphs, and probably to the

rather homologous xylostromata, which are more commonly found than rhizomorphs in hot countries (45, 67). When a rhizomorph comes into contact with a healthy root of a susceptible plant, it penetrates the root either by its tip if a young actively growing rhizomorph, or by a newly formed branch at the point of contact if an old one. The rhizomorph tip enters as a whole, without separation into, or preliminary penetration by the individual hyphae of which it is composed, and spreads along inside the root usually at the junction of the bark and wood, or of

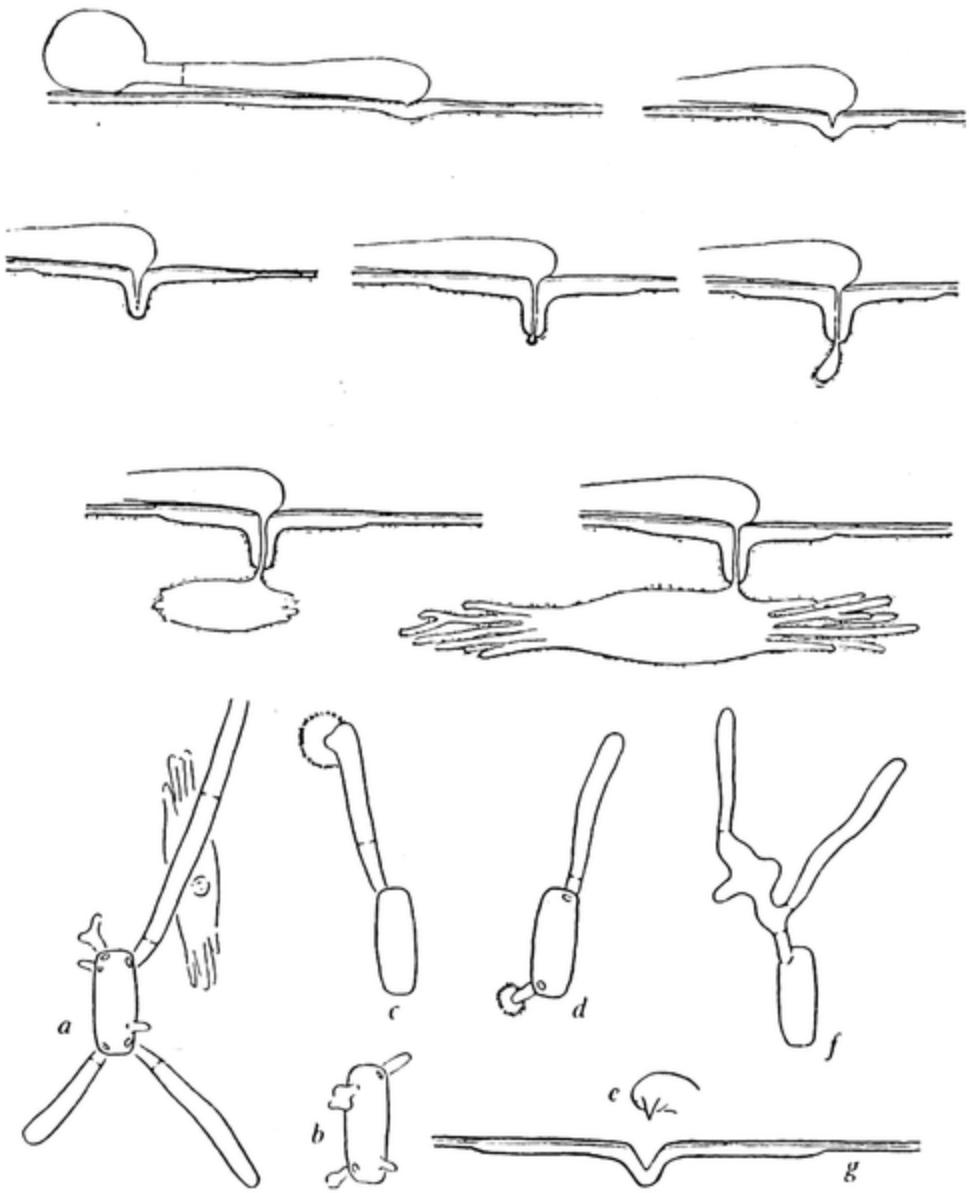


FIG. 111.—Germination of conidia of *Erysiphe graminis* on the epidermis of wheat. A fine stylar process from the germ-tube (or from a small sub-apical appressorium) pierces the cuticle, and in its passage through the cellulose layer it is preceded by a local thickening of the layer into a papilla which it eventually pierces at the apex; inside the host cell it becomes dilated into a lobed, branched haustorium, and in doing so invaginates the lining layer of cytoplasm (× 1000) (see also Fig. 82); *a*, conidium with three germ-tubes, with haustorium from the primary one (× 500); *b*, an old conidium with four tubes; *c*, a conidium with a primary germ-tube and a 'halo' (stained cotton-blue) (× 500); *d*, a conidium germinated on *Polypodium aureum*, with a halo beneath a germ-tube; *e*, tip of germ-tube with its stylar penetration peg or process (× 1000); *f*, a conidium germinated on a begonia leaf (× 500); *g*, an infection papilla, in section, with the germ-tube detached (× 2000) (after Corner, *New Phytologist*)

cortex and xylem in young roots, but sometimes in the pith (Fig. 112). It can enter through the sound periderm of the older roots or through the piliferous layer of young ones. Before entry it forms loose 'attachment hyphae' (Fig. 19), which fill all the irregularities of the root surface but do not penetrate the cells or form visible appressoria. The tip of the rhizomorph may force its way in by mechanical pressure, but there is apparently chemical action as well (see larch

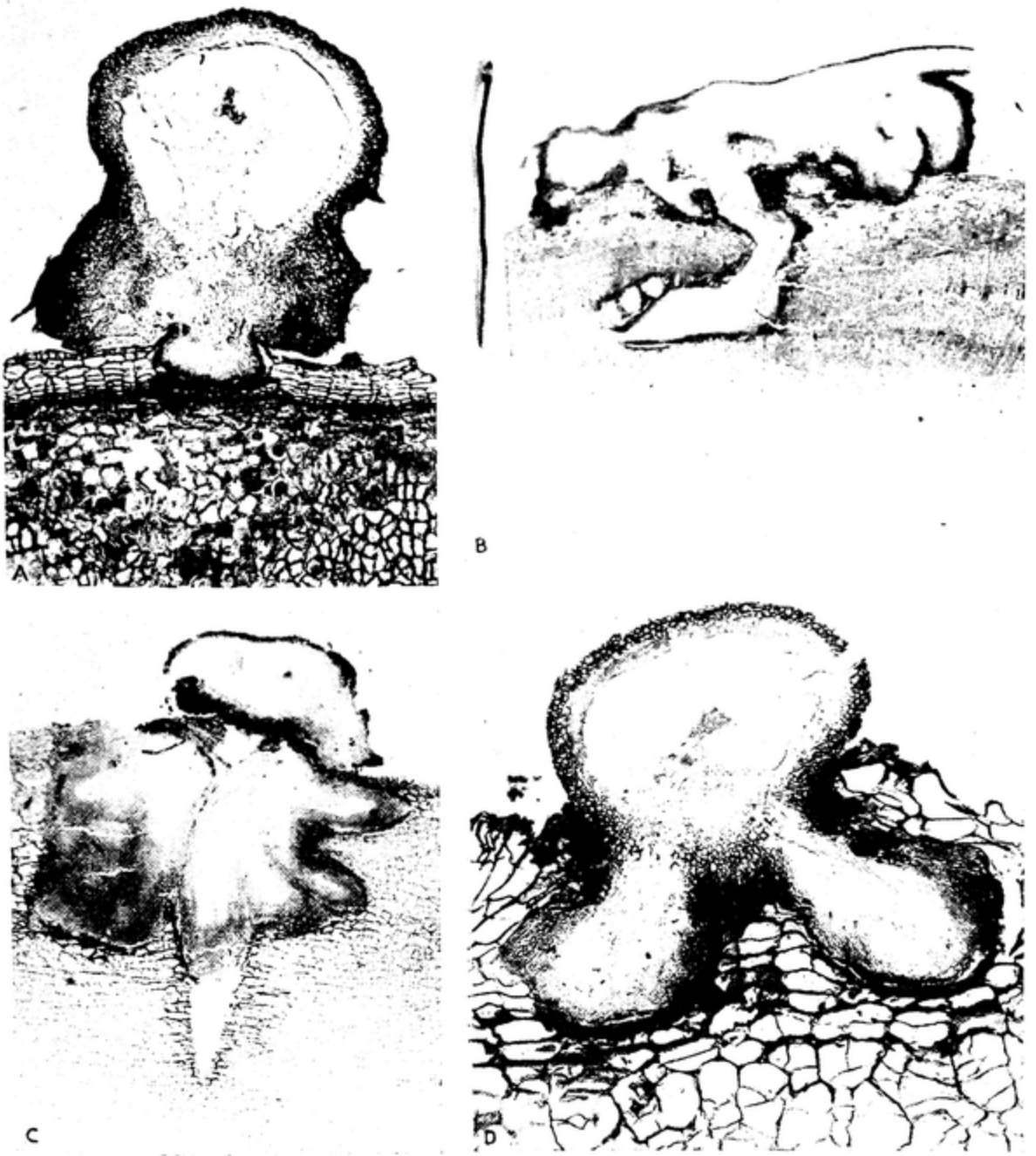


FIG. 112.—Penetration of cork by rhizomorphs of *Armillaria mellea*. *A*, cross-section of root of Persian walnut, the rhizomorph showing almost complete break-through of the cork layers. *B*, top corner, rhizomorph tip; deeper penetration and disruption of the host tissues in root of pear, the rhizomorph entering the wood below cambium. *C*, the same, invading carrot, the rhizomorph branching. *D*, the same, entering a tuber of dahlia (photos by Thomas, *J. Agric. Res.*)



FIG. 113.—*Ophiobolus graminis*. 'Runner hyphae' growing along the surface of wheat seedling root (photo by Garrett, copyright of Rothamsted Experimental Station)

root rot, Part II, p. 913). In young tea roots no attempt at defensive cork formation has been found. Behind the growing tip in the tissues individual hyphae grow out from the sides and ramify through the cells. These are, no doubt, the feeding hyphae of the fungus, and it would seem, therefore, that in fungi of this type, entry into, and extensive invasion within, the host are effected by one type of mycelium, and feeding by another. In other cases, such as, *Ophiobolus graminis* extension is effected by long 'runner hyphae' on the surface of the root (Fig. 113) and feeding by other hyphae arising from these and penetrating the roots (Fig. 114). In the parasitic 'thread blights' and 'black rots' of tropical plantation crops, caused usually by Basidiomycetes, there is, in several cases, an endophytic mycelium so restricted in space and time that the parasites concerned were long believed to be superficial and to obtain nutrition by absorption from the tissues even when these were obviously severely injured or killed by the fungus. In these cases the greater part of the fungus remains on the surface of the green parts, often forming a conspicuous white covering on them. Penetration is effected by hyphae from the lower side of this layer, and may occur only at one stage of maturity of the fungus (*Corticium keleroga*), or on one part of the plant (leaves in some thread blights). Still more remarkable is the process of infection in the western coffee leaf disease caused by *Omphalia flavida*, where the organs concerned are specialised detachable knob-shaped bodies ('gemmae') borne on short stalks, the whole forming a 'gemmifer'. The gemma, borne by the wind, falls on a leaf, usually so that its upper surface, clothed with radiating filaments, rests on the leaf. Infection occurs from these filaments. In the diseases caused by most of the above-mentioned fungi, infection from spores, though no doubt it sometimes occurs, seems to play a very minor part [54].

WOUND INFECTION

Infection through wounds is common in many fungi, especially the parasites of woody plants in which wounds are sometimes the only path of entry, e.g. in the Dutch elm disease (Part II, p. 895). In some of the destructive heart-rotting fungi of trees entry is usually effected only when the heart wood is exposed, as by the loss of large branches. In the rot of oak trees caused by *Stereum gausapatum* (Part II, p. 950), which has accounted for losses in certain forests in England,

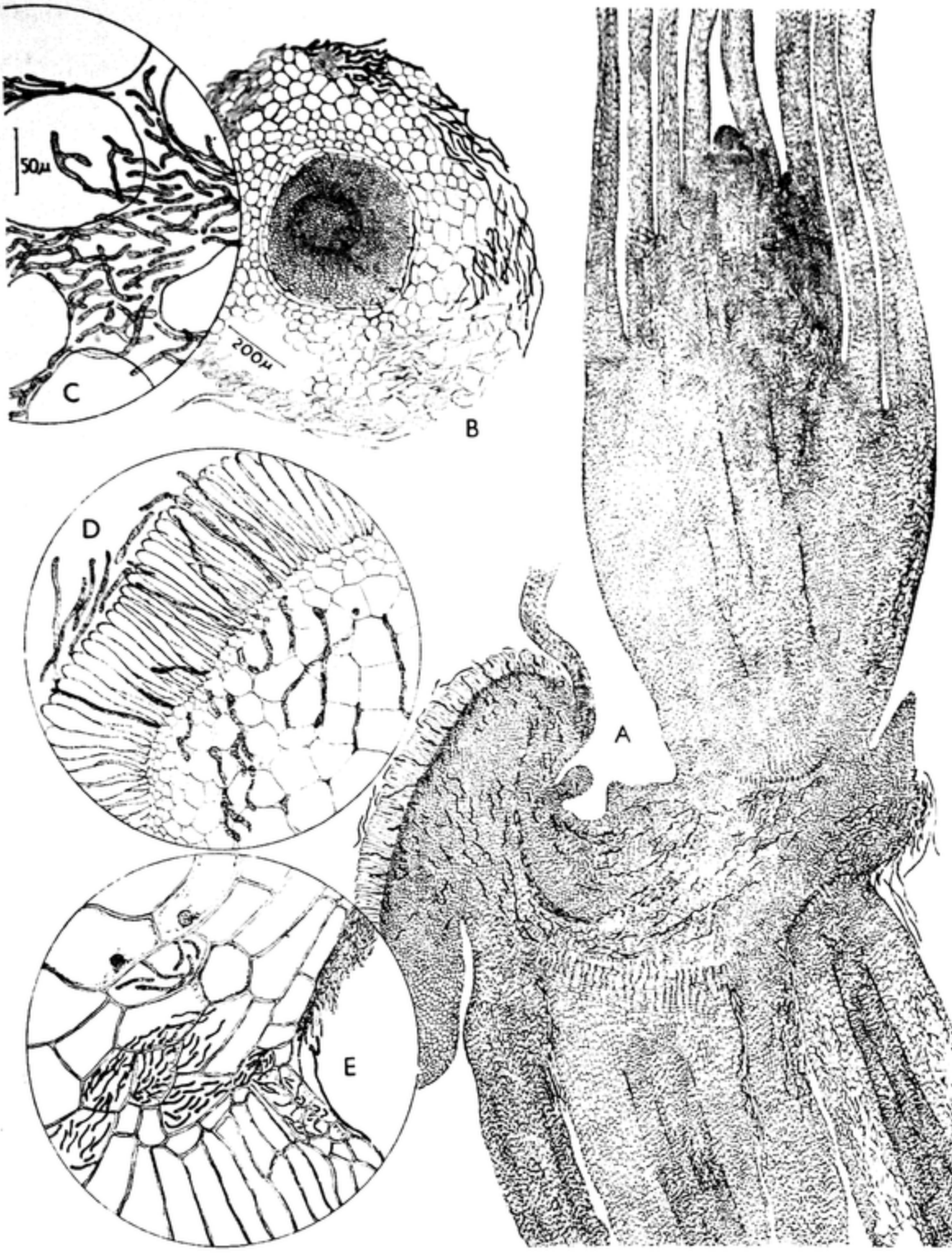


FIG. 114.—*Ophiobolus graminis*. Penetration of wheat seedling. *A*, median section through base of seedling showing, on right, the fungus penetrating the epiblast and thence travelling across base of plumule to reach the scutellum of the grain on the left (the endosperm has been omitted); note penetrations below into an adventitious root, on right, and into the coleorhiza from which the primary root may become infected. Penetrations may also be effected into the long coleoptile region above the epiblast. *B*, transverse section of a young primary root surrounded by coleorhiza (cf. *A*); note the two kinds of hyphae in the outer cells of coleorhiza, and the finer hyphae passing into the small-celled cortex of the primary root. *C*, the intercellular mycelium in the coleorhiza. *D*, the same in the scutellum and scutellar epithelium. *E*, details of the mycelium penetrating the epiblast; note the matted mycelium on the latter, and the mycelium within the cells. (Dimensions of *D*, *E*, as for *C*)

too close planting, which results in the death of large branches when the canopy closes overhead, has been determined as the primary disposing cause of the disease. In *Stereum purpureum* (Part II, p. 763), the cause of 'silver leaf' of plums and other trees, infection from spores occurs through pruning and other wounds at all times of the year except during the summer months. The reason for this, as determined at Cambridge, is that during the summer there is a rapid formation of gum barriers below the wounded surface which appears to be able to check the further progress of the fungus. A similar process occurs in the canker and die-back of roses due to *Griphosphaeria corticolax*; the resistance to this disease possessed by some varieties of roses seems to be correlated with their facility for forming gum barriers, and, even in susceptible varieties, infection through fresh pruning wounds has been found practically impossible in summer owing to rapid development of these barriers. Entry of the parasite in these cases is a passive process, the spores that fall on the freshly cut surface being drawn into the exposed vessels far enough to encounter sufficient moisture for their germination.

In other types of wound invasion the fungus may depend for its success on its speed of development relative to the rapidity with which the host forms the cork phellogen which normally occludes plant wounds. This aspect of penetration will be further considered later on.

A rather special type of wound infection occurs in the attack on oranges by the green mould *Penicillium digitatum* (5. 29). The germ-tubes from dry spores are normally unable to enter through the unbroken rind or through wounds that do not penetrate beyond it, except as already mentioned, when these are made into ruptured oil vesicles. But infection of the unbroken rind and of shallow wounds is successful if the inoculation is made with spores suspended in orange juice or dilute acids or substances causing pectic hydrolysis, such as ammonium oxalate. A suspension in orange oil has been found to have the same effect, but suspensions in water and in various synthetic culture media only infect wounds, not the unbroken surface. It is thought, therefore, that the promotion of infection by orange juice and the like is due, not to any nutritional enhancement of the aggressiveness of the parasite, such as has been suggested when decaying petals promote infection by *Botrytis*, but to a neutralising action on substances in the rind of the host that causes resistance in penetration.

Entry through Specific Parts or Organs

Very many parasitic diseases of plants are known in which entry into the plant is only possible through certain parts or organs of the plant. After entry, the parasite may infect the whole or a more or less considerable part of the plant, or may remain strictly local. It is quite possible to arrange the parasites of a given plant into those that attack the leaves, those on leaves and stem, those on the stem, those on stem and roots, and those on the roots. Sometimes it is necessary to add a group for the inflorescence and another for total 'systemic' infection. A fairly common powdery mildew (Oidium) of the mango in hot countries is often confined to the blossoms, though it can attack young leaves in moist areas (38). Many leaf-spotting fungi are restricted to the leaves, and the spots which they

cause may be quite sharply limited in size. Some may be found only on young leaves, but others, such as those producing leaf spot of strawberry (*Mycosphaerella fragariae*) (Part II, p. 785), ring spot of cabbage (*Mycosphaerella brassicicola*) (Part II, p. 639), black rot of the vine (*Guignardia bidwellii*), and leaf spot of beet (*Cercospora beticola*), are usually confined to the outer leaves. Many root parasites are restricted to the underground parts of plants, and there is quite an important group that is confined in its activities to the root-collar or ground-level part of the plant. Parasites that enter only through stomata or other natural openings are naturally limited to parts having these. In some plants there are weak spots which are made use of by certain parasites; the tough rind of sugar-cane is impenetrable to *Colletotrichum falcatum* or *Sclerospora sacchari* except near the nodes, where it is broken by shoot eyes, and by the gaps caused by the ring of adventitious root organs. *Phytophthora erythroseptica*, causing pink rot of potatoes (Part II, p. 509), infects the tubers after digging, normally only through the eyes [10a]. Similarly, the infection of cereals by species of *Fusarium* that cause seedling blights occurs readily at points where the cortex is ruptured by the emergence of adventitious roots, as well as those in the primary radicle where lateral roots emerge. Some of the wilt-inducing species of the same genus enter most easily through the root cap or the primary meristem, infections further back failing to reach the central cylinder, apparently because of the development of a suberised endodermis [23, 25]. In *Phytophthora fragariae* causing red-core disease of the strawberry (Part II, p. 780), primary infection also seems to be by entry through the tips of the roots. [36].

The smuts show many cases of restricted points of entry. In the loose smuts of wheat and barley (pp. 367, 427) it has long been known that infection occurs through the flowers. Spores blown on to the feathery stigma germinate there and penetrate the tissue, growing down the style to reach the ovary much in the manner of pollen grains. Barley, however, has also been reported to be infected by *Ustilago nuda* applied to the seed grain, the germ-tubes penetrating, when the grain germinates, through the epidermis of the coleoptile and first plumule leaves to reach the deeper tissues, though in this case it is probable that the smut concerned was really *U. nigra* [68]. In *Ustilago avenae* (Part II, p. 404) the mycelium appears to cause successful infection of oats only up to the time of the emergence of the first leaf from the sheath. It enters by penetrating the epidermis, and can do this apparently in any part of the seedling, but does not penetrate to the deeper tissues except in the mesocotyl and first internode (Fig. 121). *Tilletia caries* (Fig. 63) can infect wheat at any time from germination until a few days later, entry being by penetration of the epidermis of any part of the coleoptile. Entry through this sheath occurs usually between the cells, the infection hypha from an appressorium formed by the fusion hypha of two secondary basidiospores growing down between the radial walls of two adjacent cells which are sometimes ruptured [13]; passage into the lumen of an epidermal cell is rare. *Urocystis tritici* resembles bunt in being able to enter the wheat coleoptile in its early stages [56]; coleoptiles over 4 mm. long were not successfully infected by spore inoculations in Australian experiments. The sugar-cane smut *Ustilago scitamineae* infects through the shoot buds which can develop at every node; entry occurs through the hairs on the

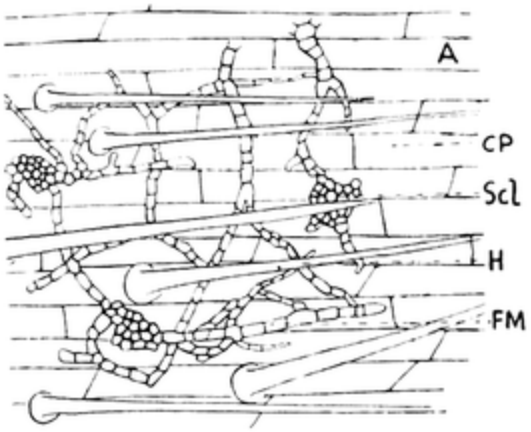


FIG. 115.—Penetration in primary infection. *Helminthosporium avenae* on oat. *A*, resting mycelium on oat husk; *cp*, husk; *scl*, imperfect sclerotia; *h*, hairs; *fm*, mycelium (× 180) (after Dennis, *West Scot. Coll. Agric. Bull.* 3)

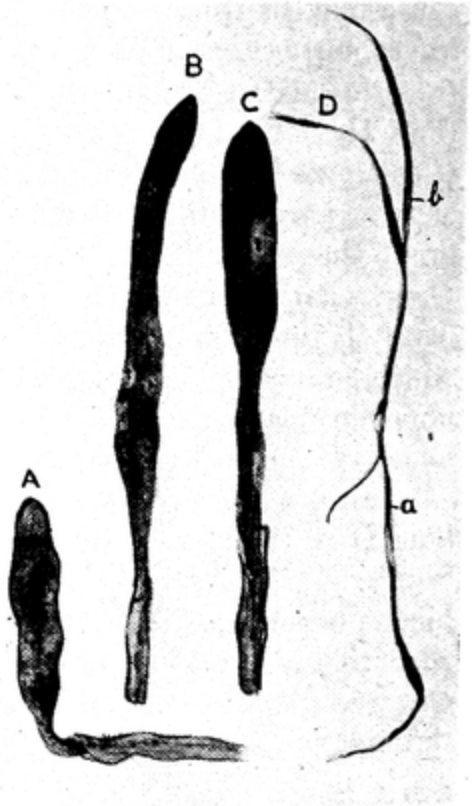


FIG. 116.—Penetration of the coleoptile. *A*, *B*, *C*, natural primary infection of the coleoptile and first leaf of oat seedlings, by *Helminthosporium avenae*, showing the characteristic distortions of the seedlings (photos by Turner & Millard, *Ann. App. Biol.*). *D*, oat seedling, artificially inoculated with mycelium of the same fungus; *a*, coleoptile lesion; *b*, leaf stripe symptoms, characteristic of leaf stripe disease of oats (after Dennis, *West Scot. Coll. Agric. Bull.* 3)

scale leaves of young buds. Leaf-axil buds, flowers at pollination, and seedlings of red campion can be infected by *Ustilago violacea* (1a, 34) and give rise to smutted flowers. Maize smut caused by *Ustilago zeae* can result from infection into any growing part. The fungus penetrates into the cells, not only of the epidermis but of the underlying parenchyma as well, unlike the other cereal smuts mentioned, which are intracellular only in the epidermis but intercellular deeper in. Even in *U. zeae*, however, the 'infection hyphae' (long runners which spread infection in the tissues) are intercellular, or, where they cross a cell, are surrounded by a cellulose sheath (see below, p. 149), and only the feeding hyphae are intracellular. This smut differs from the others on cereals in being unable to cause systemic infection; seedling penetration is obviously more likely to result in total infection of the plant than entry at a later stage.

Examples of cereal diseases arising from penetration during the seedling stage are the stripe disease of oat and barley due to *Helminthosporium avenae* and *Helminthosporium gramineum* respectively (pp. 415, 431). Successful infection results from entry into the coleoptile and may result in pronounced infection of the lowest and successive leaves, but this path does not lead to systemic infection (Figs. 115-120; 211, 217) (62a). The latter may be so complete as to involve the pollen grains, from which pure cultures of the fungus have been obtained. The grain is also infected from within, the mycelium occurring in the seed coats and endosperm; successful inoculations have been made through the flowers and also by exposing seed grain to contact with cultures of the fungus. Plants grown from seed thus infected are usually systemically invaded. In the downy mildew of the

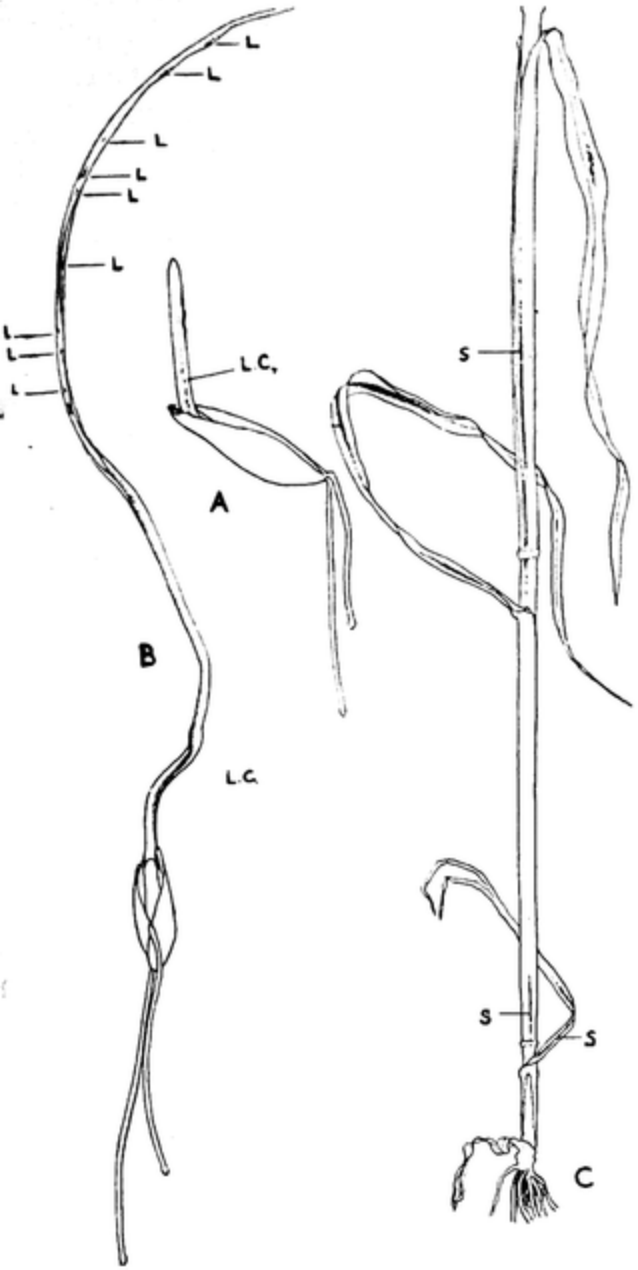


FIG. 117.—Seedling of oat showing primary infection (*Helminthosporium avenae*). *A*, early stage showing coleoptile lesion *l.c.* *B*, later stage with lesions on first leaf *L*. *C*, portion of oat plant in which primary infection has persisted throughout; *s*, leaf stripe (after Dennis, *West Scot. Coll. Agric. Bull.* 3)

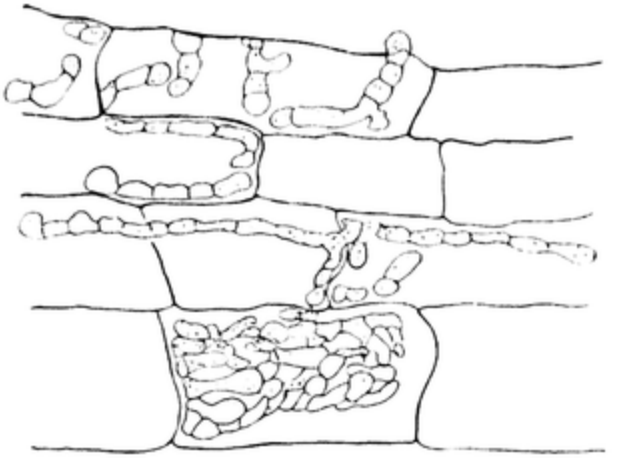


FIG. 118.—Cells of the coleoptile infected with *H. avenae*, showing intracellular mycelium (×400) (after Turner & Millard, *Ann. App. Biol.*)

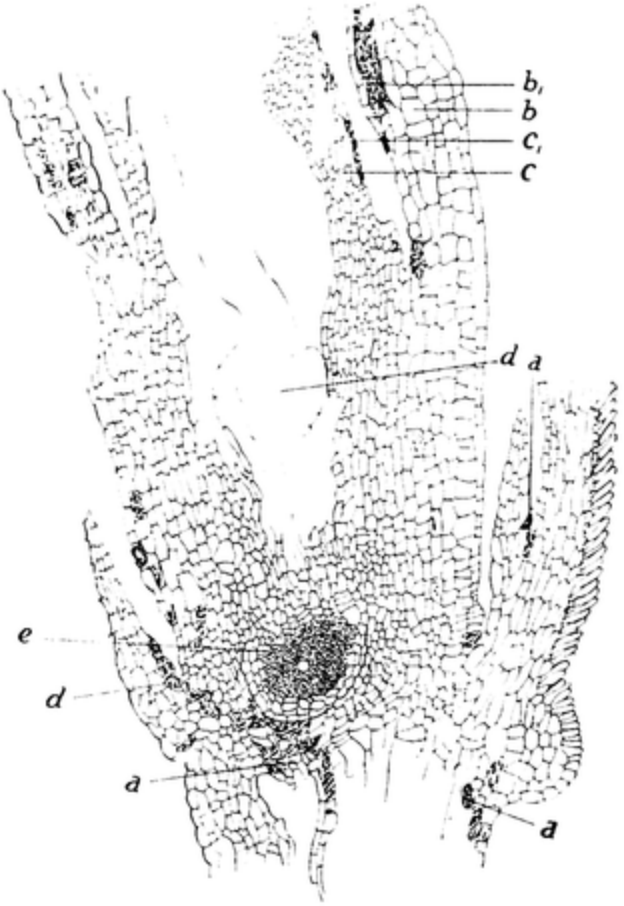


FIG. 119.—*Helminthosporium avenae* on oat (*continued*). *a*, points of penetration through scutellum and epiblast of the embryo; *b*, cells of the coleoptile; b_1, mycelium within coleoptile cells (see Fig. 118); *c*, cells of the first leaf; c_1, mycelium within the cells of the first leaf; *d*, region of the growing point; *e*, rudimentary cereal root (×250) (after Turner & Millard, *Ann. App. Biol.*)

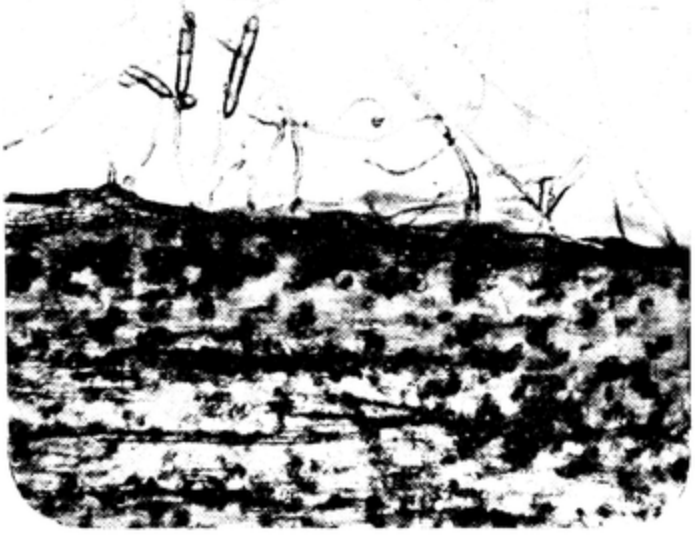


FIG. 120.—Mycelium, conidiophores, and spores of *Helminthosporium avenae* produced on oat leaves after seven days. Secondary infections would follow upon the dispersal of such spores (photo by Turner & Millard, *Ann. App. Biol.*)

hop, caused by *Pseudoperonospora humuli* (62) (Fig. 98), where there is a modified systemic infection confined to the root-stock and to peculiar spike-like shoots from this and from the leaf axils, but where considerable lengths of stem may be free from the fungus, infection occurs through buds as well as on the leaves and inflorescence (Fig. 410). Bud infection may result in the production of the systemically infected ' spikes ' and the spores borne on these can infect leaves, inflorescences, and other parts anew. Another fungus, the zoospores of which penetrate the host through buds, is *Urophlyctis alfalfae* which causes crown wart of lucerne (Fig. 224), (Part II, p. 452). Infection most readily occurs through the young leaf origins and growing points of the adventitious buds arising in succession from the woody root-stock of the host.

THE POST-PENETRATION STAGE; LIFE OF THE PARASITE WITHIN THE TISSUES OF THE HOST PLANT

So far, the discussion of the host-parasite relations has been considered mainly with the stages up to and including the first establishment of successful infection. The further discussion of the parasite within the plant, and the reaction of each to the presence of the other, brings up many physiological problems of which knowledge is still far from being complete.

The main positive advantage gained by an organism that has become adapted to the parasitic life is, no doubt, that it thereby largely escapes from the competition for food of its less specialised saprophytic fellows. Parasitism is a mode of life, and is more or less successful according to the greater or less facility it gives the vegetative body of the incomer to secure an adequate amount of food to complete its life-cycle. Obviously, the more and the longer the parasite can utilise the living tissues of its host for nutriment, the less it has to suffer from competition. Every degree of gradation is found amongst parasites, from those that are little better than saprophytes, for they must kill their food before they can use it (and are thereby frequently followed and even suppressed by true saprophytes), to those that live in a state of symbiosis with the host, doing the latter no apparent harm till the cycle of their vegetative existence is approaching completion.

Even within a single species of parasite there may be competition between

different strains. Thus, when resistant maize seedlings are infected with *Xanthomonas stewarti* the proportion of virulent to less virulent bacteria increases. In susceptible varieties, on the other hand, the proportion of virulents is reduced. As the seedling gets older, even the less virulent strains can attack it, and the suggestion has been made, therefore, that once the seedling reaches a certain age its vascular system contains organic nitrogenous compounds synthesised by the plant on which the less virulent strains can exist. In the very young seedling, where these are absent, the less virulent strains fail to become established and only the more virulent forms gain a footing [22, 48].

In the type of parasite that kills the cells of the host in advance of its progress through the tissues, the food requirement is usually simple. Most fungi of this type are readily cultivated on nutrient substances that can be easily furnished. They are facultative parasites, capable of a more or less successful existence in the absence of living hosts. Yet even in this type of parasite, specialisation of parasitism may occur and may be quite well marked, so that it is necessary that the host which they frequent should be one to their liking. Once they have crossed the barrier which the surface layers of the living plant so often present, their progress in a suitable host is fairly easy, provided that they preserve unimpaired the necessary power to kill living tissues. This they do by the excretion of substances of various kinds. The chief are probably enzymes, capable of dissolving the cell walls and directly or indirectly killing the cell-protoplasm. *Botrytis cinerea* has been more fully studied than any other representative of this type of parasite, and it is claimed that the enzymic system of this fungus, particularly the pectinase it secretes, is able to produce on the host tissue all the effects of which the fungus itself is capable. This is not necessarily true of all parasites of this class; in several cases it is claimed that while enzymes are the chief agents in attacking cell membranes, the contents are killed by other substances excreted by the parasite, organic acids such as oxalic acid and toxins.

The production of enzymes by *Botrytis* [10] has been shown to be affected by many circumstances. The composition of the nutrient medium in which the fungus grows influences the amount secreted; in media with a high carbohydrate-nitrogen ratio very little is produced, while with a reverse composition the secretion of enzyme is rapid. The enzyme is very susceptible to variations in the hydrogen-ion concentration of the medium in which it is growing, and different fungi differ in their response to this factor [52]. The substances present in the juices of different plants may have a marked effect on enzyme secretion. Tests with a group of fungi parasitic on apples but not able to attack potato tuber tissues, as compared with a group parasitic on potatoes but not on apples, showed that the addition of apple or potato juices to the purified enzyme extract, or of compounds like magnesium sulphate and potassium phosphate, could modify the behaviour of the enzymes of the different fungi and even of the same fungus. The enzyme seemed to be alike in all cases, but certain of its properties could be profoundly modified by the adsorption of substances from the medium. It was also found that the composition of the medium in which the fungus concerned grew, affected enzyme production and in some cases totally inhibited it (see above, p. 72). The reported failure of the rhizomorphs of *Armillaria mellea* to infect citrus roots unless they

were growing on the host wood is difficult to explain except in some such fashion [7], and the same seems to be true of the rhizomorphs of *Clitocybe tabescens* on pear and tung (Aleurites) trees in Louisiana [58]. The secretion of the pectinase enzyme, which is the main weapon used by *Pythium de baryanum* and *Phytophthora erythroseptica* in attacking the potato, can be inhibited by growing the fungi in various decoctions [52].

In such fungi as *Botrytis* the destructive or necrotic action of the parasite is necessary if the fungus is to live. In other parasites, however, the injury to the host is incidental and may sometimes be a positive disadvantage by shortening the time during which the organism can carry on its parasitic life. In some of these it seems that other products of the metabolism of the parasite than enzymes are of importance. What these are is not well known.

Numerous attempts have been made to assign symptoms of injury to the host to the production by the parasite of toxins, as distinct from enzymes. Many of these studies have been on the fungi and bacteria that occur mainly in the water-conducting vessels of the host and induce wilting and sometimes necrosis of the green parts, even when the parasite is confined to the roots and base of the stem. One of the earliest was with the bacterial wilt of tobacco in India caused by *Xanthomonas solanacearum* [41]. In this disease it was shown that mechanical obstruction of the vessels caused by the presence in them of bacterial masses was not enough to cause wilting, and that tissue necrosis occurred at a distance from the seat of infection. Both these symptoms were experimentally produced by the injection into the stem of a solution from cultures of the organism freed from living bacteria. At a later stage wilting is further promoted by the accumulation of gum in the vessels, and this water shortage is thought by some to be sufficient to account for the necrosis [50].

It was found in the United States that the filtrate from cultures of *Fusarium oxysporum* was as effective as the fungus itself in causing wilting of potato plants, and that the substance or substances involved were heat resistant (thermostable), unlike enzymes [73, 74]. Similarly in a wilt disease of the Michaelmas daisy at Wisley [21], caused by *Verticillium vilmorinii*, it was shown that the solutions in which the fungus was grown induced complete wilting even after they were filtered and dialysed, but it was noted that wilt-resistant varieties of the host were equally affected. A similar result occurred at Zürich with the thermostable non-volatile toxic substance obtained by growing *Fusarium lini* in culture solutions [49]; the filtrate caused wilting of the wilt-resistant Bison flax as well as of susceptible varieties, and also wilted species of *Prunus* and *Pyrus*. This fungus and *Fusarium bulbigenum* var. *lycopersici* seemed to liberate their (possibly identical) toxins mainly on the death of the mycelium.

The non-specific action of the wilt-inducing toxins seems to have been established in other *Fusarium* wilts. Other species than that responsible for the disease in nature can produce substances that cause wilting to appear in a plant. Thus, both *Fusarium oxysporum* and *F. vasinfectum* yielded filtrates that were as well able to cause cabbage to wilt as were those produced by the true cabbage wilt species, *F. conglutinans* [40]. Even the weak parasite *Penicillium expansum* (Part II, p. 721), a fungus that usually only attacks apples and other fruit after ripening, and never,

so far as is known, causes a wilt disease, was shown to secrete a thermostable non-volatile substance that could induce wilting in various plants when their cut stems were immersed in the culture solution or when it was absorbed through their roots [4].

Nevertheless, in some cases it has been claimed that the action of the toxin is specific. Thus in the *Fusarium* wilt of tomato, susceptible hosts are stated to be more readily wilted by the excretory products of *F. bulbigenum* var. *lycopersici* than resistant, and the more virulent strains of the parasite produce the most active filtrates [33].

Too much significance should not be attached to the fact that the filtrates can induce wilt in resistant varieties or in host species not ordinarily attacked by the fungus in question, when artificially introduced into the vessels. It has been established in a number of these diseases that resistance to infection in nature is a property of the cortex and not of the central cylinder (e.g. in cabbage wilt, flax wilt, some bacterial wilts).

It seems evident that various fungi and bacteria can excrete thermostable substances which, when introduced into the water-carrying vessels of the roots or stems, will cause the green parts to wilt. The evidence, as a whole, indicates that the wilt-inducing substances are not specific, and that they may act mainly by incapacitating in some way the vessels from supplying the water necessary to maintain turgor in the living cells. It has been shown, in some cases, that recovery occurs if the plants are transferred back to fresh water after wilting has been induced by growing them in the culture solution [40]. In others, however, as in bacterial wilt of tobacco, necrosis is caused and the ' toxin ' seems to act on other tissues than the vessels.

In some other types of diseases, also, there is evidence that parasitic fungi can produce thermostable substances capable of acting on tissues at a distance. The silvering effect in the leaves of plums attacked by *Stereum purpureum* (Part II, p. 763) results in an inhibition of cell division in the palisade tissue during the stage of meristematic development of the leaf, which causes the epidermis to become separated from the palisade (Fig. 361), an action at a distance to which the name ' silver leaf ' disease is due [66]; it has been reproduced by the injection of substances produced by the fungus in culture, and is attributed in part to enzymic action on the cell walls, in part to a thermostable substance which inhibits nuclear division. The wildfire disease of tobacco caused by *Pseudomonas tabaci* [14] and eye-spot of sugar-cane due to *Helminthosporium sacchari*, are examples of leaf-spotting diseases in which the symptoms can be reproduced in much the same way.

The nitrogenous and mineral nutrition of such fungi as *Botrytis* and *Armillaria mellea*, which kill the host cells in advance of their progress through the tissues, offers little difficulty, for they find in the cells nitrogenous compounds and mineral solutions which they can readily utilise. They grow well in culture if provided with meat bouillon or peptone, and in synthetic media containing similar nitrogenous and mineral compounds to those found in cell sap. It is different with their carbohydrate requirements. These they find chiefly in the form of cellulose, starch, and sugar. Cellulose must be dissolved, and that it must be more difficult to dissolve than pectin is evident from the facts that most of these fungi

first separate the cells from one another by action on the middle lamella, and that penetration of the wall is accomplished with greater or less ease according as the original pectic framework of the wall is less or more heavily thickened by the cellulose deposits on it; when lignin infiltration occurs, solution of the wall is usually much more difficult, and cutin and suberin deposits are apparently refractory. Starch must be hydrolysed, and here again, as with the wall-dissolving process, the enzymes concerned, amylases or diastases, vary from fungus to fungus or from strain to strain of a species, and are influenced even in the one fungus by the surrounding conditions. The feeding hyphae of *Armillaria mellea* in the roots of tea seedlings accumulate in the pith and deplete it of the starch which is stored there; they are not found in the more accessible cortex, which is normally devoid of starch. The fungus requires a nutritive medium rich in carbohydrates and has its maximum development where these are found. The Texas root rot of cotton (*Phymatotrichum omnivorum*) develops vigorously in the seedling roots as soon as their tissues accumulate starch, but not earlier [6]. Other root parasites such as *Rhizoctonia bataticola* develop freely in conditions of low carbohydrate nutrition and can colonise the tea root cortex, in sharp contrast to *A. mellea*. *Xanthomonas campestris*, a type of the destructive, rot-inducing organisms, hydrolyses starch and ferments the resulting reducing sugar in potato decoction media, whereas *Bacterium tumefaciens*, a growth-stimulating organism, the cause of ' crown gall ' of numerous hosts (Part II, p. 794), does not attack starch [11]. Many other parasites are known that do not hydrolyse starch, even though some of them, like *Pythium de baryanum* and *P. ultimum* (Part II, p. 507), cause rotting of the tissues. In the potato tuber rot caused by *Phytophthora infestans* the starch grains are usually unaltered until the rotted area is colonised by various bacteria and fungi that develop wet-rotting of the tubers. At the other extreme are fungi like *Claviceps purpurea* (Part II, p. 445) that utilise starch more freely than any other formed constituent of the cell contents, but as the best examples of this occur amongst the non-cultivable forms that have the capacity to establish a symbiotic state of life in the host, they will be referred to later. Even as regards the utilisation of the sugars that are usually the ultimately assimilated carbohydrates in the food of fungi there are differences in the kind of sugar assimilated by different fungi. The sugar-cane stem parasite *Colletotrichum falcatum* inverts the cane sugar contained in the host and assimilates the resulting glucose. The enzyme involved can be excreted into the surrounding medium in considerable quantity and much of the damage done by this disease from loss of purity of the juice is due to its presence. A few cases have been reported in which the parasite is stated to prefer organic acids to sugars as a source of food; the susceptibility of young fruit of the vine to black rot caused by *Guignardia bidwellii*, which ceases to attack grapes approaching ripeness, has been ascribed to this. More commonly, however, acidity is considered to impede the growth of many fungal parasites, which remain in a dormant condition after penetration has been effected in young fruit, until with increasing ripeness the fruit becomes poorer in acids and richer in sugar.

The progress in the tissues of the host of the more specialised types of parasites, especially those that are obligate parasites with no, or a very restricted, vegetative

life outside their living hosts, differs materially from that described above. In the rusts and smuts, and in some of the downy mildews, the host tissue is not killed until a late stage of vegetative development, and up to that time the injury to the cells may be imperceptible. There is no question of mutualism in the relations between host and parasite in these cases, for the host gains nothing from the fungus, while the latter obtains food from the higher plant. But the feeding is regulated so as not to be beyond the capacity of the host to meet without appreciable injury, often up to the time of sporulation.

The obligate and facultative parasites are not always easy to differentiate on a nutritional basis alone. Thus the cane gall due to *Bacterium rubi* which causes multiple galls, outwardly rather like crown gall, in blackberries and in purple and black raspberries (*Rubus neglectus* and *R. occidentalis*), in North America, is intermediate between these two extremes. It induces the development of small galls or ridges of white hyperplastic tissue, but the organism dissolves a path between the cells by its zoogloeae ; this is followed by degeneration of the protoplasts of the neighbouring cells, and lytic cavities filled with bacteria result, causing the gall to remain soft. The process is repeated as new gall tissue forms in advance of the bacteria. Some other gall-forming organisms seem to fall into the same category [3. 37].

In the majority of the obligate parasites feeding is through haustoria, and the presence of these organs is usually evidence of the status of the fungus as an obligate parasite. There are exceptions, however, as in several species of *Phytophthora* (e.g. *P. palmivora*) and in *Diplocarpon rosae* (Part II, p. 858) — the cause of black spot of roses — where haustoria occur but the parasites can grow apart from their hosts. As a rule, however, the presence of haustoria appears to signify an advanced degree of specialisation in food requirements. What the substances are that the obligate parasite requires — and that so far it has not been possible to furnish artificially, so that cultivation under laboratory conditions on non-living food has proved impossible — is not known. It is also not known why so many of the obligate parasites obtain their food through the mechanism of haustoria, organs which characteristically cause no visible damage to the living contents of the cell under ordinary feeding conditions. It is possible that the method of feeding adopted is associated in some way with the advantages gained in not killing the tissues. These advantages are not necessarily nutritional, though the nutritional gain in preserving the host cell as a functioning organ must be great. Obviously a cereal loose smut that killed its host before the latter forms ears would lose the great evolutionary advantage which the fungus has secured in developing its sporophores in such a perfect position for dissemination of the spores. Rusts on annual plants may also secure an adaptational advantage if they do not kill the first invaded leaves and check the development of others. In the other, less specialised, type of parasite the living protein of the cell has to be killed before it can be used as food, but the feeding of an obligate parasite like a rust may be a complex process, and it has been suggested that the haustoria may take in only certain essential materials, probably nitrogenous compounds, and that, once these are available, the vigour of development of the mycelium and its ability to form new haustoria may depend on such factors as the sugar supplied. It has been suggested that the

obligate parasite requires its nitrogen in the fully elaborated protein form and that the haustorium is a structure permeable to such nitrogenous substances of high molecular weight, whereas the ordinary mycelium is not.

As regards minerals and carbohydrates, there appears to be no great difference of behaviour between the facultative and obligate parasite. Starch and minerals are replaceable with little strain on the plant in view of the relatively small requirement of the parasite and the powers of replenishment of the host. One of the most clearly marked instances of starch consumption without injury to the cell is seen in the non-haustorial symbiont of the type of endotrophic mycorrhiza formed in roots invaded by *Rhizophagus*. Here, the cells containing the fungus are depleted of starch, which remains in other cells, but no injury is caused, and after the fungus ceases activity starch reappears in the emptied cells, the vitality of which seems to have been quite unaffected. Cells of susceptible hosts from which haustoria are drawing nutrients also often suffer little apparent injury, losing only such carbohydrate material as is surplus to immediate needs. In maize rust (*Puccinia maydis*) the haustoria appear to intercept the soluble carbohydrate manufactured by photosynthesis in the palisade cells before it reaches the bundle sheath, where, on bright days in uninfected plants, the surplus over what can be conducted away is temporarily deposited as starch. In infected plants this starch is lacking, in spite of the fact that haustoria are rarely found in the cells of the bundle sheath itself. In resistant strains of the host, the contents of the cells entered by haustoria tend to be injured and the chloroplasts to a considerable extent destroyed [60].

At the end of the vegetative life the fungus forms its spores. In some fungi the two stages of vegetative and reproductive activity are sharply distinguished. The life-cycle of a smut or a rust thallus is usually ended with the production of spores. In other fungi the vegetative thallus continues to grow after sporing, and many successive crops of sporophores and spores may be produced from a single infection of such a parasite as *Sclerotinia fructigena* (Part II, p. 743), the *Monilia* (conidial) stage of which occurs on rotting fruit. This feature of continued mycelial growth after sporing may even be found in obligate parasites like onion mildew (Part II, p. 693). Frequently, however, the obligate parasite ceases to spare its host from injury when sporing, and the tissues are killed locally while a smut ball or rust sporophore is forming. This is not by any means due solely to an excessive drain on the food supplies of the cells where the parasite is forming spores ; often a mechanical crushing or destruction of the tissues results from the heavy accumulation of hyphae at the base of, and around, the sporophore. Though the downy mildews are less prone actually to kill the tissues when sporing, it has been found that *Pseudoperonospora humuli* (Part II, p. 879), *Peronospora effusa* (Part II, p. 691), and *P. destructor* (Part II, p. 693) caused a reduction in the green weight of leaves of hops, spinach, and onion, of 17, 48, and 55 per cent., respectively, as a result of sporulation. The dry-matter weight of the sporangia and their stalks in *P. destructor* produced during the night of sporulation amounted to 5 per cent. of the dry weight of the onion leaf, but the loss of water could not be satisfactorily estimated [69].

Thus far, the completion of the life-cycle of the fungus from the time that

penetration has resulted in the establishment of parasitism to the sporing stage has been considered from the side of the fungus. During the parasitic life, however, the host is not inert. Against the aggressiveness of the parasite must be set the receptivity of the host, and this raises the consideration of parasitism from another angle.

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Chapter IV

PATHOGENESIS II: RESISTANCE AND SUSCEPTIBILITY

The Receptivity of the Host Plant Tissues

It has been mentioned above that the receptivity of the host plant when exposed to infection may depend on factors affecting the pre-penetration stage (waxiness or hairiness of the epidermis, etc.) or the stage of penetration (absence of stomata, toughness of the cuticle, etc.). These factors act mechanically; the subtler factors that affect the host-parasite relationships in the living tissues usually have little or no influence on the pre-penetration and early penetration stages. There seem, however, to be some exceptions. Thus, when a strain of *Rhizoctonia solani* from radish was tested on sea-kale (susceptible) and potato (resistant) penetration was rare or absent on potato leaves and sprouts, and this was shown by stripping the cuticle to be due to some internal factor in the host [31]. More usually, however, as with apple scab conidia, the infection hypha penetrates equally readily whether the host variety and fungal strain are compatible or not [22]. In the later stages of penetration, however, it has already been seen that the parasite may come under influences derived from the vital activities of the living cells. In no other way can be explained the differences in the reception which a rust or a powdery mildew encounters in suitable and unsuitable hosts. As already mentioned in the previous chapter, the thick papilla or peg on the inside of the epidermal wall which a haustorial branch of *Erysiphe graminis* is seeking to penetrate (Fig. 111) is a product of the activity of the cell. Failure to penetrate it, when *E. graminis* tries to infect an unsuitable host, must be due either to a lack of mechanical boring power on the part of the fungus or an increased resistance in the peg as deposited by the threatened cell. It is difficult to conceive how the former of these alternatives could be correct, and the latter is by far the more probable explanation. This is supported by the fact that in most highly resistant varieties of oats to smut, all attempts at penetration are frustrated by the development in the cell wall of a pad of cellulose-like substance (Fig. 121). In the less resistant varieties penetration occurs, the degree of subsequent mycelial development varying according to the grade of susceptibility [34]. Even in so simple a parasite as *Olpidium viciae* the zoospores are prevented from entering uncongenial hosts by failure of their germ-tubes to penetrate thickenings formed on the inside of the epidermal wall, though in this case it has been suggested that the presence or absence of repellent or attractive substances within the cell plays a part [16]. *Erysiphe graminis* from wheat will only grow on the older leaves of *Hordeum sylvaticum* when inoculated on the uninjured epidermis near a wound, susceptibility here being clearly conditioned by physiological factors affecting the epidermis. Similarly it is only in suitable combinations of parasite and host that infection from apple scab conidia leads to

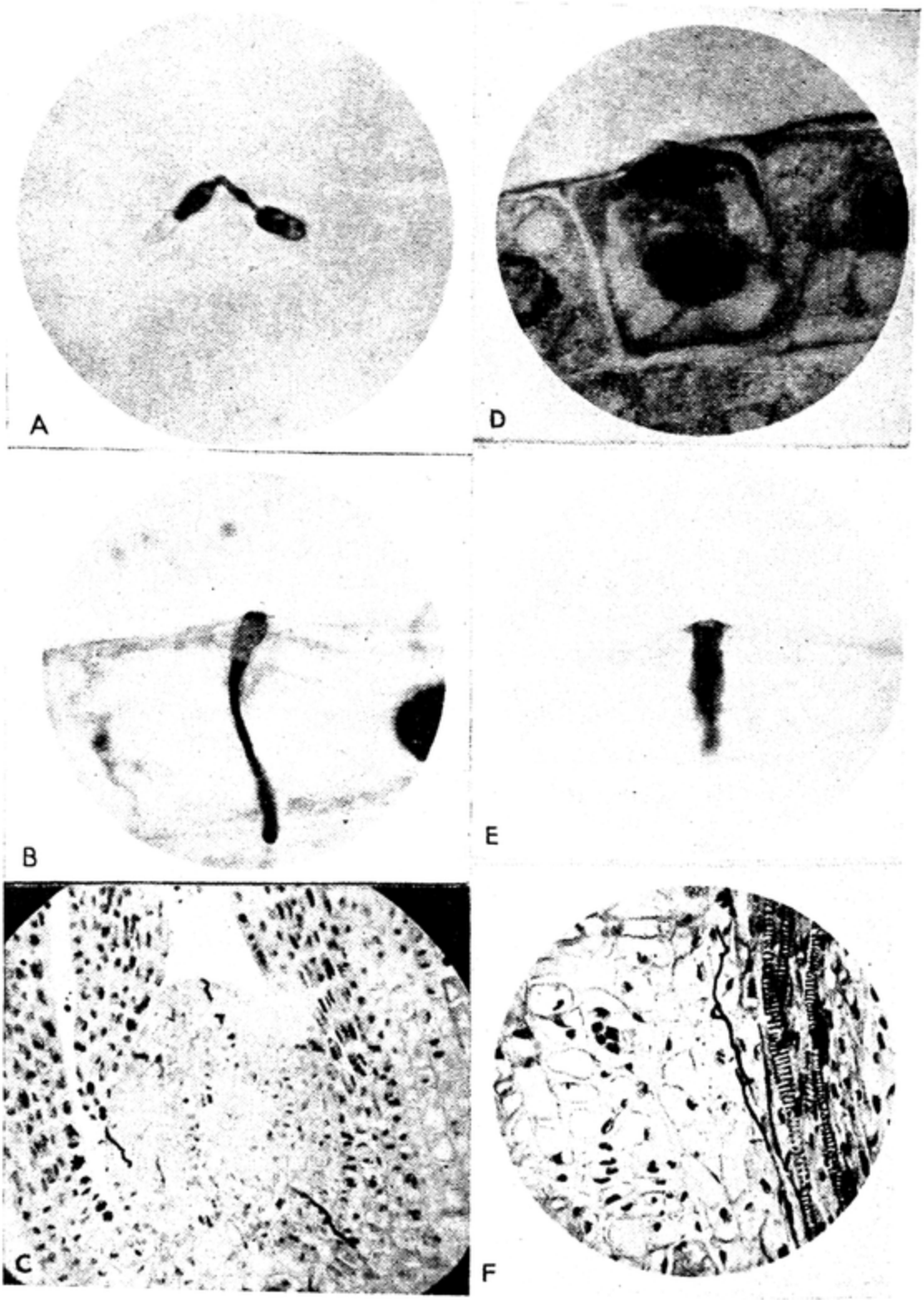


FIG. 121.—Infection of susceptible and resistant varieties of oats with the smut fungi *Ustilago avenae* and *Ustilago kolleri*. *A*, *U. avenae*, showing fusion of sporidia. *B*, *U. kolleri*, a three-day infection on the susceptible 'Markton', by strain 'C_2', showing no host reaction. *C*, effect of strain '*L*' on *Avena strigosa*, a susceptible oat, after 3 weeks; note the hyphae in the growing point (systemic infection). *D*, strain 'C_4' of *U. kolleri*, showing initial stage of penetration, after 2 days. *E*, the same, after 3 days, showing host reaction in the form of a deposit around the penetrating hypha, checking infection in this 'resistant' host. *F*, strain 'L_1' of *U. avenae* on *Avena strigosa*, resistant to it, but showing mycelium near stele of mesocotyl (photos by Western, *Ann. App. Biol.*)

nutritional depletion of the underlying cell contents. Furthermore, in the penetration of the epidermis of the roots of some orchids by the *Rhizoctonia*-type of mycorrhizal endophyte, the walls at or near the point of entry of the infecting hyphae may become greatly thickened (up to 6 or 7 μ thick) by the deposition of a suberin-like substance, and a sheath of a similar substance may be formed around the hyphae that have entered the epidermal cells, and seems to prevent their further growth. These are reactions of the threatened cells. In the rust or mildew haustorium in the cell of an unsuitable host, it is again the reaction of the host cell to the invader that is the decisive factor in determining whether the cell is killed (hypersensitiveness) or the haustorium, and often a short length of the hypha from which it is given off dies from the absorption of something toxic to the fungus. Even in the cases in which the invading mycelium is simply starved out through failure to reach its food inside the cell, the factors involved concern the receptivity of the host and not the aggressiveness of the parasite, which up to that stage is living on the food reservesof the spore, and starts haustorial development presumably with the same energy in unsuitable as in suitable hosts [11].

Influence of the Cell Membranes

The receptivity of the host depends in part on the composition of the cell membranes, in part on that of the cell contents. Parenchymatous tissues with unthickened walls are, in general, the most easily dissolved by the enzymes of the fungus, or the most easily penetrated mechanically. It is generally accepted that the penetration of internal walls is largely due to enzymic action, but this appears to be by no means universal. *Colletotrichum lindemuthianum*, causing anthracnose of French beans (Part II, p. 603), penetrates not only the epidermis but also the underlying cortical cells of beans by a process which seems to be largely mechanical, and it is only about 100 hours after entry that there is evidence of enzyme action in the cell walls. *Fusarium conglutinans* also seems to penetrate the cortical cell walls of cabbage roots by mechanical pressure. In the attack of *Pythium de baryanum* on potato-tuber tissues it has been demonstrated that there is a correlation between the resistance of the tuber tissues to mechanical penetration and their resistance to the passage of the fungus, while in *Sclerotinia laxa* on plums the softening of the fruit tissues that occurs as they ripen has been considered to be the cause of their increased susceptibility at this stage. In the soft-rotting fungi, in general, it is probable that enzymic dissolution of the walls is the main means of advance, and the same is true for bacterial pathogens. The composition of the wall is of great importance in such cases, as will be further illustrated in the section on morbid anatomy and histology. Walls that have undergone secondary thickening, those of lignified tissues, or those impregnated with suberin are much more resistant to solution than those that are unaltered. In many parasites (not in all) attacking the wood of plants the hyphae can only pass from cell to cell through the pits in the walls, the rest of the lignified wall offering an impassable barrier. So also the lignified tissues of the bundles in leaves may check the extension of leaf parasites and cause angular spots limited by the veins, as in the hop mildew (Part II, p. 879), or linear lesions, as in yellow rust of wheat

(Part II, p. 352), or the sharply defined white stripes on sugar-cane leaves caused by *Xanthomonas albilineans*.

Suberised walls oppose the passage of fungi whose offensive weapon is their enzyme system. Suberisation may perhaps also play some part in resistance to mechanical penetration, but it does not seem to be correlated with lenticel infection in potatoes resistant to common scab [36]. Similarly, though the strains of cabbage highly resistant to *Fusarium conglutinans* are the only ones in which the walls of the cortical cells of the root become suberised in advance of penetration, the reaction is not constant. The fungus readily enters through the root tip and cortex of the young root and the hypocotyl in resistant varieties, but its limitation to the outer cells in these, whereas in susceptible varieties the vascular system is reached, is not thought to be due to histological factors. So also there is no sign of the formation of reactionary cork in the seedling roots of the tea plant attacked by the rhizomorphs of *Armillaria mellea*, and the thrust of the tip of the rhizomorph is sufficient to break through the normal periderm of the older root [17]. Amongst the parasitic leaf-spot diseases there are several in which no reaction to the advance of the parasitic mycelium other than the killing of the cells can be detected, as in celery leaf spot due to *Septoria apii-graveolentis* (Part II, p. 630) where the spots are not bounded by a ring of cork but often merge gradually into the sound tissues [4]. On the other hand, the readiness with which cork forms, appears to be of real importance in limiting the attack of many parasites, especially those of the roots, stems, and branches of woody plants; in the tobacco varieties resistant to *Thielaviopsis basicola* the path of entry of the fungus through the ruptures caused by emerging lateral roots is rapidly closed by meristems in the pericycle, a reaction which occurs much more slowly in susceptible varieties [3]. Still more evident is the value of cork cambium in the cicatrisation of wounds so as to preserve them from the action of rotting organisms. There seem to be many intermediate stages of efficiency in cork barriers, from the wholly effective to the useless. Mature cork is not readily penetrated by many fungi, as is clearly seen in apple and pear scab fungi (*Venturia inaequalis* and *V. pirina*) in which work at Bristol has shown that the parasite progresses most freely where it can outflank the cork, or finds gaps in it (Fig. 122). In pear scab especially the fungus can penetrate cork in progress of formation, so that successful shoot infections occur to some distance behind the shoot tip and small stromata develop below the earliest formed phellogen. New 'wound' cork is formed inside these stromata before the end of the summer but may fail to reach the perimeter of the lesion and unite with the normal cork there in time to form a barrier completely surrounding the stroma before growth ceases. No new cork is formed during the winter, but the fungus extends through the gap thus left and develops a ring of subsidiary stromata around the primary one. These form spore-bearing protrusions as early as January, and during the next few months flakes of bark are forced away by the pressure from below and the spore-bearing stromata exposed [21]. The generally similar course of events in apple scab is sometimes disturbed by invasions of the scab pustules by the apple canker fungus *Nectria galligena* (Fig. 106 D) which develops so rapidly around the scab lesion in the spring that a sunken area of about 5 mm. in diameter, with the scab pustule in the centre, may be formed before

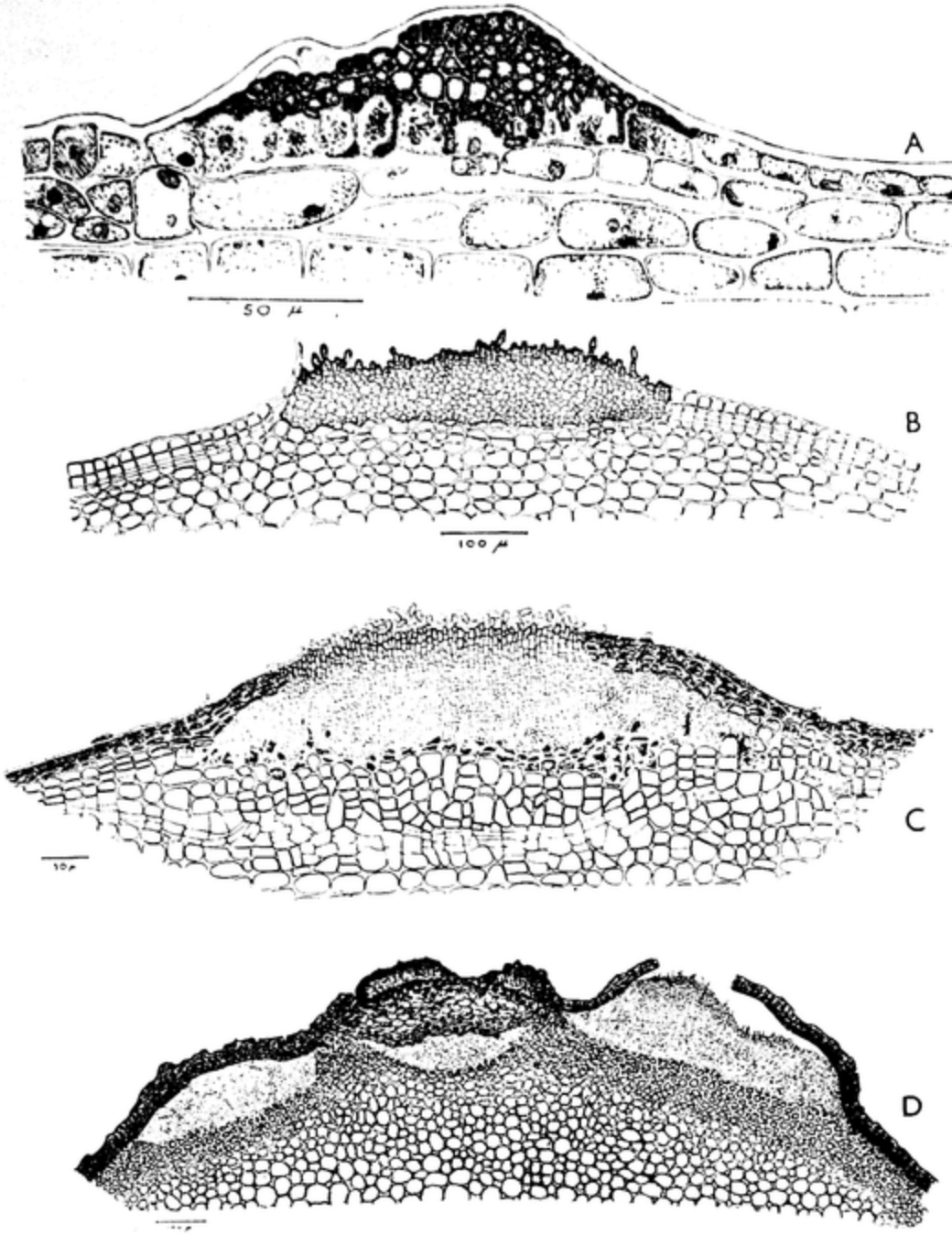


FIG. 122.—Infection in relation to cork barriers. Apple scab (*Venturia inaequalis*). *A*, section of six-day-old infection on shoot of variety Lord Suffield; the fungus has formed a pseudo-parenchyma below the cuticle, and is beginning to invade the epidermal cells; cork formation by the epidermis has not yet started. *B*, a three-week-old infection; the fungus has ruptured the cuticle and is producing spores; cells in the cortex below the fungus are dividing preparatory to forming a cork barrier. *C*, a section ½ in. from tip of the shoot; the fungus has formed a stroma about 100 μ thick, spreading peripherally beneath the bark; at the surface, conidiophores are producing conidia, and at the base may be seen partially digested cortical cells, and below them a cork barrier with its phellogen; note, on the right, a small patch of fungus not enclosed by the cork barrier. *D*, section of shoot; in the centre, the blackened remains of stroma of previous year, with dead cortical cells and cork layer below; one subsidiary pustule lies below the cork layer, and two others on either side, the one on the right having broken through the bark, and forming spores; no cork barrier formation has yet started below the subsidiary pustules (from original drawings by Marsh & Walker, *J. Pomology*, by permission of the Editor)

cork checks development. Unless the tree is sufficiently vigorous to form a cork barrier around the lesion before the wood becomes infected, the canker fungus penetrates to the deeper tissues and produces a canker of the normal type involving the wood. The canker fungus is less able to penetrate cork in the early stages of its formation than *Venturia* and it seems to find the scab lesion a suitable point through which to establish itself rapidly in the tissues during the growing season [35]. During the winter months, when cork formation is suspended, the direct inoculation of superficial wounds with *N. galligena* sometimes succeeds, but on growing shoots in the spring and summer it mostly fails.

The activity of the cork phellogen which is formed below the cut surfaces of

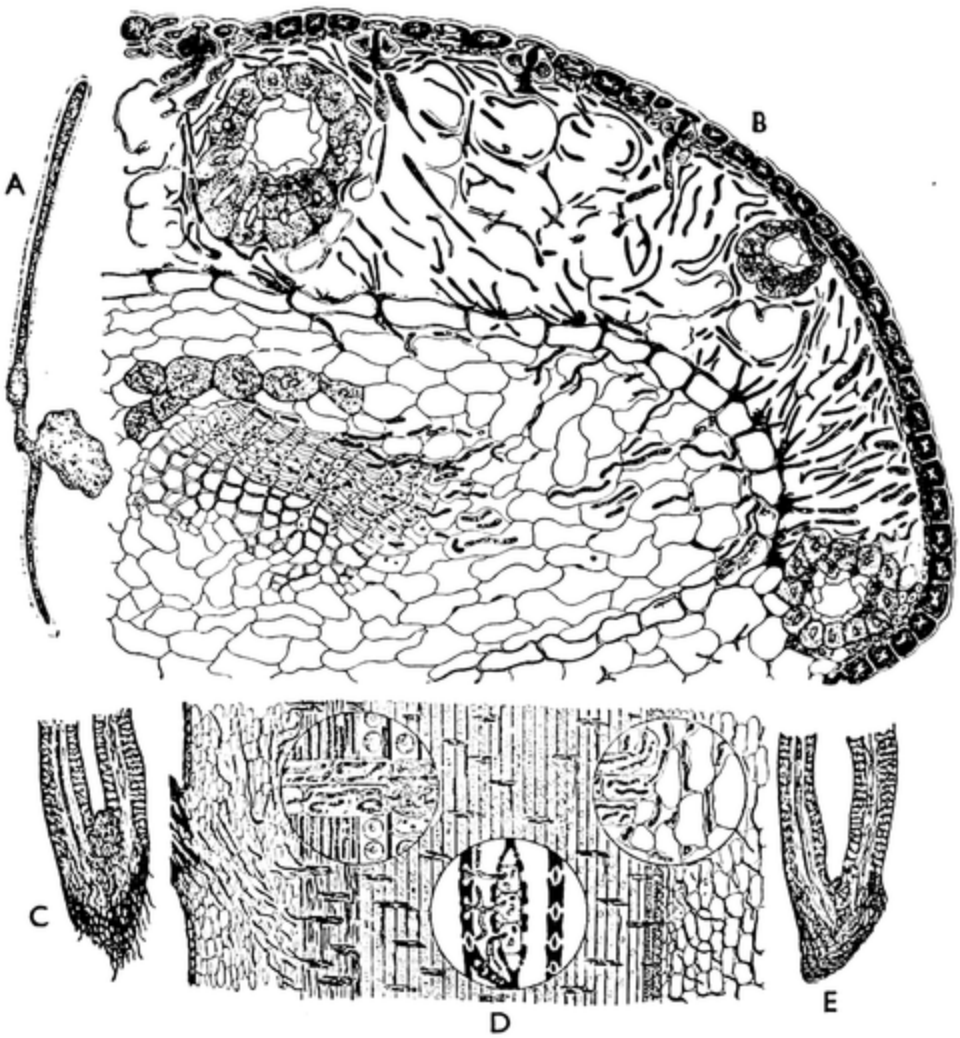


FIG. 123.—Infection of leaf and stem of Scots pine by *Lophodermium pinastri*. *A*, a germinating ascospore (in prune juice); note the vesicle. *B*, section across needle-leaf showing the fungus in the sub-stomatal cavities, and advanced destruction of the mesophyll; the fungus is crossing the endodermis by penetrating between the radial walls, thus reaching the pericycle and phloem. *C*, longitudinal section of basal part of a dwarf shoot infected with the fungus, with dense deposit of black substance. *D*, radial section of a young stem invaded by the fungus from such a dwarf shoot as *C*; the fungus traverses the cortex, living cells of the medullary rays, phloem, cambium, and the xylem, by means of the rays, thus finally reaching the medulla; three insets of the fungus in the tissues indicated. *E*, basal part of an infected dwarf shoot which has developed normal cork layers, before fall of the shoot (after Jones, *Ann. Bot.*)

potatoes differs from one variety to another (' Majestic ' and ' King Edward '. for instance, show delayed activity), and the suberised deposits that accumulate in the cells nearer the wound and that promote the formation of a phellogen may be broken if the cut surface is allowed to dry quickly, and still more if exposed to sunlight. The loss of cut ' setts ' from fungal and bacterial rots can often be traced to these delayed or faulty healing processes. The reaction of the presence of parasites resulting in wound cork formation will be further considered under the head of morbid anatomy (Chapter VI). The suberisation (' Casparian strip ') of the endodermis of roots is an effective barrier to the passage of some fungi. The best marked example of this, not however in a normal parasitic fungus, is the absolute restriction of the *Rhizophagus*-type of mycorrhizal endophytes to the cortical layers outside the endoderm. Good examples are also found in the water-melon wilt caused by *Fusarium niveum*, which, as already mentioned, reaches the central cylinder usually only when the hyphae have entered the root tip before the endodermis has developed, and in the seedling blights of maize caused by the *Fusarium* stages of *Gibberella moniliformis*, and *Gibberalla zeae* (Part II, p. 396), in which there is evidence that the endodermis in the mesocotyl and in the primary radicle acts as a barrier to the hyphae and that its efficacy, as determined by the degree of suberisation, is correlated with resistance to these diseases in some varieties of corn [33]. *Lophoderium pinastri* (Fig. 123), however, appears to be able to cross the endodermis of pine needles by passing along the radial walls to undergo intracellular development in the phloem and sometimes also the tracheids of the wood. Not only are the needles cast off in the normal way by the formation of a band of cork at the base of the dwarf shoot, but a deposition of a black substance by the fungus, in the same place, also brings about a casting off of the shoots, but the substance is not able to prevent the hyphae from passing down into the stem [13].

Influence of the Cell Contents

Leaving the conditions of receptivity based on the membranes of the host, a much more obscure set of factors concerning the cell contents must be considered. Many efforts have been made to determine the existence of chemical substances, the presence or absence of which in the cells impedes or furthers the advance of the parasite. Some light on these may be obtained from the distribution of the hyphae of endotrophic mycorrhizal fungi which avoid cells containing chlorophyll, or resin, or crystals of calcium oxalate. Those of orchid roots rarely extend to the tubers, apparently because of the presence in the latter of a thermolabile toxin, which is, however, inactive against the allied *Rhizoctonia solani*. Tannin has been supposed to be an important substance in checking the growth of the hyphae, as also has, more recently, some phenolic derivative of tannin present in the cell vacuoles. But it is hard to get positive evidence of their action. Tannin inhibits the growth of some fungi, but in the ' esca ' disease of the vine the Basidiomycete, *Fomes igniarius* (Part II, p. 949), to which the disease is attributed, grows best in culture when supplied with pyrogallic acid and only penetrates the wood of the host plant after tannin has accumulated in it. Young vines with little tannin appear to be immune from this disease, but if the tannin content is increased

(as occurs in certain soils), or if the variety is one naturally rich in tannin (*Vitis rupestris*), infection may pursue a more rapid course. An intensive study of the tannins found in species of currant (*Ribes*), which differed markedly in their susceptibility to the rust *Cronartium ribicola*, showed that the quantity present in the cells gave no measure of their reaction to the rust, but qualitative differences were found, and nothing is known regarding the influence of these [23]. As to phenols and phenolic compounds, while some inhibit the growth of most fungi, others stimulate it at dilutions not much greater than those which cause inhibition. A concentration of a phenol solution of 200 p.p.m., considerably stimulated the growth of the germ-tubes of *Puccinia graminis tritici*, whereas at 600 p.p.m., germination and growth were completely inhibited. Many of the phenolic substances found in plants have little or no toxicity to some of the fungi tested. Thus, even though tannin and phenolic compounds can hinder the advance of parasites in the tissues of plants, their effect is by no means universal. The success of the parasitic relationship between the leaf cells of oat plants invaded by the mycelium of crown rust (*Puccinia coronata*) was shown in Louisiana to depend on the liberation of phosphoric compounds from the host for the benefit of the fungus [11a].

Similarly, the relation of organic acids found in cells to susceptibility or resistance to parasites, which has been often believed to be close, appears to be so only in certain cases, if at all. It is possible to influence the acid concentration in the cell sap by mineral nutrition without, in many cases, having any apparent effect on their receptivity to a parasite. Furthermore, numerous attempts have been made to correlate the resistance of certain varieties of a given host with the acidity of the extracted sap, with such discordant results as to throw doubt on the reality of the relationship. On the positive side are cases where correlation has been found in fruits between the changes in acidity that occur during ripening, or the difference in acidity in different varieties of a fruit, and susceptibility or resistance to fungi parasitic in the fruit tissues. The progressively increasing susceptibility of apples during ripening to various apple-rotting fungi runs a course parallel to progressive diminution in the acidity of the sap. Negative results are more frequent. Several attempts have been made to correlate the reaction of resistant and susceptible varieties of cereals to rust with the acidity of their expressed juices, in general without success. It is possible to alter the hydrogen-ion concentration of the juices by various external factors to a greater extent than the inherited variations in acidity found in different varieties of the host, as by liming the soil, which lowers acidity, or by checking the growth of the plant or inducing debility in various ways which increase it, without finding any regular trend of reaction to the rust according to the greater or lesser acidity. So also there is a regular curve of acidity during the growth of wheat from the young seedling to maturity which is not followed by the curve of rust reaction, whether in susceptible or resistant varieties. The high acidity resulting from severe infection with *Erysiphe graminis* is accompanied by increased susceptibility to wheat rust, but that from etiolation by lessened susceptibility; light may be the controlling factor here, for wheat subjected to short day length and reduced light intensity becomes increasingly resistant to *Puccinia triticina* [21a], while grass rusts produce much smaller sori in darkness than in light [24]. So also the reaction to bunt of

susceptible and resistant varieties of wheat was not found to be correlated with the titrateable acidity of *p*H value of their juices.

Sugars have been considered to be important factors in tissue reaction to invasion, but no differences could be found in wheat varieties resistant to, as compared with those susceptible to *Puccinia graminis* in this respect. An indirect influence of the type of sugar formed from the endosperm during the germination of wheat and maize has, however, been established in the seedling blight of these crops in the United States caused by *Gibberella zeae*. Open-pollinated maize seedlings are susceptible to blight at soil temperatures below 24° C., at which they are low in hexose and soluble polysaccharide (e.g. cellulose) building material, but high in pentoses and soluble nitrogen. In resistant strains a similar low level in membrane-building substances and rise in total pentosan does not appear until the temperature falls below 12° C., and at such relatively low temperatures for maize the resistance of the strain breaks down. Resistance and susceptibility in this case appears to be correlated with the composition of the cell walls. Below 24° C. in the open-pollinated, and 12° C. in the resistant strains the walls remain long in the easily hydrolysable pectic condition, while at about 28° C. and 16° C., respectively, they become rapidly thickened by the deposit of cellulose upon the pectic framework ; in the resistant seedlings also, suberin is found in abundance in the cortical cell walls, whereas it is scarce or absent in the susceptible ones. The conditions in wheat as affected by temperature are, in general, the converse of those in maize. There is also some evidence that at a much later stage of parasitism a correlation can be traced between resistance of the tissues to mechanical penetration and fructification of the fungus. In rusts some of the factors which limit the formation and rupture of the uredosori seem to be the thickness of the cuticle, the amount of hypodermis formed, and the isodiametric shape of the epidermal cells [30].

Similarly, attempts to correlate the germination and germ-tube growth of the uredospores of *Puccinia graminis tritici* in total extracts of the juice with the observed resistance or susceptibility of the variety of wheat from which the juice was derived, have failed. In a series of experiments in Canada the inhibitory effect of the extracts varied from day to day, but though there was a fairly constant difference in the deleterious action of the extract from certain varieties, this was not correlated with their resistance to the rust. In the cabbage wilt (' yellows ') caused by *Fusarium conglutinans* there is no constant difference in the effect on germination of growth of the fungus in culture between extracts from highly resistant and susceptible varieties. Cases have been reported, however, in which positive correlations have been found in the behaviour as a culture medium for certain parasites of the extracted cellular juice, according as to whether it has been taken from a resistant or susceptible host. This has been reported for maize with *Ustilago zeae* [26], and for flax with *Fusarium lini* [27]. It is very questionable whether these effects are not due merely to the presence in the resistant hosts of some straightforward substance toxic to the fungus such as is well known to occur in timber. The durability of the heartwood of many trees is due mainly to certain extractives soluble in water, alcohol, or benzol which are formed as the sapwood is changing to heart wood, and are toxic to wood-destroying fungi. It

is possible to grow many of these fungi on woods which normally do not support them, provided the wood block is subjected to lixiviation in hot water before inoculation so as to remove substances antagonistic to the growth of the fungus in question. Serious decay was found to result from the attack of wood-rotting fungi on the heartwood of *Thuja plicata* only when the hot-water extractable matter was under 5 per cent. by weight of the block [2, 8, 20]. In the flax wilt caused by *Fusarium lini* there is reported to be a higher percentage of a glucoside giving rise to hydrocyanic acid on hydrolysis in the resistant than in the susceptible varieties, and this has been suggested as the cause of the difference. In the very interesting case of the resistance to onion smudge (*Colletotrichum circinans*) of the varieties of onions with coloured bulbs while those with white bulbs are susceptible, it has been found that the former contain protocatechuic acid and catechol, and that their resistance is due to the presence of these two compounds. In the other plants which this fungus can attack, resistance is shown by the formation of infection pegs which often succeed in barring its entry ; but onions do not show this reaction, and their resistance, when present, depends on the cell contents [12, 18]. Various other onion and garlic bulb parasites have been found to be inhibited in their spore germination and growth by extracts from the coloured bulbs, while extracts from white ones had a lesser effect or none (*Helminthosporium allii*, *Fusarium cepae*, *Botrytis allii*, etc.). The coloured bulbs were found to resist only those parasites which attack the outer scales where the pigment and its associated toxic substances are located ; when infection occurs through wounds leading to the inner white scales, successful infection results.

The effect of grafting resistant and susceptible varieties of plants upon one another might be expected to throw some light upon the nature of the factors concerned with reaction to disease, and especially their movement within the plant. Unfortunately the influence of stock and scion upon one another's reaction to disease has shown, what was already evident from other lines of investigation, that the factors involved are different in different cases. In some it seems clear that the substances responsible for resistance or susceptibility do not pass readily through the plant. Grafts of potatoes susceptible to and immune from wart disease are readily effected, but each component retains its inherited reaction unchanged. Even in graft hybrids it has been found that the component layers maintain their reaction to certain parasites without modification.

In several diseases of fruit trees, however, the influence of the root-stock on the susceptibility of the scion to various diseases is well marked. The effect on apple scab of some of the well-known East Malling series of root-stocks has been frequently reported and there is some evidence that the disease can be more easily controlled by spraying or dusting on trees worked on certain stocks than on others. Similarly, it has been found that Cox's Orange Pippin and Stirling Castle apples worked on the root-stocks ' XVI ' and ' XIII ' were more susceptible to canker (*Nectria galligena*) than any other stock. Stock ' XVI ' also seems to predispose the scions of several varieties to apple mildew (*Podosphaera leucotricha*) [37]. Even the susceptibility of the fruit to the storage rots caused by *Botrytis*, *Penicillium*, *Fusarium*, *Phomopsis*, and other weak parasites can be similarly affected by the stock ; Bramley's Seedling apples are more resistant when the scion is on

stocks 'IV' or 'VI' than on 'V' or 'X'. In plums the number of recoveries from 'silver-leaf' disease (*Stereum purpureum*) is significantly higher when the scion is worked on common plum stocks than when on myrobolan stocks; in one test lasting for ten or eleven years, 20 out of 23 of the former and 26 out of 41 of the latter recovered [1]. Victoria plum trees have also been found to be more susceptible to the bacterial canker caused by *Pseudomonas mors-prunorum* (Part II, p. 758), when worked on Pershore and Denniston Gage, than on stems of President, Purple Egg, and Utility. In the United States, cherries on Mahaleb stocks seem to resist infection by the 'buckskin' disease better than those on Morello or Mazzard stocks. In the opposite direction apple stocks grafted with Lord Derby, Lane's Prince Albert, or Worcester Pearmain scions are much more heavily infected with crown gall (*Bacterium tumefaciens*) at and below ground-level than those bearing Bramley's Seedling. The causes of these stock-scion influences are quite obscure.

Though the above catalogue shows that only rarely has it been possible to correlate the different grades of receptivity of the host to a parasite with special chemical substances present in or absent from the tissues, this must not be taken to imply that there is any doubt regarding the existence of such substances. There is abundant evidence that the cells contain materials which either permit or prevent the development within them of a specific parasite The absence of the particular substance may be as important as its presence. The clearest cases are found in the early stages of infection in fungi that can enter unsuitable as readily as suitable hosts; the only explanation of such a fact as that the zoospores of *Synchytrium endobioticum* can freely enter the epidermal cells of 'immune' varieties of potato but cannot develop sufficiently to cause a wart and only remain alive for a day or two in the cells which they first entered, then shrinking and dissolving, is that the cell either has something deleterious to the fungus or is lacking in something necessary to it. What these substances are is usually unknown, or even whether they are nutritive, or affect chiefly the enzyme apparatus of the parasite or the balance of osmotic pressures as between fungus and host cells. There is evidence in favour of each of these possibilities. A strong solution of pectinase enzyme in limited quantity can be rendered inert by some property of living tissue, while a rust can have its infection intensity accentuated when the leaf is borne on a stem, the cut base of which is immersed in a solution of sugar, or when the leaves, cut after infection but before the appearance of sori, are placed in a 2 per cent. sugar solution. It is difficult to separate the enzymic from the nutritional influences of the cell contents in such cases, and the distinction, so far as the intracellular life of the parasite is concerned, may be more apparent than real, for one of the major functions of the enzymes is, no doubt, to render the substances that they act upon more suitable for the nutrition of the parasite. It is, therefore, of little consequence whether the sugar solutions act by direct feeding of the parasite or by stimulating its enzymic system or giving the enzymes a more suitable environment for their activity. Some investigators attach great importance to the effect of osmotic pressure in favouring or hindering parasitism. It has, indeed, been suggested that parasitism is only possible when the fungus can maintain a higher osmotic pressure than that of the sap of the host cell [14, 32].

The presence of one parasite in a plant has sometimes been found to influence the activity of another. This may well be due to changes in the cell contents caused by the first parasite, changes which increase or diminish the activity of the second. Bearing in mind the differences in the enzyme armament of different fungi, it is not surprising to find that simultaneous infection by two or more fungi, or a successive attack by secondary parasites in the wake of a primary one, may produce results at variance with those given when each parasite acts alone. In combinations of two or more of the citrus parasites *Diplodia natalensis*, *Colletotrichum gloeosporiodes*, *Phomopsis* (*Diaporthe*) *citri*, and *Elsinoe fawcetti* tested on healthy orange trunks in Florida in 1912 this was very evident, for some of the combined inoculations killed the trees in a few weeks, a much more severe result than any could produce alone [6]. So also lemons can be rotted much more rapidly at certain temperatures by a mixed infection with blue mould (*Penicillium italicum*) and green mould (*P. digitatum*) than by either alone, while the addition of *Oospora citri-aurantii* accelerates decay at most temperatures [29]. Pink root of onions caused by *Phoma terrestris* in America is accentuated by *Fusarium malli* which is often found following the *Phoma* in field outbreaks [10]. The resistance of the leaf of certain varieties of wheat to particular physiologic races of *Puccinia triticina* may be broken if the leaf is infected with *Erysiphe graminis*. This has been observed in Warden wheat with ' form 9 ' of the rust in the United States and in Malakoff, Webster, and Democrat with several forms at Cambridge. The action extends through the leaf, although the mildew infects only cells of the epidermis, often those of the opposite surface to the rust pustules. Experiments have shown that liquid solutions in which mildew conidia had germinated stimulate the germination of the rust spores, so that it is possible that there is a passage of some rust-stimulating substance from the epidermal cells containing haustoria of *E. graminis* into the mesophyll where the rust mycelium is situated. Similarly, susceptibility to *Puccinia glumarum* may be increased if the wheat is infected by bunt. Thus the moderately resistant Little Joss wheat became susceptible at Cambridge when previously infected with bunt. In the more resistant American Club variety, however, bunt did not increase susceptibility to rust, probably because, as in the action of certain fertilisers in increasing susceptibility to rust the very resistant strains of the host do not receive enough of the modifying influence to change their reaction from resistant to susceptible. In this case the modifying influence must act at a distance, for in the mature wheat, bunt is largely confined to the apical region of the shoot, while yellow rust occurs on the leaves and ears. In Australia it has been found that the presence of *Urocystis tritici* in wheat seedlings increases injury from the seedling blight caused by *Fusarium culmorum*, and vice versa. In one test, combined inoculation with the two fungi caused a mortality of 37·8 per cent. as compared with 3·6 per cent. and 1·8 per cent. for *U. tritici* and *F. culmorum* used alone [9]. Smutted oats are said to be more susceptible to rust than non-smutted, while virus infection is commonly believed to increase the injury to potatoes caused by blight. Sometimes, however, the occurrence of a particular parasite reduces the activity of others, as in several of the lemon-rotting fungi which show lessened aggressiveness in the presence of *Botrytis cinerea*. This is possibly due to causes similar to those which reduce the

virulence of some soil parasites when certain soil-dwelling organisms are also present.

It has already been shown that many parasites can only effect an entry into specific parts or organs of the host. In their subsequent life also they may be similarly restricted. Sometimes the parasitic mycelium is only capable of growth in young or embryonic tissues. In some of the cereal smuts, for instance, the mycelium cannot continue its growth unless it early reaches the multiplying cells of the growing point (Fig. 121, *C*); otherwise it gradually disintegrates. Invasion of the whole plant by *Peronospora parasitica* is usually found only in seedlings; in older plants the fungus is often restricted to the young inflorescences (Fig. 83) or the stem galls caused by *Cystopus candidus*. The frequent association of these two fungi (Part II, p. 635) on some hosts is well known. In maize smut also the mycelium is confined to young tissues, but if the plant has been exposed to ether or heated to 70° C., total invasion may result.

The separation of the influence of the cell contents from that of the membranes is somewhat unreal, for as was shown above with the seedling blight of maize, modifications in the membrane may only reflect previous conditions in the cell contents. This holds good also with the sheaths that develop around many haustoria, and the infection pegs that so frequently appear in response to the attempt of an invasion hypha to enter a cell. Particularly good examples of infection pegs are given when seedlings grown in diffused light and high humidity are exposed to the attack of certain of the less specialised facultative parasites (*Alternaria*, *Cephalosporium*, *Helminthosporium*, *Diplodia*, etc.) which show greatly accentuated powers of penetration and a much wider host range than is ordinarily seen when the hosts are grown under more normal conditions [38]. The pegs develop on the inner side of the wall

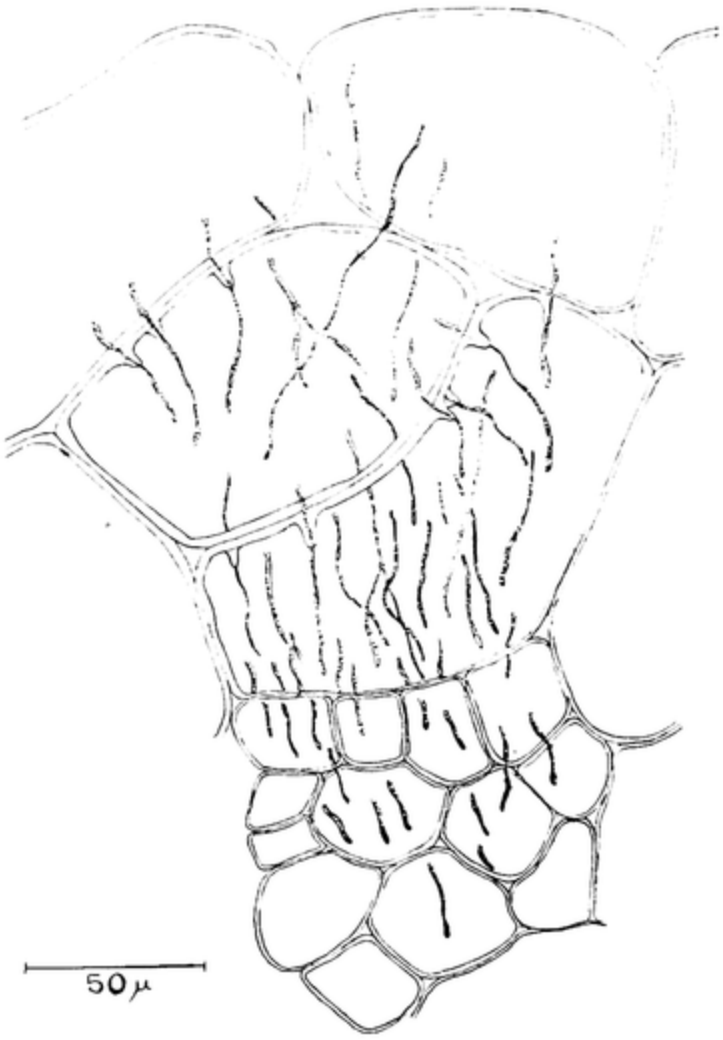


FIG. 124.—Penetration of root-cortex of wheat accompanied by swellings of inner wall of the entered cells to form 'lignitubers', in infection by *Ophiobolus graminis*. Note that lignitubers are developed more in the larger cells than in the smaller cells, entered by the fungus



FIG. 125.—Lignitubers formed by *Ophiobolus graminis* in the deeper tissues of the root (photo by Robertson)

of epidermal and deeper tissue cells, each marking a point of entry or attempted entry by a hypha. The wall in the neighbourhood is also often thickened beyond the limit of the pegs by the deposition of membrane-building substances between the outer plasmatic membrane of the protoplast and the secondary lamella of the cell wall. The pegs formed by the more aggressive parasite *Ophiobolus graminis* (Figs. 124, 125) are sometimes mainly composed of lignin with a little suberin and have been termed ' lignitubers ' (7), but they do not always give lignin reactions (19), and it seems probable that they may or may not undergo secondary modification (lignification and suberisation) according to the speed with which penetration is effected. In most cases it seems that these reactionary deposits are not composed of unaltered cellulose, but their exact composition is unknown. The same presumably applies to the ' cellulose sheaths ' deposited around the intracellular hyphae of systemic Phycomycetes (such as *Peronospora pulveracea* in *Helleborus*, and *Sclerospora* in cereals), maize smut, etc., and also to the wall thickenings referred to on p. 137. All these are formed by the protoplast and not by the pre-existing cell wall (15).

Whatever be the explanation of the variations in the receptivity of the host to a parasite within its tissues and of the consequent specialisation of parasitism that is found in all groups of parasites, facultative as well as obligate (though not in every member of the facultative group), the factors which are involved are concrete. Susceptibility and resistance to a parasite are in large measure inherited characters. The laws governing their inheritance are the same as those governing other characters of the host. Sometimes they are as simple as the simplest type known to the geneticist, sometimes complex, but in all cases in which heritable resistance has been fully worked out it is possible to relate it to the presence or absence of one or more genes for this character. It has even proved possible to locate the position of the gene for resistance to physiologic form ' 3 ' of maize rust in a plant homologous for the dominant factor for resistance to this form. It was found to be situated in the tenth (shortest) haploid chromosome. There are also indications in certain maize strains that the position of the genes responsible for reaction to smut (*Ustilago zeae*) has been identified (28). The environment can, it is true, greatly influence the intensity of a disease ; it can even modify the inherited reaction, of particular races of plants to it as will be seen in the next chapter, but in most cases it has a limited action in that it merely heightens or lowers the relative inherent resistance to the disease in question. The type of receptivity which determines whether a variety will, or will not, be attacked by a given parasite and which governs specialisation of parasitism is not easily changed in kind by external conditions. The breeding of resistant varieties of crops, which is so valuable a means of controlling plant diseases, is based on this fact.

The Genetics of Host Plant Resistance to Disease

Factorial analysis of the character of resistance or susceptibility to disease has been successfully accomplished in a number of cases by the crossing of varieties of the plant in question, and all kinds of combinations have been found. The first case investigated, that by Biffen in 1905, showed that the inheritance of the resistance or susceptibility to his strain of *Puccinia glumarum* (Part II, p. 357) in the wheats he tested followed a simple Mendelian ratio for single factor dominance of susceptibility. Subsequent work by many cereal pathologists and geneticists has shown by systematic hybridisation that the inheritance of resistance to this and other cereal rusts is not constantly dominant or recessive nor governed by a constant number of factors. Thus at Halle in Germany resistance to *P. glumarum* in spring wheats was (as at Cambridge) recessive and due to a single factor, but with winter wheats it was dominant and due to more than one factor. In other German work with 18 physiologic races of *P. glumarum* on wheat, a type of absolute immunity is distinguished from relative resistance and is said to have been dominant and unifactorial in all the crosses in which it occurred, whereas relative resistance may be due to more than one factor and be dominant, intermediate, or recessive. The forms of the rust could be arranged in groups, all the members of which behaved similarly in the reaction they produced in the progeny of the crosses tested. Reaction to the group was inherited as a unit but might be quite independent of reaction to another group.

Against *Puccinia graminis tritici*, on which most work has been done in the United States, there may be one or more factors inherited as dominants or recessives. In one series of investigations, the evidence obtained indicated that the variety Hope had a single dominant inhibiting factor for near immunity, Marquis and Reliance had a dominant factor for susceptibility, ' H–44 ' carried both these dominant factors and the resistant Ceres carried the double recessives. In another series, when the highly resistant Hope was crossed with the moderately resistant Marquillo, the inheritance appeared to depend on three or more factors and those for resistance of the Hope type did not appear to be allelomorphic to those for the semi-resistance of Marquillo. In this cross there were indications of linkage or association in the reaction to the rust of seedlings and mature plants, but in others these characters were independent. The factors for resistance to different physiologic races of the rust, as to those of *P. glumarum* and of most other parasites with specialised races, may differ ; some varieties of wheat, especially some durums and emmers, are resistant to a considerable number of forms. Evidently, therefore, the appearance of physiologic races not previously known in the area may render nugatory the breeding work which had previously been carried on in that area with a view to the development of rust-resistant varieties. Thus, in Australia, where up to 1926 only six physiologic races of *P. graminis tritici* had been found, breeding against these had resulted in the production of several rust-resistant commercial wheats. Then form ' 34 ' of the rust rapidly spread throughout the country ; it had evidently been recently introduced and proved capable of attacking all the wheats commercially grown. Breeding against it had at once to be undertaken and has again resulted in the selection of resistant varieties. Two other

forms, ' 11 ' and ' 59 ' have since appeared, but as they behave like ' 34 ' in the wheats bred against the latter, their occurrence is not so serious. In this work it was found that, whereas the resistance possessed by certain of the parent varieties to the six earlier forms (which behaved like a single unit) was conditioned by two dominant genes, cumulative in their effects, susceptibility to form ' 34 ' was dominant.

The factors controlling resistance of wheat to bunt are equally complex. Thus, in the west of the United States three main factors for resistance have been distinguished and have been termed the Hussar, Martin, and Turkey factors from certain resistant varieties containing them. The Martin factor shows absolute dominance, the other two relative dominance. But at Halle in Germany the resistance of the wheats tested there to German races of bunt was found to be recessive and due to several factors, while in Australia also a recessive factor for resistance has been found. The genetics of resistance to the loose and covered smuts of oats have also been extensively investigated. In the western United States the existence of three dominant factors for resistance to covered smuts, any one of which prevents successful attacks, has been demonstrated in hulled oats. In the eastern States, while in some crosses resistance to covered smut was dominant and dependent on a single factor, in one at least it appeared to be recessive. In some cases there was evidence that resistance to loose smut was due to the same or a closely linked factor. An interesting observation was made in crosses between two entirely resistant varieties when it was found that 25 per cent. of the F_3 progenies were smutted with *Ustilago avenae*. This suggests that the two parents had complementary factors for susceptibility which, when brought together, induced liability to smutting. In Canada the study of inheritance of resistance to covered smut in one cross indicated that it was governed by two factors, a dominant one which, when homozygous, conferred a high degree of resistance and a weaker supplementary one giving only partial resistance when homozygous. The work at Halle indicates that in Germany resistance to loose smut of oats may be due to one, two, or three dominant factors.

The breeding of wart-immune varieties of potatoes has assumed great importance in all countries, like Great Britain, in which the disease has become established. In this case it is a fortunate circumstance that there is a considerable number of varieties of potatoes that are practically immune from the disease and new ones are constantly being produced by breeding and selection. Several studies have been made of the genetics of this character, but the results have been variously interpreted. Many of the immunes appear to be heterozygous and segregate on selfing or inter-crossing into 3 immunes to 1 susceptible, on a single factor, immune dominance basis. Crossing of immunes with susceptibles gives varying results. There appear to be several factors for resistance, differing in potency. Very ingenious hypotheses have been advanced to explain the action of these; it is suggested, for instance, that the reaction is governed by three factor-pairs of different potency, and that it is only when the cumulative value of these exceeds a certain degree, which can be expressed numerically, that resistance is manifest.

On the other hand, breeding against potato blight, the cause of which, *Phytophthora infestans*, is not strictly an obligate parasite though having many points

of resemblance to one, has proved to be very difficult. The mode of inheritance of resistance to the disease is variable, dominant in some crosses, recessive in others, but usually showing much irregularity and evidently depending on interacting, cumulative or duplicate factors. Some of the complications have been attributed to the polyploid nature, not only of the potato (tetraploid), but of some of the other species of *Solanum*, such as the immune *Solanum demissum* (hexaploid), which have been used as parents. Polyploidy will also account for some of the complications that have been met with in the breeding of wheat and oats for disease resistance. A number of physiologic races of *Phytophthora infestans* have been recorded in Germany, and those that have appeared in Britain have seriously interfered with the breeding work. It is possible that the occasional occurrence of sexual reproduction in this fungus may be responsible for some of these races, and somatic mutation or saltation for others.

In another type of obligate parasite, breeding has been successful in producing wheats resistant to *Erysiphe graminis*. In some crosses in the United States a single dominant factor for resistance to particular forms of the fungus has been found, but in others the single factor was recessive. Experiments with *Erysiphe polygoni* on peas in Sweden indicated the presence in a cross between an immune and a susceptible variety of four cumulative factors for susceptibility.

Turning to the inheritance of resistance in facultative parasites studies have been carried out in several diseases caused by Deuteromycetes in which marked differences in reaction to the disease are known in different varieties of the crop concerned. The species of *Fusarium* that cause vascular wilts are often narrowly specialised in their parasitism. The wilt known as 'cabbage yellows', caused by *F. conglutinans*, has been very fully investigated, and homozygous resistant varieties of cabbage have been bred in the United States, which show complete immunity. The resistance to 'yellows' is dominant and due to a single factor which seems to be the same in different sub-species of *Brassica oleracea*, e.g. cabbage, wild cabbage, Brussels sprouts, and kohlrabi. But there is strong evidence that modifying factors for resistance occur in some of the resistant varieties that have been found; in these heterozygosity is shown by the behaviour of progenies of selfed plants and the resistance of the variety tends to break down under certain conditions. In the bean anthracnose, caused by *Colletotrichum lindemuthianum*, work at Halle in Germany showed that in the varieties and physiologic races tested, resistance was dominant and governed by three factors. Similar work at Halle on the stripe disease of barley caused by *Helminthosporium gramineum* also indicated that resistance against this disease is dominant and caused by more than one factor. Resistance to an allied barley parasite, *H. sativum*, was found in certain crosses in the United States to depend on at least three factors, each of which was linked with certain morphological and pigmentative characters.

In a few cases the inheritance of resistance to bacterial diseases has been investigated. The resistance of cotton to *Xanthomonas malvacearum* in a cross between the fully susceptible Sakel type and a resistant type of American Upland was found in the Sudan to depend on two dominant cumulative factors which can be separately inherited and only produce their full effect when both are combined.

Modifying factors were found in the American genotype. In the bacterial wilt of maize caused by *Xanthomonas stewarti* resistance was found in the United States to be dominant and probably due to two major complementary genes, with perhaps a third modifying one. The factors involved in reaction to the bacterial wilt of lucerne, however, proved to be so complex that genetic analysis was not found practicable in preliminary studies.

Acquired Immunity

It has been shown above that there are many types and grades of resistance to disease in plants. In certain types the resistance is due to a reaction of the cells or tissues of the host to invasion by the parasite. The hypersensitive reaction, by which, for instance, a cereal becomes immune from rust, only occurs after the parasite has come into close contact with the internal tissues. It is natural, therefore, that the question whether there is in plants a type of induced or acquired immunity, such as is so important a factor in animal and human pathology, should have received much attention.

The organisation of plants is much simpler than that of animals in the sense that each component part is less closely linked with, and less dependent for its functioning on, the integrity and well-being of all the other parts. There is no central nervous system, no blood or lymph streams, and every living part has, or may have, within it the capacity to regenerate the whole plant. Analogies drawn between what occurs in animal disease, where any local disturbance or localised necrosis may have rapid repercussions on distant parts, and disease in a plant where an organ such as a leaf or branch may be lost with little or no effect on the rest, must necessarily, therefore, be limited in measure. Unless one of the vital channels of translocation, such as the phloem, or the main water supplying system is involved, what happens in a localised plant tissue is of little concern to the plant as a whole ; the ' fevers ' of plants, though these are known, are limited to the region around the exciting cause [5, 25].

In the majority of cases where the resistance of a plant to a disease can be heightened by artificial means, such as drainage of the soil, use of balanced fertilisers and so on, it is probable that some cause of weakness in the plant is being removed, so that normal vigour is being restored. The difficulty here is to know what constitutes normal vigour and the normal behaviour of a given plant to a particular disease, for without knowing these, it is uncertain whether resistance is being heightened or weakness removed. If increased resistance due to an acquired immunity similar to that conferred on animals by vaccination or serum treatment can be induced in plants, it should be possible to do so in diseases which can develop vigorously in plants that are growing luxuriantly as, for instance, in those caused by many rusts and other active parasites (see p. 180). If this immunity depends on the development of antibodies, as in induced animal immunity, evidence of the presence of these should be given when the plant is reinfected after a short lapse of time.

In spite of claims to the contrary, no good evidence of the existence of antibodies elsewhere than in the immediate vicinity of the site of infection by fungal

or bacterial parasites appears to exist. It has been claimed that in rusts and powdery mildews of perennial plants (e.g. coffee rust and oak mildew) a certain degree of resistance is conferred by a severe attack in the preceding year. As such an attack will result in early leaf-drop, the immunising effect would have to be far reaching for the newly formed leaves to show it in the following year. There is, however, no evidence of the development of immunity from diseases of this type, even in the immediate neighbourhood of the first infections. On the contrary, the first infection in many diseases, from potato wart and blight to the cereal rusts, is the more rapidly followed by new infections in the neighbourhood, the more actively the fungus develops and produces new spores; except where the tissues have actually been injured by the first attack, the secondary ones are not less vigorous. As to the inducement of systemic immunity in perennial plants, there is no evidence of it in scab of apples or pears, leaf-curl or rust of peaches, hop mildew, or other diseases of orchard or garden crops, where if such a systemic induced resistance occurred it would be most likely to be noticed.

In experiments to determine whether antibodies are formed in response to introduced antigens, as in many animal and human diseases, discrepant results have been obtained by different workers. Some have found that a first infection with the crown gall organism prevents the development of galls on re-inoculation on the same stem to a varying but often considerable distance. Others have quite failed to obtain similar results. Even where success has been reported, the suggestion has been made by some of the investigators that it is due, not to a formation of antibodies, but to autolysis or to the presence of a bacteriophage in the cultures used for inoculation. Similarly, several investigators have succeeded in conferring immunity on plants by vaccination or other preliminary treatment with an attenuated strain of the parasite or with the culture media in which the latter has grown. The most important experiments have been carried out with *Botrytis cinerea*, the attack of which was found to be prevented or reduced when the host plant was watered with the culture solution in which the culture had been grown. Not only have such results failed of confirmation by other workers, but it seems to be going beyond the evidence to regard them as comparable with the use of antigens. Similar results were obtained with an aqueous extract of *Helminthosporium sativum* on wheat, but subsequent work showed that extracts of various other fungi and of *Pseudomonas fluorescens* had the same effect. There seems, therefore, to be no clear proof of the existence in plants of systemic antibodies in the sense used in animal immunology (agglutinins, precipitins, lysins) and other explanations of the above recorded effects must be sought.

Some of the most positive indications of the occurrence of acquired immunity at a distance from the infecting organism have been obtained from observations and experiments in the mutualistic association (symbiosis) of fungi and bacteria with roots, encountered in orchid mycorrhiza and in the root nodules of the *Leguminosae*. The most recent study of the defensive mechanism in orchid mycorrhiza gives no evidence for or against the existence of antibodies. While the results may well be interpreted as due to the presence of a thermolabile toxin, it is considered that this substance need not be of the nature of an antibody. Similarly the repression of parasitic activity of the nodule bacteria, which has been

attributed by some investigators to antibody formation, can be neutralised, at least in lucerne [32a], by cutting off the carbohydrate supply, as by growing the plants in the dark, when the organism becomes an active parasite. It is easier to suppose that the organism remains non-parasitic so long as it receives carbohydrate nutrition from the above-ground parts of the host, but, when this is cut off, seeks it by dissolving the cell walls, than that its activity is due to antibody formation.

At the same time, none of these considerations tells against the possibility of an intracellular localised acquired immunity. Indeed there seems to be good reason for supposing that substances having the characters of antibodies can be formed in cells in response to their invasion by a parasite. What the substances are that check the development of certain parasites in the tissues of resistant hosts is usually unknown, but there is evidence that they are, sometimes at least, formed in response to invasion even when, as in ' hypersensitive ' plants the capacity to form them is inheritable. There is evidence also that, in some cases, the repressive action is delayed, so that the parasite can make some progress in the tissues of resistant hosts before it is checked. Antibody formation is not the only hypothesis that might explain such cases, but it is a possible one. In view of the differences in organisation, it would not be expected that such an intracellular formation of antibodies would be followed by their transmission to other parts of a plant, as occurs so often in animals, and the failure to demonstrate induced immunity outside the vicinity of infection does not weaken the possibility of an intracellular reaction.

It is a remarkable fact that the only cases in which it has been fully demonstrated that infection can lead in plants, as in animals, to immunity from subsequent reinfection occur in the most completely systemic plant diseases that are known, those due to viruses. It is now well established that in several virus diseases, such as that caused by the ' X ' virus in potatoes, a previous ' vaccination ' will confer immunity against a later attack, even when the second infection is with a much more virulent strain of the virus. Similarly, in the ' curly top ' virus of tomatoes apparent recovery sometimes takes place and new shoots are produced that appear to be free from the disease ; clones from these are resistant to further infection although they have been proved to carry the virus and to be able to transmit it to healthy plants. Many of the diseases of this type appear to infect every part, and probably every living cell, of the plant, and it seems to be perfectly feasible to explain the results in terms of an acquired intracellular immunity ; this is the more so, in that it has been found, in cases where the virus is not completely systemic, that the immunity produced by the first infection is limited to the parts of the plant actually reached by the virus.

Analogies between animal and plant diseases must be limited within comparatively narrow bounds and sought mainly in intracellular pathology. Induced immunity in plants, in the best authenticated cases as in the virus diseases, is an intracellular phenomenon, perhaps of the same order as the natural resistance shown by the cells of resistant hosts when invaded by a rust fungus. There is no *a priori* reason why the reaction should be confined to invaded cells, and the movement of viruses, hormones, and the like, shows that complex substances can pass

from cell to cell, but there is little good evidence that the antigenic action, if one exists, spreads beyond a very limited range. On present knowledge it seems to be very doubtful whether any close analogy can be established between the means employed by animals and plants in their fight against parasitic diseases.

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Chapter V

THE INFLUENCE OF ENVIRONMENTAL AND NUTRITIONAL CONDITIONS ON PLANT DISEASES

Meteorological Factors

The paramount influence of climate on the prevalence of parasitic diseases of crops has been recognised since the earliest times. The obvious relation between weather and such diseases as potato blight or vine mildew could not fail to be noticed alike by growers and plant pathologists. But the analysis of this influence is not easy, for the interplay of different factors leading to a heavy outbreak of disease, or enabling a parasite to gain entrance into, and to spread within its host, is such that exact methods for determining the effect of each constituent in the presence of the others are required ; and they are seldom available.

Meteorological factors may act on the parasite or on the host or on the two when brought into association (in scientific jargon, the ' host-parasite complex '). Temperature, humidity, light, and nutritional conditions are the chief of these factors affecting plants. Their effects on fungal cultures in the laboratory are a matter of everyday observation (Figs. 126 to 130). Temperature is the most pronounced in its action, and the heat or cold of the air or of the soil in which a crop and its parasites are growing may be decisive in determining the presence and the amount of a disease. Many crop parasites are limited in their distribution ; they fail to become established when introduced into areas where either the maximum or minimum temperatures reached, or the seasonal fluctuations, are such that they cannot survive. This is sufficient to explain why smut has not obtained a permanent footing in the maize crop of southern England and the Channel Islands or in the Egyptian crop of onions, nor potato blight and wheat bunt in the hot plains of India. Even heavy artificial inoculation has failed to induce wheat bunt at Pusa, while onion smut was reported in 1926 to be unknown in the Gulf States though constantly distributed to them on setts from the northern United States.

Humidity appears to act less frequently as a limiting factor in the distribution of parasitic fungi, though, combined with temperature, it is responsible for the exclusion of citrus scab (*Elsinoe fawcetti*) from the Mediterranean region and California [15] ; however, moisture is all important in the annual fluctuations in the numbers of individual parasites and their spores. Light, the third of the meteorological factors affecting plants, is of much less significance in controlling the actual distribution of fungi than either heat or moisture, but it is sometimes concerned in the development of sporing stages and hence can be a factor in dissemination, while shade and the length of day are occasionally important in the incidence of disease.

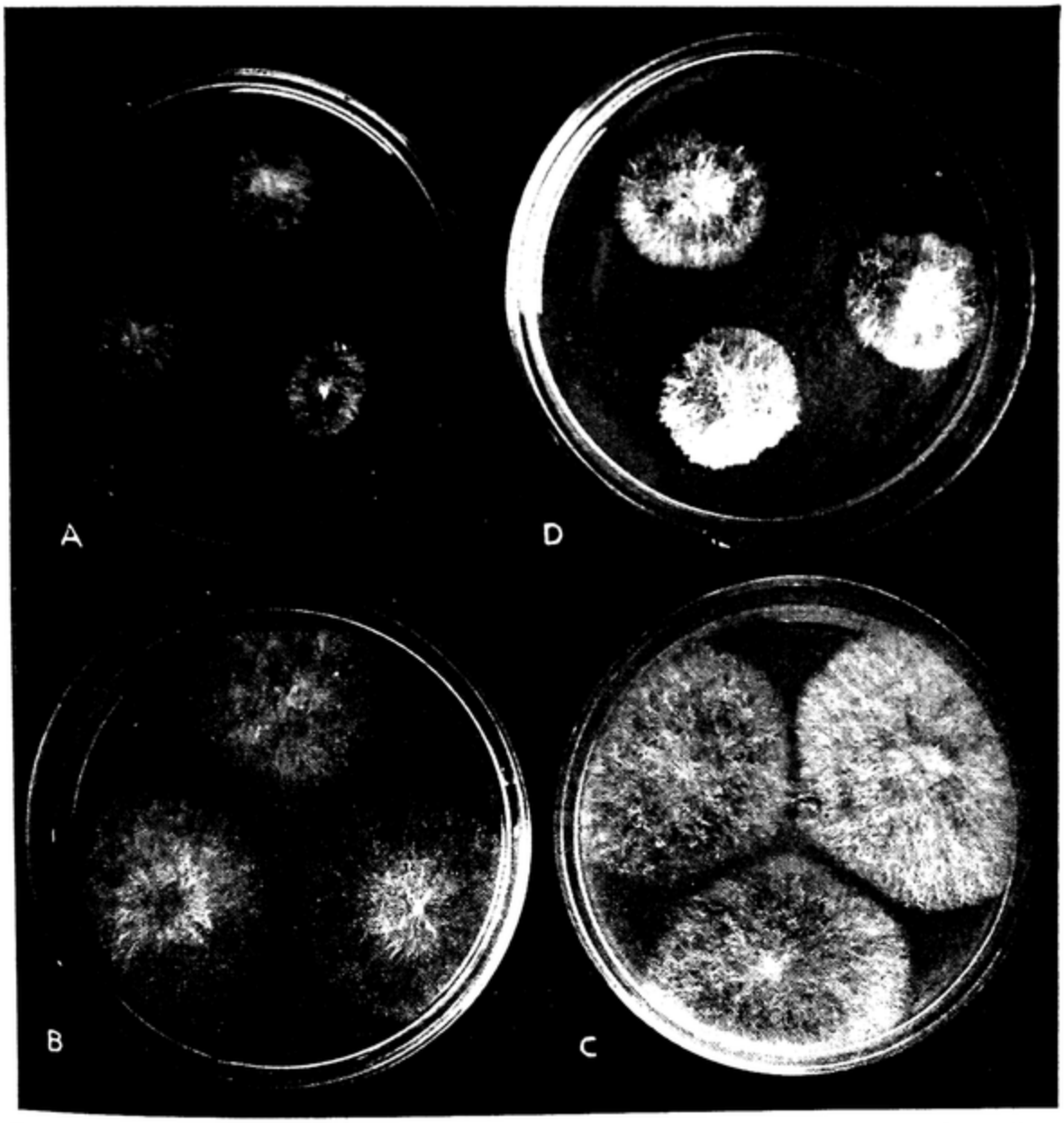


FIG. 126.—Influence of temperature on fungal growth. *Fusarium dianthi* on Brown's medium. Incubation, 3 days. *A*, relative size of colonies at 5° C.; *B*, at 20° C.; *C*, at 25° C.; *D*, at 30° C.

Variations in one environment factor may alter the action of the others. Thus, it has been found that no single temperature could be considered to be the optimum for growth of any of eight species of wood-rotting Basidiomycetes when tested on different good nutrient solutions [56]. The same seems to hold for strains of *Actinomyces scabies* from potatoes [59]. Similarly, the range of humidity over which a fungus will grow is greater the more nearly the fungus is kept to its optimum temperature for growth and this also extends its *p*H range, as in *Fomes annosus* [49], while its temperature range is greatest when the humidity is near its most favourable point [52]. Indeed, it seems that when any one of the factors that influence growth rate is changed so as to lessen the rate, the resistance of the fungus to the other factors is also decreased.

It might be expected that comparison between the plant diseases of areas with

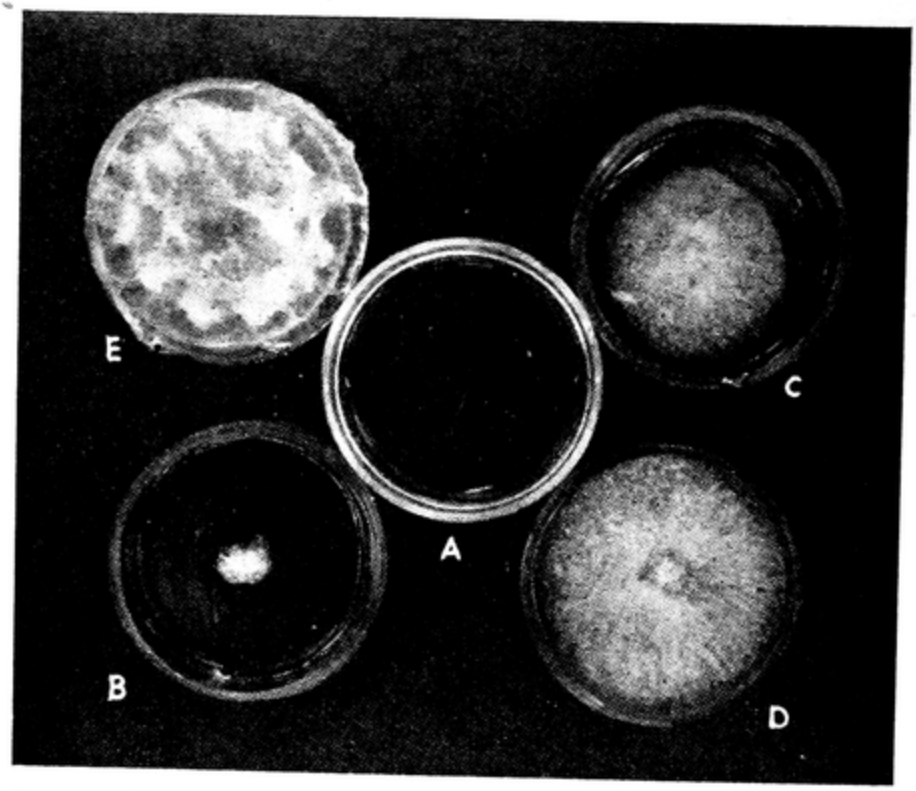


FIG. 127.—Influence of relative humidity on fungal growth. Cultures grown in open Petri dishes (enclosed in large, sealed crystallising dishes containing variable concentrations of sulphuric acid). *A*, at centre, over the pure acid, no growth, the medium drying quickly. *B*, over the 50 per cent. acid. *C*, over 30 per cent. acid. *D*, over 10 per cent. acid. *E*, over water, the mycelium (*Fusarium dianthi*) growing over the edge of the dish. Three days' incubation at 20° C.

differing climates would be difficult because the host plants would not be the same. However true this may be for the wild plants it is much less true of the major cultivated crops. The latter have been subjected to long periods of artificial selection in such fashion as greatly to extend their original geographical range. Wheat and potatoes, for instance, are grown over very considerable ranges of climate, their present adaptation to which sometimes pays tribute to the remarkable skill of the plant breeder. Their parasites, on the other hand, have not been subjected to the same human influence, and they have had to follow the hosts as best they could. In the larger continental areas, such as the United States and India, the wheat, sorghum, maize and potato areas are much wider than the areas in which certain of their parasites are found. Still there are a good many parasites whose range is greater than that of certain of their hosts (the ' damping off ' fungi, *Pythium* and *Rhizoctonia*, for instance). Furthermore, even

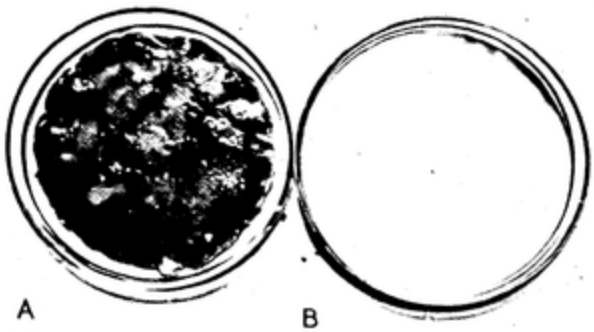


FIG. 128.—Composition of the culture medium. On Richard's solution (no agar), six days' incubation at 20° C. *A*, the colony filling the entire dish on the complete medium, the carbohydrate being sucrose. *B*, without the carbohydrate. (*F. dianthi*)

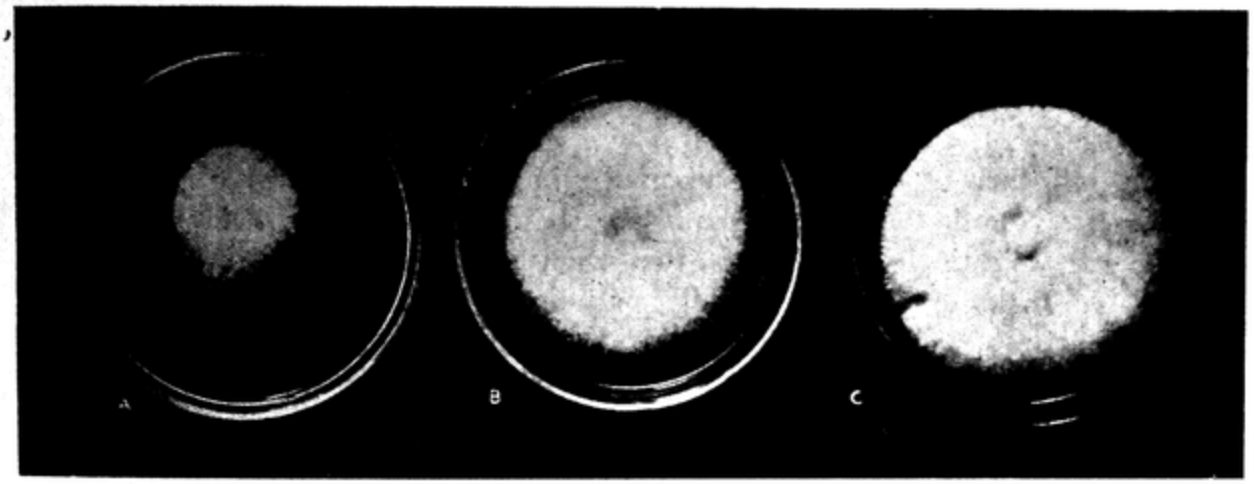


FIG. 129.—Effect of changing the source of nitrogen in a medium containing maltose, magnesium sulphate, potassium dihydrogen phosphate. *A*, plus ammonium chloride. *B*, potassium nitrate. *C*, asparagin. (*F. dianthi*; at 20° C.; six-day incubation)

when host and parasite are both present it is by no means certain that disease will occur, for the host may prove to be resistant under the local climatic conditions. Tomatoes inoculated with the wilt-producing *Fusarium bulbigenum* var. *lycopersici* showed no symptoms of wilting when the air temperature was kept down to 17° C., though the fungus will grow at considerably lower temperatures than this and though the roots were penetrated by it; when the soil was warmed to the optimum for the disease (about 27° C.) root infection was heavy but visible wilting still did not occur provided the air was cool [8]. In England this fungus causes wilt only during periods of high temperature (above about 80° F.), the usual cause of tomato wilt being *Verticillium albo-atrum* and *V. dahliae* (Part II, p. 669) and the disease developing most rapidly about 22° C. (71° F.) when due to these [6].

In Chapter III the influence of various environmental factors, including temperature, moisture, and light, on the germination of fungal spores has been mentioned and their significance in the pre-penetration stage of a parasitic attack stressed. The effect of these factors on the actual progress of a disease remains to be briefly considered. As already mentioned, the conditions prevailing within the crop (the 'microclimate') may be much more important than those shown by the standard meteorological equip-

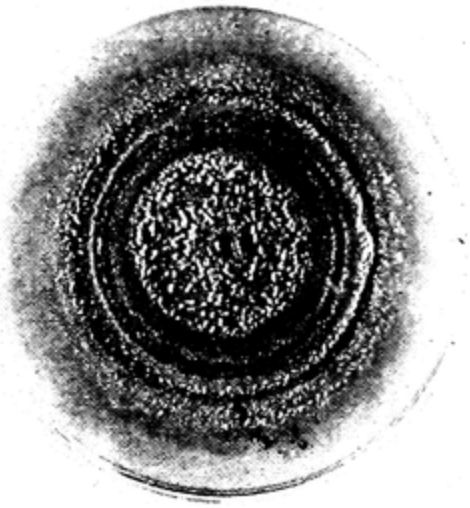


FIG. 130.—Staling of the medium and incidence of sporulation. A culture of *Fusarium* sp. showing concentric rings of spore formation. At the centre, growth at the start is fairly even, with even distribution of spores. Later, the growth of the fungus outwards into the medium sets up zones of sterile mycelium with alternating sporulating zones, as intermittent staling of the medium in the zones occurs. Other fungi produce asimilar concentric effect in cultures when exposed to alternation of light and dark

ment in their influence on spore production and germination. This is equally true at times for the later stages, when the parasite has become established in the host.

Temperature

The influence of temperature upon the inception and course of a parasitic disease, that is upon the host and parasite in their communal life, is not necessarily the same as that upon either of the two when living alone. Conditions that are favourable to the one may be equally, or more, or less favourable to the other, while sometimes both are found to withstand conditions which prevent the development of disease. The often delicate balance that is established between host and parasite may be upset by temperature variations. A cold snap in summer may be followed by an outbreak of rust in wheat, or onion mildew may come out to the surface of the leaves to form an efflorescence of fructifications in a systemically infected variety when warm and moist (' muggy ') weather sets in. In several of the seed-borne diseases or those caused by infections from the soil, the climatic conditions at the time of sowing may determine whether the crop will be injured or not; the correlation between low soil temperature during germination and the incidence of bunt in wheat has been frequently observed. Some examples of the influence of temperature on particular diseases will, perhaps, best illustrate the kind of effect that may be produced.

The disease known as ' sore shin ' of cotton in Egypt is caused by the almost ubiquitous soil fungus, *Rhizoctonia solani*. The damage done by this disease, which often necessitates re-sowing, is usually restricted to the seedling stage. Once cork forms, damage ceases. Both parasite and host plant have similar curves of accelerated growth with rising temperature, ceasing about 37° C. In the fungus the cessation of growth appears to be due to auto-intoxication or ' staling ' from the products of its own metabolism. At lower temperatures these are formed too slowly to inhibit growth, but at about 33° to 37° they form too rapidly to be outgrown. At 33° the cotton is near its optimum temperature for active development, and can form defensive cork rapidly. The early sown Egyptian crop at the end of February or March finds temperatures of, say, 20° to 25° which enable the fungus to penetrate the root and pass from cell to cell, destroying the tissues before its toxic products have time to accumulate. At about 33°, in the later sowings in April or May, the parasite is checked after producing only a small scar, occluded by wound cork. At 37° not even a scar is produced. A cold spell of only a few days in May, however, will cause the death of many seedlings. In the greater heat of the Sudan at cotton sowing time, sore shin is negligible. Thus, in this disease temperature in the early seedling stage influences the attack by its action on both host and parasite, though mostly on the latter [3].

Another very striking case of a similar influence of temperature is found in the seedling blight of maize and wheat caused by *Gibberella zeae* (Part II, p. 401), in the United States. The corn (maize) belt extends much further to the south than wheat can grow successfully, but not so far north as wheat. It was noticed that the southern part of the maize belt and the northern wheat areas escaped this disease. It was found by experiment that the favourable soil temperature for infection of maize was between 8° and 20° C., infection being most severe between

12° and 16°. With wheat, on the other hand, infection occurred between 12° and 28°, but was most severe about 24°. The fungus can grow normally over a wide temperature range from 3° to 32°, though its optimum for growth is about 24° to 28°. Spring wheat is favoured by a temperature range of about 16° to 20°, winter wheat about 12° to 16°, and maize about 24° to 28°. Hence seedling blight is found in both crops when they are grown outside their optimum temperature range, wheat too far to the south, maize to the north. Histological and biochemical studies of the roots have shown that, at the low temperature for maize and the high temperature for wheat, the cell walls remain for a considerable time in the primary pectic condition, easily penetrated by the hyphae. Under the converse conditions the cell walls are rapidly thickened by deposits of cellulose on them, so that they are less readily penetrated by the fungus. The metabolic processes responsible for this variation in development have been referred to above (Pathogenesis II, p. 139). In this example the influence of temperature on the disease acts mainly through the host plant.

In a crop such as wheat, cultivated through long ages in many climates, it is possible to group the races into ecotypes adapted to extremes in the climatic range of cultivation. Tests in a collection of such ecotypes near Leningrad have recently shown that those from northern regions wintered well and resisted the attacks of the 'snow mould' fungus, *Fusarium nivale* (*Calonectria graminicola*) (Part II, p. 479), while those from the south were badly attacked [31].

Unfortunately it is not possible to generalise from these examples. Other root diseases of wheat are favoured by temperatures near the optimum for its root development and are absent when the soil temperature is higher, unlike seedling blight. 'Club-root' progresses most rapidly and severely in cabbage roots in soil at about 20° C., the optimum for growth of the roots. Furthermore, in a seedling disease of swede turnips in which a species of *Pythium* was concerned, the pre-emergence killing by this fungus was greatest at low soil temperatures (about 6° C.), whereas the damping-off of the seedlings after emergence was greatest at 23° when pre-emergence deaths were at a minimum [21]. The delayed emergence at the lower temperature is a likely reason for the enhanced mortality at this stage.

A simple temperature effect appears to be the heavy mortality of spruce seedlings in nurseries in some parts of Central Europe in June from attacks by both *Pythium de baryanum* and *Fusarium bulbigenum*. The latter does little harm below about 30° C., whereas the *Pythium* is active above about 15°. At 30° both are destructive [42].

A temperature effect of great importance in some parts of the world is the prolongation of the incubation period of certain diseases, especially the rusts, by low temperature. The over-wintering of the cereal rusts *Puccinia glumarum*, *P. triticina*, and *P. dispersa* (but not *P. graminis*) is attributed to long incubation periods in the mycelial stage within the autumn-sown plants, especially when these become covered with snow, as regularly occurs in Russia. Even in *P. graminis*, which is seldom found to survive in this manner, experiments in the United States showed that its first sori only appeared after about 70 days at a temperature of 0° to 1° C., while they are occasionally seen in midwinter in France.

There is some evidence that high temperature may have a similar effect, for the incubation period of the uredo stage of *P. graminis* may be prolonged in the hot weather in India from the usual 10 or 15 to 35 days.

In recent years, and particularly in 1935, spring frosts caused serious reductions in the fruit crop, which led the Ministry of Agriculture to institute an enquiry into the effect of meteorological factors on the distribution of frost damage (10 a).

It was found that spring frosts were of two main kinds, radiation-frosts and wind-frosts. These occur in the proportion of about two radiation-frosts to one of wind-frosts. A radiation frost has the following characteristics. It occurs only on a calm clear night, when there is insufficient wind to stir up the air layers, and no cloud to ' blanket ' the earth. The heat in the soil, plants, and air passes by radiation into the sky. Relatively cold air that forms on a hillside is denser than warmer air and moves downhill at about 3 m.p.h., forming a katabatic wind. On and near hill-tops a relatively warm wind is found, which is the day wind persisting at night. Hence a radiation-frost causes cold valley-air and warm hill-air. Observations made on a hill which rose 600 feet above a valley showed that temperature differences of 12° F. were common in radiation-frosts. In level fields on calm clear nights air temperature at 3 feet above the ground varies with the vegetation. Long dense grass gives rise to the coldest air and bare soil to warmer air. Temperature differences of 6° F. may be caused in this way. Damage caused by a radiation-frost is confined chiefly to valleys and to low fruit trees growing among long dense grass. Measures which are most likely to prove beneficial under these conditions are the use of orchard heaters, the shortening of grass, and covering plants with light screens of hessian.

In a wind-frost the air has been cooled in polar regions to a temperature below freezing point, and blows across country, usually at a speed of more than 3 m.p.h. There are no katabatic winds, and the hill air is *colder* than the valley air at the rate of 1° F., for every 300 feet of ascent. Vegetation has no effect on these nights. A wind-frost damages crops on hills more than those in valleys. Fortunately, in the spring the temperature in a wind-frost is not so low as that in the more severe radiation-frost. Orchard heaters are not effective in a wind-frost. Strawberries may be covered with hessian (10 a).

Moisture

While temperature is the main controlling factor in the geographical distribution of disease, moisture is probably more important in the local fluctuations of diseases within a particular climate, such as is found in the greater part of the British Isles. Not only the total rainfall of a locality or season must be taken into account but also the prevalence of fog and dew. The infective process requires a degree of relative humidity that is more or less high according to the fungus concerned ; with the cereal rusts it has to be usually above 92 or 93 per cent., with bean rust (*Uromyces appendiculatus*) at or above 95 per cent., but with *Erysiphe graminis* it may be below 75 per cent. The influence of moisture and temperature on potato blight will be referred to later (p. 168).

The wilt of tomatoes caused by *Fusarium bulbigenum* var. *lycopersici* has been found to be worst in a moderately damp soil, the disease being checked when the

soil is either very wet or very dry [9]. In the very wet soils, at least, the inhibiting action appears to be associated with the metabolism of the host, and shows a correlation with the absence of nitrate nitrogen; this absence does not inhibit infection but increases the plant's resistance to the toxic effects of the fungus. Infection of wheat by *Fusarium culmorum* (Part II, p. 389), is stated to be much more prevalent in soils with a moisture content equal to 30 per cent. of saturation than in those with 50 per cent. or more; in this case the factors involved are thought to be increased aeration of the parasite (a weak one under the conditions of the experiments), and possibly injury to the host, at the lower humidity [46]. The progress of *Urocystis occulta* in winter rye is definitely expedited in dry soil (25 per cent. of the water-holding capacity) [30].

Several diseases are known to be influenced by the water content of the host. This may affect the penetration of the parasite through the surface or its subsequent growth in the tissues of the host. The turgor of the cells has a bearing in some of these cases. Flaccidity of the leaves hinders infection by the tomato leaf mould, *Cladosporium fulvum*. A lack of turgor may inhibit attack by *Botrytis cinerea* on potato tuber tissues. It also reduces the dissolving action of the pectinase produced by this fungus as well as by a cruciferous strain of *Rhizoctonia solani*, when extracted and used alone. The flooding of the intercellular spaces with water, either by injection or naturally (as by driving storms of rain or when high air humidity, perhaps accompanied by a fall in temperature, checks transpiration at a time when the vessels are water-laden), increases the growth rate of the pathogen and the susceptibility of the host in the bacterial diseases of tobacco due to *Pseudomonas tabaci* and *Ps. angulata* and also in firelight (*Bact. amylovorum*) of pear and apple [44]. This tendency to flooding of the intercellular spaces is increased by low potassium and high nitrogen fertilisation. By itself, even without the presence of pathogenic organisms, it can cause injury if prolonged, so that its effect on the diseases should not be over-rated [53]. Very wet potato tubers kept at a moderate temperature do not resist *Botrytis cinerea* well, owing to delayed suberisation [40]. While, as just mentioned, the enzyme from a cruciferous strain of *Rhizoctonia solani* was most active on turgid swede tissue, the injection of water into the tissues of the swedes made them much less susceptible to attack by the fungus itself. The reason for this difference between the action of the enzyme and of the fungus, so unlike what occurs in *Botrytis*, is unknown [49]. On the other hand, the moisture content of the potato tuber seems to be without effect on the aggressiveness of *Alternaria solani*. The rate of growth of the potato blight fungus, *Phytophthora infestans*, within the tissues of infected leaves is directly proportional to the water content of the leaves; its spores also are produced more freely the higher the moisture in the tissues, though, as mentioned in an earlier chapter, their immediate germination is best secured if they are exposed to a short preliminary period of drying.

It is not easy to find explanations of the effect of water content of the host tissues on the course of a disease that will cover the observed facts. There is evidence that certain parasites of the wood of trees are dependent for their activity on aeration, which in turn depends on the water content of the vessels. Aeration may also be necessary for some of the intercellular parasites. Speed in host reaction to attack, as by lignification or suberisation of the membranes, may also

be a factor of importance in some cases. But the factors concerned in other cases are more complex and are not clearly understood.

The factors conducive to attack by the powdery mildews, such as *Erysiphe graminis* have been closely studied in Italy (41). In particular the receptivity of the host plant is correlated with the turgor of the tissues. Plants such as cereals, oak, rose, and others commonly affected by these fungi are relatively resistant to attack so long as their green parts are fully turgid, but when they lose turgidity, as from hot or dry air or dry soil, they are readily infected. The *Erysiphaceae* do not differ materially from most other fungi in being favoured by humidity, though their moisture requirements for spore germination and infection are well below those of potato blight and the rusts. But even in hot dry summers, such as that of 1921 in southern England, when mildew was unusually prevalent on many plants, there are sufficient cool nights and dew deposits to secure a copious supply of conidia, and it is to the succession of hot dry days with dry soil and cool nights with increased humidity that outbreaks of this class of diseases are attributed. With a limited root system such as may result from heavy manuring or high soil moisture during the plant's previous growth, a mere increase in air temperature or dryness may induce flaccidity of the leaves even when soil moisture is ample. There are difficulties, however, in explaining fully the susceptibility of plants to the powdery mildew on a basis of turgor alone. Artificial infections at Cambridge have shown that parasitism can be established in forty-eight hours in a saturated atmosphere, and conidial chains appear in ninety-six hours (10). In India, too, observations have been recorded that the mildew on tobacco is frequently restricted to the shaded parts of fields, as under trees, though these should be less likely to be affected by conditions leading to loss of turgidity in the leaves. Radiation may be a dominant factor in this case.

Moisture may be a factor of importance in the infection of twigs by the fire-blight organism, *Bact. amylovorum*. The continuous cork barrier formed in the cortex in advance of the lesion develops rapidly when moisture is low, as it does in resistant varieties of the host, but is delayed when the air is humid or the variety susceptible (44).

Radiation

There is less evidence of the effect of light and radiation on plant diseases than of that of either heat or moisture. The intensity and actinic power of sunlight has been invoked to explain cases of greater prevalence of apple mildew at the top of a sloping orchard than at the bottom where the sun only reached late in the afternoon, and of oak mildew in exposed situations. The conidia of the latter fungus are produced much more vigorously on shoots exposed to the light than on those in the shade (36). Continuous artificial illumination throughout the whole twenty-four hours will prevent *Erysiphe graminis* from becoming established on wheat, though if the exposure begins after infection is well developed continuous illumination is rather favourable to the fungus (43). In the other direction, it has been stated that extremely virulent attacks of *Thielaviopsis basicola* on beans (*Phaseolus vulgaris*) can be provoked by drastic shading, while shade makes mature plants of Hope wheat as susceptible to race '21' of *Puccinia graminis*

tritici as seedlings in the upper Mississippi valley, where the intense sunlight ordinarily confers resistance once the seedling stage of this wheat is past [25].

In an ecological study of the parasitic fungi of a valley in the Pyrenees up to 2,000 metres, radiation was found to be the most important factor influencing the vertical distribution of the parasites [13]. Here again the oak mildew was most prevalent towards the top, as were some of the rusts, while other rusts flourished best near the bottom of the valley. Experiments in the United States indicate that certain rusts are not affected, while others are diminished in their prevalence and intensity, by darkness during and subsequent to the time of infection. In 'artificial sunlight' a six-hour day increased the length of the incubation period of *Puccinia glumarum* on barley, as compared with a twelve-hour day, by nine days. High light intensity (960 foot-candles) nearly halved the incubation period found in ordinary daylight with this rust, but prolonging the daylight artificially beyond twelve hours caused a normally fully susceptible variety to become highly resistant [5]. On the other hand, artificially lengthened daylight increased the percentage infection of winter rye with the smut *Urocystis occulta* [30].

That light, through its action on the carbohydrate nutrition (photosynthesis) of the plant, can have a marked effect on organisms living within the tissues is shown in striking fashion by its action on the symbiotic bacteria of the nodules of the *Leguminosae*. As mentioned in Chapter IV, when the host plants are kept in darkness the bacteria become actively parasitic in the roots (p. 156).

Wind

Wind is a factor of primary importance in the dissemination of many diseases and its significance in the annual recurrence of wheat rust in certain areas in North America and India, and in the dispersal of the winged green flies that carry certain virus diseases to the potato crops in Great Britain, is discussed elsewhere.

It has already been stressed that neither the parasite nor its host is ordinarily a constant entity, but that both usually consist of populations which vary amongst themselves to a greater or lesser degree in different areas. This affects their reaction to diseases. It has been found, for instance, that physiologic races of *Puccinia glumarum* showing similar virulence and rapidity of spread under one set of conditions may have quite different epidemiological significance under others, owing to variations in the rate of germination of their uredospores and in their ability to withstand high temperatures [51].

It is obvious that meteorological factors can have very different effects on primary and secondary infection in a crop. In the black arm disease of cotton caused by *Xanthomonas malvacearum*, the development of primary infection in cotton seedlings, which in nature usually comes from infected seed or contaminated cotton debris in the soil, is chiefly governed by the mean soil temperature at the time of sowing and for the first few days of germination. The lowering of temperature as a result of rain at this period may have disastrous effects, but subsequent variations in soil temperature are of little consequence. Primary infection is also higher at soil moistures approaching saturation for the particular soil type; it varies at a given soil temperature and soil moisture with the type of soil. Secondary infection, which in nature is frequently the result of driving rain storms,

depends on the mean air temperature prevailing during the incubation period of the disease. The actual air temperature at the time of inoculation was found in artificial inoculations to be unimportant, while air humidity was important only during a short period (less than forty-eight hours) following inoculation, in that it controlled the time during which the infective droplets persisted on the leaves; variations in atmospheric humidity had little direct effect on the disease after its establishment. Though light is not a factor likely to be of significance under the natural conditions of the areas where the disease is serious, experiments have shown that plants kept in total darkness are relatively resistant to black arm infection [50].

The bad epidemic of black rust of wheat in the main wheat-growing areas of the United States in 1935 was due to a combination of circumstances. The crop in Texas matured late and under conditions of favourable temperature for rust and of high humidity [2]. Southerly winds carried the inoculum, which was produced over a long period, to the north. Kansas had similar conditions in May and June and contributed to the dissemination of the rust northwards [26]. The rust over-winters successfully on wheat in Texas and the importance of this focus of infection far outside the State boundaries has been discussed in Chapter I.

A case like this last is straightforward enough, for it depends on the facility with which the parasite can reach the host in sufficient numbers and under sufficiently favourable conditions to provoke an epidemic. In other cases it is the host plant on which seasonal variations of weather have their chief effect, as in the spring infections of apple trees with scab; tests at East Malling showed that the stage of development of the trees varied much more in different seasons than the date of discharge of the ascospores of *Venturia inaequalis*, and if spraying is not adjusted to meet these variations (the second spraying should be finished before the ' pink bud ' stage) the disease may become established.

Most of the cases that have been cited above are due to temperature and moisture conditions which directly affect the parasite or its host. Light and shade effects, however, seem to act on the disease as a whole (the ' host-parasite complex ') and is it much more difficult in such cases to determine the real causes of the result.

Forecasting Outbreaks of Disease

Forecasting services have been organised in some countries (France, Germany, Italy) in order to notify vine-growers of an impending outbreak of vine mildew (*Plasmopara viticola*) (Part II, p. 833) so that spraying may be undertaken in advance of the attack [7]. They are based on knowledge of the meteorological conditions influencing the germination of the spores of the parasite, infection from these, incubation in the tissues, and subsequent sporulation. The similar services for spraying against apple scab in the United States and Canada are based mainly on the occurrence of conditions favourable for the discharge of the over-wintered ascospores of the fungus, a source of primary infection of major importance in some, but not in all, parts of England.

Attempts have been made with some success in Holland [24, 55] and England [4, 4b, 58] to forecast outbreaks of potato blight from its known weather requirements. Humidity is the chief of these so far as local fluctuations are concerned,

but temperature, which is the main factor in the geographical distribution of the disease, also plays a part in its prevalence within endemic areas. Work (mainly in the United States) [32] has shown that the zoospores of the fungus are liberated most rapidly and copiously at the rather low temperature for summer time of 10° to 13° C., and they swim about in water longer at low than at higher temperatures (24 hours at 1° to 2° C. but only 15 minutes at 24° and rapid killing above 28°); at high temperatures (up to nearly 30°) the sporangia mostly give rise to germ-tubes instead of zoospores. At still higher temperatures, but yet below those that injure the tubers, the mycelium of the fungus is killed; it cannot be maintained in culture above 32°, though within the tissues it can survive short exposures up to 40°. Infection from the germ-tubes produced by the zoospores can establish the parasitic mycelium within the host in $2\frac{1}{2}$ hours from germination at temperatures from 10° to 25°, but the growth of the parasitic mycelium is most rapid and the incubation period (appearance of visible lesions) shortest at 20° to 23°. Records obtained by the United States Weather Bureau indicate that blight makes its most rapid development when the daily average temperature is about 72° F. (22·2° C.). A relative air humidity of 95 per cent. or more for a duration of 8 hours is necessary for free production of viable sporangia, and another 11 or 12 hours are required, with free water on the leaves and a temperature neither too warm nor too cold, for the germination of the sporangia, the dispersal of the zoospores by swimming, their germination, and the infection of the leaf [11]. To secure successful penetration of the leaves they must be kept damp for $1\frac{1}{2}$ hours at 20° to 25°, 2 hours at 15°, and $2\frac{1}{2}$ to 3 hours at 10°.

In the south-west of England, where the attempt to forecast outbreaks of blight has been in progress for a number of years, a day is counted as favourable for the spread of the disease (which is present to some extent in the fields every year) when there is: (1) dew either the night before or in the morning, (2) minimum temperature not below 50° F., (3) sunshine of less than 5 hours, (4) rainfall of at least 0·01 inch, (5) relative humidity at 3 P.M. (when experience has shown that a high humidity generally indicates high humidity all day) not less than 75 per cent. If warm dry weather succeeds such a day no outbreak will follow, but if these conditions are prolonged or recur every few days a generalised attack may be expected. So important is the humidity factor that in the counties of Devon and Cornwall the 3 P.M. humidity alone from late June to September may serve as a danger signal of probable outbreaks [4]. In Holland the pre-requisites are held to be (1) night temperature below the dew point for at least 4 hours, (2) minimum temperature about 10° C. or above, (3) minimum degree of cloudiness next day 0·8 or above, (4) rainfall of at least 0·1 mm. during the next 24 hours [54]. High temperatures such as govern the geographical distribution of the disease in the United States and India are not sufficiently prolonged in the British Isles to prevent its annual recurrence.

Soil and Nutritional Factors

Next to the meteorological conditions of the environment which influence plant diseases, those of the soil, its composition, texture, water content and so on,

are of the greatest importance. Unlike the weather, which cannot be changed and only rarely can be 'dodged' by altering sowing dates, soil conditions can be modified by cultivation and manuring. Much work has been done on the influence of fertilisers on plant diseases and this also requires consideration, though the analysis of the factors involved is not less difficult than that of climatic factors, and clear-cut results are hard to obtain.

The Influence of Soil Conditions on Disease

Soil conditions may affect the parasitic fungi that attack mainly the roots of plants [17]. These organisms may be strict aerobes and unable to survive in closely textured or water-logged soils, or the soil reaction may be too acid or too alkaline for their normal development. Soil conditions also affect to a marked degree the host plants, whether by modifications of structure or of physiological function, both of which may influence their resistance to disease. This appears to be very noticeable in the irrigated orchards and gardens of the Nile Valley, where a high water table increases the injury caused to peaches, plums, and apricots by rust (*Puccinia pruni-spinosae*) (Part II, p. 760) and shot hole (*Clasterosporium carpophilum*) (Part II, p. 776), by *Sphaerotheca pannosa* (Part II, p. 855) to peaches, and by *Erysiphe cichoracearum* (Part II, p. 644) and *Colletotrichum lagenarium* (Part II, p. 647) to vegetable marrows and watermelons [16].

Of recent years, numerous surveys have been made of the fungi living in the soil. Taken as a whole these show that a distinct soil fungus flora can be recognised. Most of the organisms thus repeatedly found are, however, either saprophytes or weak or unspecialised parasites, capable of attacking many hosts under suitable conditions but, when the land is fallow, capable of supporting a prolonged non-parasitic existence. Several of the damping-off fungi such as *Rhizoctonia* and *Pythium* belong to this class. In addition, however, many soils contain more specialised and more virulent parasites which appear able to support life in the soil only for a certain length of time in the absence of their proper hosts. When such fungi produce durable spores or sclerotia they may remain dormant in a soil for years, at least ten in the fungus that causes wart disease of potatoes. In some parasitic fungi, especially those in tree roots, survival after the death of the host may be limited only by the rotting of the roots, which may take years. Closely allied fungi, such as different species of the genus *Fusarium*, may belong to these two main groups : some may be true soil inhabitants normally found in the soil of a particular region, others are soil invaders and disappear more or less rapidly when the plant whose roots they attack is killed. This failure to survive is probably due to inability to hold their own against the competition for food, etc., of the other soil organisms, as well as susceptibility to injury from metabolic products excreted by the latter. The wilt-inducing species of *Fusarium* mostly belong to this second group and hence they can ordinarily be avoided by rotation with non-susceptible crop plants.

Most of the work on the influence of soil conditions on parasitic fungi relates to the group of soil invaders. Many of these appear to be favoured by good aeration and develop best in open or light soils. This seems to be true of the *Fusarium* parasites, smuts, foot-rotting fungi of cereals such as that causing take-

all, the collar-rotting Sclerotinias, and common and powdery scab of potato tubers. In some of these it is difficult to secure infection in heavy clays. Reaction is also of importance in a number of diseases. Its influence seems to be quite fortuitous, that is to say, it is impossible to anticipate on present information and without testing whether a particular parasite or type of disease will be favoured by acidity or alkalinity. Some of the species of *Fusarium* that cause vascular wilts and seedling blight and root rot of cereals do most damage in an acid soil (*F. bulbigenum* var. *lycopersici*, *F. vasinfectum*, *F. culmorum*, etc.), others in an alkaline one (*F. orthoceras* var. *pisi*). Potatoes growing in acid soils are most liable to injury from powdery scab, those in alkaline from common scab. Even the two common vascular wilt diseases of the tomato differ in this respect, that caused by *Verticillium albo-atrum* being favoured by an alkaline soil, while that caused by *Fusarium bulbigenum* var. *lycopersici* is worst in acid soils. Other important diseases favoured by acid soils are club root of crucifers, wart disease of potatoes, and several tree root rots, including that caused by *Armillaria mellea*. How far the prevalence of certain types of root rots in impermeable soils may be due to acidity and how far to accumulations of CO_2 or toxic substances in these soils is not known. Among the fungi favoured by an alkaline soil reaction *Ophiobolus graminis* is one of the most important and most fully studied. The relation in this case affects only the parasitic phase of take-all. At Woburn little or no disease occurs at a pH_5 or less [19] and in South Australia it is never severe on neutral or acid soil but often causes epidemics in sandy soils at $pH_{8\text{-}9}$. It has been suggested that growth of the fungus along the roots is checked in acid soils by the accumulation of respiratory carbon dioxide in the neighbourhood of the root, whereas in alkaline soils the CO_2 is rapidly disposed of. In support of this view is the fact that forced aeration of acid soils destroys their inhibiting action.

Another soil condition that may profoundly affect the parasitism of fungi that attack underground parts of plants is the above-mentioned competition, or even sometimes the antagonistic action of the pre-existing soil inhabiting fungi with parasitic soil invaders. The importance of this factor has been particularly stressed in studies of the biology and parasitism of *Ophiobolus graminis* (Part II, p. 377) and *Actinomyces scabies* (Part II, p. 486) in England and of several parasites of cereals, potatoes, and flax in Canada and the United States. Potato scab can be reduced by green manuring and ploughing in organic material, because of the antagonistic action of saprophytic species of *Actinomyces* and other organisms which develop in abundance on the fresh organic matter [20, 34]. A difficulty in infecting wheat with the ascospores of the take-all fungus was overcome when the seedlings were grown in sterilised soil, while in experiments in Canada and England not only was infection by this parasite suppressed by the antagonistic action of several fungi and bacteria but the filtrates from the cultures in which the latter grew had the same effect [29]; the effect was most marked under acid conditions. This factor rather than CO_2 accumulation has been held in Germany to explain the poor growth of *O. graminis* in compacted soils [60]. In Canada also it was found that the growth of *Helminthosporium sativum* in sterilised soil might be completely suppressed by the addition of a little unsterilised soil [23]. This cereal parasite appears to be very sensitive to growth inhibition by other fungi in culture, as it

was unable to grow on nutrient agar nearer than 15 to 25 mm. from colonies of *Aspergillus niger* and several species of bacteria. Similarly the seed-borne flax parasites *Polyspora lini* and *Colletotrichum linicola* were shown in Canada, inactivated to a marked degree when the contaminated seed was sown in natural as compared with sterilised soil. The common soil inhabitants of the genus *Trichoderma* have a marked destructive action on some of the less specialised hemi-parasites, such as *Rhizoctonia solani* and *Pythium de baryanum*. In the United States it has been found that *Trichoderma lignorum* behaves as a parasite on *R. solani*, its hyphae coiling round those of the latter and killing them. The lethal action is due to a substance which can be extracted from the young hyphae or obtained by filtration. Filtration has also yielded a crystalline toxin from the culture media in which *Gliocladium fimbriatum*, a mould antagonistic to *Rhizoctonia*, was grown. This ' gliotoxin ' has a greater fungicidal effect on *Rhizoctonia solani* and *Sclerotinia fructicola* than copper sulphate and is also bactericidal; its elementary formula and molecular structure have been described [14, 57].

Of recent years extensive literature [55a] has accumulated on the antagonisms of micro-organisms — of fungi against bacteria, of fungi against fungi, in mixed cultures. The more important investigations are, perhaps, those concerned with the action of various Penicillia and Aspergilli in repressing the growth of bacteria. Even *Penicillium notatum*, however, the producer of ' penicillin ', represents a highly variable organism, some strains producing the substance in relatively great quantities, others producing little.

The Influence of Nutritional Factors on Disease

The influence of fertilisers and other nutrients applied to the soil on the initiation and course of plant diseases is more difficult to assess than that of the factors hitherto considered because of the modifications of structure and metabolism that may result from differences in the nutrient supplies of the host plant and may affect the entry and growth of the parasite. The vast amount of work that has been done on the effects of nitrogen, potash, and phosphorus on many diseases has added little to the conclusions reached long ago that high nitrogen increases susceptibility to many diseases affecting the green parts of plants, while potash increases resistance, and the influence of phosphates is variable. Many explanations have been given of these actions, some little more than guesses. The factors which determine the resistance or susceptibility of the host plant are mostly unknown ; they are often intimately associated with intracellular processes, and the substances concerned have seldom been identified. Variations in nitrogen level seem to act mostly on the cell contents, solutes, amino-acids, or proteins, while potash is known to intensify cell wall development and this has been correlated in several cases with resistance to parasitic attack. Actual tests of resistance of the membranes to mechanical penetration, as already mentioned, have shown a correlation between this resistance and resistance to a parasite. The considerable number of cases reported in which discrepant results have been obtained by different investigators, or in which no influence of the nutrient on the intensity of the disease could be detected, has suggested to some pathologists that other factors than nutrition have a preponderant effect. It may be noted that a similar difficulty

in obtaining precise and constant data on the influence of nutrition on parasitic diseases has been encountered by human and animal pathologists.

It has been pointed out that, in endeavouring to determine the influence of particular fertilisers on a disease such as rust, varieties of the host plant should be selected that are neither highly resistant nor highly susceptible to the parasite [18]. In a very resistant plant the factors for resistance (possibly some constituents of the cell contents) may be present in such quantitative amounts that the modifying influence introduced by the treatment is insufficient to effect the change over from resistance to susceptibility; the same quantity introduced into the cells of a moderately resistant plant, however, may turn the balance. This consideration seems to apply whatever the factors of inherent resistance may be, and equally to the reverse change from susceptibility to resistance. It has more than once been reported that while fully immune varieties are little, if at all, influenced in their reaction by nutritional treatment, varieties that are merely resistant can be modified to some extent (e.g. in club root of turnips) [38].

In cases where an influence on parasitic diseases from these three essential nutrients has been detected, it has generally been found that the intensity of the disease is least when a well-balanced 'complete' fertiliser is used. Thus in experiments on potatoes in Lincolnshire it was found that the highest percentage of healthy plants was given by the 'well-balanced' plots, and that this increased up to nearly double that in plots receiving no manure with increasing quantities of the fertiliser, to the limit tested of one ton per acre. The diseases involved were blight and some tuber diseases such as those caused by *Rhizoctonia solani* (Part II, p. 524) and *Colletotrichum atramentarium* (Part II, p. 531). Excess nitrogen favoured disease, but this could be counteracted by an adequate amount of potash [33]. Other experiments, however, indicate that the increased susceptibility due to excess of nitrogen cannot be fully counteracted by any increase in the potash supplied. The number of healthy plants increased with increase in the potash, but a heavy dressing of phosphate appeared to decrease resistance in a manner similar to that caused by nitrogen.

In the long-established and classical series of fertiliser experiments at Rothamsted, the high nitrogen plots are the most susceptible to rusts, and the same is true in the plots on the lighter soil of Woburn, which have also been established for many years. It is of interest to note that while nitrogenous manuring has been observed to increase apple scab in England, grassing down of the orchard diminishes it [35]; putting an orchard under grass tends to deplete the nitrogen available to the trees. Prolonged investigations in the United States and elsewhere indicate that, on certain soils and under certain weather conditions, the increased susceptibility to rusts of wheat receiving an excess of nitrogenous fertilisers is in large part due to indirect effects of the fertilisers in increasing the density of the stands and lengthening the period of exposure to infection. Similarly with crown rust of oats under different manurial treatments, the more vigorous host tissue supported a more luxuriant parasitic mycelium, though under a given treatment the largest plants had the least infection per unit area of the most severely infected leaf [39]. But on some of the Woburn nitrogen plots, for instance that getting only nitrate of soda for many years, the growth of the wheat is far from luxuriant,

so that density of stand cannot be responsible for the increased rust susceptibility observed in this case. Wheat and barley mildew, *Erysiphe graminis*, is also worst on the nitrogen plots at Woburn, especially that getting nitrate of soda, and in general it has been found that the powdery mildews resemble the rusts in their response to fertilisers. In water cultures, also, it has been found that both rust and mildew are worst when the quantity of nitrogen required for healthy growth is doubled or trebled, and this occurs whether the nitrogen is given as sodium nitrate or ammonium sulphate [47]. Ammonium carbonate, however, was found, in Italian experiments, to be much less potent than ammonium sulphate in equivalent quantities of nitrogen in enhancing susceptibility to cereal rusts [37].

In the United States some evidence has been obtained that both potash and phosphorus increase the resistance of wheat to *Puccinia triticina* (Part II, p. 358). The potash-starved plots at Rothamsted are always the first to succumb to wheat and mangold rusts in bad years of disease. Potash manuring is sometimes recommended as a means of decreasing damage from various parasitic fungi. In water cultures in which the amount of potash necessary for satisfactory growth was doubled or trebled, a reduction in the amount of wheat rust and mildew was obtained, though this effect did not fully neutralise the predisposing action of an excess of nitrogen. In some experiments in the United States, flax plants supplied with phosphates were more severely attacked by flax rust, *Melampsora lini*, than those receiving nitrates [22]. In these experiments the intensity of the rust attack was directly proportional to luxuriance of growth of the host; plants receiving phosphate were particularly luxuriant, and the effect of the phosphate, therefore, may have been indirect. In the Italian water-culture experiments mentioned above, phosphoric acid given alone slightly increased the susceptibility to rusts, but when combined with potassium, resistance to oat and bean rust was increased, that to *Puccinia glumarum* on wheat was slightly diminished, and maize rust was unaffected.

In such obligate parasites as the rusts and mildews, direct nutritional studies on the fungi are impossible, since they cannot be grown in culture on nutrient media, and only their behaviour as influenced by the nutrition of the host can be observed. In field experiments on them, modifications in the microclimate are caused by the variations in the density of the stand due to different fertilisers treatment, and it is difficult to distinguish between the effects of these two sets of factors. In water cultures, however, the microclimate can hardly play a part. Other factors that have to be considered are the intensity of carbon assimilation and the rate of growth of the plants, both liable to be affected by fertilisers. In general, it appears that active carbon assimilation increases susceptibility to obligate parasites of the green parts of plants. It is possible to check the development of the uredo mycelium of some cereal rusts after infection by depriving the air around the plants of carbon dioxide. There is evidence also that the rusts do best on the plants making the most rapid growth and those having the greatest transpiration. Heating or cooling the roots so as to interfere with their normal functioning will sometimes check the uredo stage of cereal rusts. The interaction of these and other factors adds to experimental difficulties.

Extensive tests have also been made in the United States of the influence

of the three fertilisers on the oat smuts, *Ustilago avenae* and *U. kolleri* (Part II, p. 403). When grown in sand alone or containing a complete nutrient solution, or the same with no or excess nitrogen, potassium, or phosphorus, there were marked differences in the rate of growth of the plants, the height, the number of tillers produced, and the length of the period from planting to heading; there were, however, no significant differences in the percentages of smut resulting from inoculation. Provided that the parasites were given suitable conditions for penetration into the base of the plant (in these smuts a soil temperature of about 20° C., a low soil moisture, and a neutral reaction seem to be most favourable for infection), modifications in its subsequent growth, produced nutritionally, were without any evident effect on the amount of visible disease. With most smuts that infect during the seedling stage, any treatment that tends to delay the emergence of the seedlings from the soil may increase the chances of entry of the parasite, but this action must be distinguished from nutritional influences, which appear to be slight on this class of diseases. The results of experiments in Germany, in which potassium chloride dressings increased the amount of bunt in wheat, while phosphate and calcium cyanamide, especially the latter, reduced it, may have been due to action on the spores, or on penetration, rather than on the course of the disease.

The influence of fertilisers on the late blight of celery due to *Septoria apii* (Part II, p. 630) has also been studied in the United States. As with flax rust, though *S. apii* is not an obligate parasite, it was found that, in a general way, anything that favoured the growth of the host increased susceptibility. Both the number of spots on the leaves and the size of the individual spot were affected in this way. Etiolated plants, grown in the dark for nine days, had less than half as many spots on each leaf, and these were smaller than those on the plants in normal light; the reduction in leaf area in the darkened plants was not enough to account for this. Susceptibility was also much reduced when the plants were severely infested with the root knot nematode, *Heterodera marioni*. In the allied fungus, *Septoria lycopersici*, the cause of tomato leaf spot in the United States, it has similarly been found that favourable conditions for growth increase susceptibility, unfavourable diminish it. Enlargement of the leaf area by increasing amounts of sodium nitrate increased the number of infections; while the leaf size was increased to a maximum of 77 per cent., the infections were increased by 55 per cent. Phosphate and potash sometimes increased, sometimes diminished the leaf area and the number of infections in this case. It is difficult to secure successful infection with *Cystopus candidus* on plants heavily infested with green-fly, and both with this fungus and the cucurbit mildew, *Erysiphe cichoracearum*, young leaves that have gone yellow may be immune. In many of the attacks by strong parasites there appears to be a critical stage of nutritional receptivity in the organ attacked, above which infection occurs, while the depleted organs become immune once their health is permanently lowered. Of the downy mildews generally it has been said that the more healthy a plant is, the more the parasite appears to prosper on it.

Some of the cases cited show that it may be possible by judicious manuring to increase the vigour and the extent of the assimilating parts of a plant in greater

proportions than the increase in the development of its parasites, even when these are of a type that is favoured by the particular fertilisers used. The destructive coffee-leaf disease, caused by the rust *Hemileia vastatrix*, first broke out in Ceylon in some of the best young plantations in the island, and its development is favoured by the luxuriance of the young foliage, but not to such an extent as to balance the improvement in growth caused by liberal manuring. Hence coffee-leaf disease is fought in some areas quite successfully by heavy manuring. The converse is naturally true also, for even a less severe attack on a weakly plant may do more damage than the heavier infection of a more luxuriant one. Disease endurance is often nearly as important in such cases as true inherited resistance.

Many diseases have been reported to be favoured by unbalanced nutrition or by deficiency in one or other of the three primary fertilising elements or in the food reserves stored in the plant. Most of these, though by no means all, are diseases caused by less virulent parasites than those considered above. It is well known that there is a host of fungi and bacteria that ordinarily do little damage to plant organs in full vigour but that increase in their parasitic capabilities as the host plant becomes enfeebled from any cause. Unbalanced nutrition has been implicated in the cereal browning root rot in Canada, caused by *Pythium arrhenomanes*, and the sugar-cane root rot in Hawaii which is associated with the same or allied species. A high nitrogen and low phosphate content is characteristic of the soils in which these diseases occur. That other factors than nutritional may be involved in this type of disease appears to be shown by experiments with the sugar-cane Pythium root rot in Louisiana, where it occurs in water-logged heavy clay soils. Additions of salicylic aldehyde, one of the substances that accumulate in soils under semi-anaerobic conditions, greatly increased the injury over that caused by either *P. arrhenomanes* or the chemical compound alone. Indeed the latter by itself, at the strengths used, had little effect on the growth of the cane.

In a high proportion of the cases in which nutritional unbalance affects the incidence of parasite disease, the action is mainly, as might be expected, on the receptivity of the tissues of the host rather than on the parasite. Stored apples are liable to rotting from a number of weak parasites, and it has been found that the manurial treatment of the trees from which the apples were picked affects their liability to these fungal rots. For instance, *Cytospora ludibunda* has been found to produce a more active rotting, as judged by the rate of advance of the mycelium in the tissues, in apples from trees that had received sulphate of ammonia than from those receiving no nitrogenous manure. The resistance of potato tubers to the storage rots caused by certain bacteria was similarly found in Germany to be greatest when the plants were grown without nitrogen ; the heaviest injury occurred when the plants had received an excess of ammonium sulphate or calcium cyanamide, while the middle lamella of the cell membranes was most resistant to solution by the enzymes of the bacteria in tubers grown with an excess of potash but no nitrogen or phosphate. Resistance to the dry rot of the tubers caused by *Fusarium caeruleum* (Part II, p. 532) was highest in tubers from plants receiving an excess of potash and no nitrogen, and lowest in those deprived of potash.

Sometimes, however, it is far from easy to decide whether the influence of

nutrition on disease is due to action on the host or the parasite or on the two in association. In the allied wilt diseases of cotton in America and pigeon pea in India (caused by strains of *Fusarium vasinfectum* or by distinct though closely related species, according as one defines loosely or tightly the limits of species in the genus *Fusarium*) the reaction to manurial treatment differs sharply. In the former disease nine years' experiments carried out at fifteen centres showed that applications of potash reduced wilt in almost all cases, while acid phosphate increased it, as did, to a lesser extent, acid phosphate and nitrate together. In tests on a resistant and a susceptible variety of cotton, while the same general results were obtained, excessive applications of potash lowered yields in the resistant variety while not appreciably affecting those in the susceptible one. In much of the area covered by these experiments, however, the well-known symptom of potash deficiency known as ' rust ' is common and the correction of potash hunger may be a factor in these results. In the pigeon pea wilt in India there is no indication that potash deficiency contributes to the injury. In the series of ' permanent ' manurial experiments started at Pusa shortly after its foundation, in which pigeon pea was one of the rotational crops and always showed some wilt, it became apparent after several years that the disease was markedly affected by the fertiliser treatment. Careful records during a number of years showed that applications of superphosphate at the annual rate of about 13 lb. phosphoric acid (P_2O_5) per acre or of farmyard manure sufficient to give 30 lb. nitrogen per acre, consistently increased the percentage of wilt, while green manuring reduced it. When a plot received green manure with superphosphate, more wilt occurred than in the no-manure plots, but the increase was always significantly less than when superphosphate alone was used. Chemical fertilisers other than superphosphate had no consistent effect on the incidence of the disease. The *p*H value of the soil was not appreciably different in the different plots, and there was no relation between moisture content down to two feet and the percentage of wilt. The plants grew most luxuriantly in the superphosphated plots (average diameter of the main stem 50 per cent., and average weight of shoots at harvest time in two sets of plots 37 and 47 per cent. greater than in those not given superphosphate), while in culture the rate of growth of the fungus increased with concentrations up to 0·5 per cent. of the phosphate. The fungus remains in the soil from year to year, for there is only a small proportion of seed-borne infection, and it was shown to be able to spread nine feet through the soil during the growing season in the unmanured control plots, mainly along the roots which were traced for 6·5 feet laterally. But whether the soil treatment acts on the fungus or the host or the two in company is not evident.

Similarly there is no evidence whether the increase in the eyespot lodging of rye, caused by *Cercosporella herpotrichoides* (Part II, p. 384), that has been observed in prolonged experiments in Germany to result from fertilising with nitrogen or phosphorus and nitrogen, without potash, is due to action on the fungus or on the host or on both when associated. A complete fertiliser or one deficient in phosphorus alone had no effect on the disease, which was reported as being generally most severe in the better soils. This fungus persists from year to year mainly on stubble or debris from previous cereal crops. It resembles the *Fusarium* wilts

last mentioned in having more decided parasitic tendencies than some of the species that rot storage organs, discussed earlier.

In the truly obligate parasite *Plasmodiophora brassicae* (Part II, p. 559) it has been found that susceptible (but not immune) varieties of turnips had a higher percentage of infection when nitrogen and potassium were in excess, the nitrogen effect being the most marked. Lack of nitrogen also increased the percentage of attack but lack of potash greatly reduced it. In the other direction it has been reported in the United States and Australia that the pea-root rot caused by *Aphanomyces euteiches* (Part II, p. 609) is reduced by nitrogenous fertilisers. This, and the similar experience with the Texas root rot of cotton (*Phymatotrichum omnivorum*) are amongst the very few cases in which a beneficial result from nitrogenous applications to an annual crop has been recorded in pathological literature, but at least they serve to indicate how obscure the interactions in nutritional experiments may be even when nitrogen, the element which has given the most consistent results, is concerned [1].

As regards the influence of other elements than nitrogen, potassium, and phosphorus the effects sometimes produced by them are not clearly known to be due to any direct nutritional action. Deficiency diseases due to lack of iron, manganese, copper, and so forth (separately discussed in Chapter IX) are sometimes accompanied by increased injury by certain parasites, but it would be stretching analogy too far to attribute the injury to nutritional factors. Calcium is chiefly of importance as affecting the reaction of the soil, though in some diseases its action appears to be on the metabolism of the host. Thus in Australian experiments with *Urocystis tritici* on wheat, calcium deficiency reduced, and calcium excess up to twice the 'normal' dosage increased, infection in the susceptible Free Gallipoli variety; four times the normal dose had no effect, nor when the resistant Gurkha variety was used could a consistent action be observed to result from any treatment. In Free Gallipoli wheat the calcium content of the plants was correlated with reaction to the smut, for there was an optimum concentration of calcium which promoted the expression of the symptoms. Silica has been reported to increase resistance to wheat mildew and to the rice diseases in Japan caused by *Piricularia oryzae* and *Ophiobolus miyabeanus*, but this is evidently correlated with the increased silicification of the cell membranes through which these parasites penetrate. Lack of sulphur increases the percentage of turnips attacked by *Plasmodiophora brassicae* [48]. Lithium has been shown to have a marked effect in enhancing rust resistance and also the resistance of seedlings of *Phaseolus vulgaris* to attack by *Botrytis cinerea* [12]. Recent studies at Cambridge [28] established a high inverse correlation between the concentration of lithium in celery leaves and the amount of leaf spot caused by *Septoria apii*, and a less marked but significant relationship between the amount of lithium absorbed by wheat seedlings and resistance to *Erysiphe graminis*. Wheat brown-rust and tomato crown-gall were also reduced by lithium. In the last-mentioned disease lithium chloride application increased the weight of the plants relatively to the controls, but it is very doubtful whether any nutritional factor can be concerned.

Apart from mineral nutrition, the depletion of the starch reserves caused by over-bearing or over-pruning or, in tea, by over-plucking has been observed to

predispose plantation crops to damage from certain weak parasites, such as the root and stem infecting fungus, *Botryodiplodia theobromae*. The interesting relationship to carbohydrate nutrition presented by the root rot of forest trees in tropical Africa due to *Armillaria mellea*, the honey agaric, has already been mentioned. This fungus becomes destructive to tea and other plantation crops after the trees have been felled in clearing the land for planting. Unless the roots contain starch, the fungus is unable to spread within them or to form the rhizomorphs which enable it to infect the roots of the planted crop. For the production of rhizomorphs a medium rich in carbohydrates is required. It has been suggested that control may be effected by ring-barking the trees so as to deplete their roots of starch before felling. It is possible, in this case, that the weak parasites, such as *Botryodiplodia*, which rapidly colonise the starch-depleted roots, may compete with, or exert an antagonistic action on, the honey agaric, and that the inhibition of its growth in the ring-barked trees may be due to a combination of factors. Other root diseases may be similarly affected, e.g. that caused by *Poria hypolateritia* in Ceylon.

In many cases in which the enfeeblement of a plant predisposes it to parasitic attack it is not known whether nutritional or some other factors are involved. Insect attacks are known sometimes to increase liability to fungal damage, and vice versa. In the West Indies the amount of root disease in sugar-cane, associated with *Marasmius* and other weak parasites, has been observed to be almost proportional to the severity of the infestation of the cane by froghoppers (*Tomaspis saccharina*), while it is sometimes possible to identify cacao and coffee plants suffering from the root rots caused by species of *Rosellinia* by the readiness with which they become affected by aphides and other insects. Assimilatory disturbances are possibly at the bottom of these effects. The surface canker of apples in the south and west of England attributed to *Myxosporium corticola*, and of stone fruits due to the same and *Diaporthe perniciosa*, were found to occur only on trees weakened from other causes. Similar instances could be multiplied, but it is important to note that precise data of the kind are the most easily obtained and the best-authenticated the more the fungus belongs to the class of facultative parasites, that is to say organisms that develop and persist normally as saprophytes but can act as parasites at times. On the other hand, the more closely the organism approaches to the obligate parasites, even when it is to some degree a facultative saprophyte able to live at times apart from its living host, the more difficult it is to get positive evidence of a consistent nutritional influence on disease outside that produced by nitrogen and, perhaps, potash. Failure to appreciate the difference between these two classes of disease-inducing organisms, even though the difference between them may be one of degree rather than of kind and should not be exaggerated, is responsible for many misleading opinions and even for such preposterous statements as that crops grown under optimum conditions will remain free from fungal disease.

There is no reason to doubt that reaction to disease can be and is influenced by nutrition. The evidence regarding the influence of nitrogen on obligate parasites seems to be conclusive, and that regarding potash is little less convincing. Outside these two elements it is often contradictory or capable of interpretation

on other than nutritional bases. Until there has been a considerable advance in knowledge of the influence of excess or paucity of particular elements upon the nature of the cell contents or of the cell membranes (in short, upon metabolism), the pathologist is left with no sure guidance as to the limits of nutritional influence on disease ; in this he seems to be little better or worse off than his colleagues in the fields of animal and human pathology. The grosser effects are sometimes evident enough but the intracellular reactions are not well understood. It is, perhaps, in this field that the plant pathologist is most in need of help from the physiologists, for it seems that the progress of the applied science in a direction which might have significant practical results is being delayed by inadequate basic knowledge.

Influence of External Factors on Inherited Reaction to Disease

In the course of genetical investigation, light has been thrown on the extent to which external conditions, meteorological, nutritional, and so forth, can modify the expression of the reaction of plants to disease when this reaction has a genetic basis, that is to say, when it is due to the presence or absence of genes affecting the character of resistance or susceptibility.

When Biffen's work on the inheritance of resistance to yellow rust (*Puccinia glumarum*) of wheat was repeated at Cambridge between 1917 and 1920 it was found that the inherited predisposition or resistance to attack, though following the Mendelian laws for monofactorial inheritance, was liable to modification by external environmental characters, such as abnormal temperature conditions and the application of certain fertilisers. In the cases in which the resistance of the ' immune ' lines of wheat was reduced, however, the action of the parasite was of a very limited nature. Furthermore, in a line which was homozygous for the main factor for resistance to the rust, it seemed that other separately inherited factors, possibly those affecting the metabolism of the plant, might modify the degree of resistance. Many other cases of the kind have since been recorded. So also it was found possible, in German investigations, to effect considerable modifications in the reaction of the standard collections of wheats that are used to differentiate physiologic forms of *P. glumarum* and *P. triticina* by drastic variations in the nitrogen and potash supplied. In Australia physiologic form '59' of *P. graminis tritici* has been found to modify its degree of attack on one of the test varieties, so as to behave like form ' 23 ', under certain conditions of temperature, and in America the resistant reaction of some of the test wheats to a particular form of this rust, at 50° F. has been changed to a full susceptibility one at 70°. Hence it is that under the temperature conditions of the Gangetic Plain in India wheat that had shown a high degree of resistance to rust during the normal and relatively cool wheat-growing season from October to April, became heavily rusted when the growing season was extended into the hot weather : in North America, also, high temperature has been found to break down resistance to black rust in a number of cases [27]. Similarly the reaction of oats to some of the physiological forms of crown rust, *P. coronata*, can be greatly modified by temperature ; certain varieties of oats are resistant to form ' 7 ' of the rust at low (55° F.) and susceptible at high (85°) temperatures. In a cross between Red Rustproof and

Scotch Potato Oats carried out in Wales, the resistance of the former to the physiologic race used was found to be dominant and unifactorial.

The resistance of cabbage to *Fusarium conglutinans* in varieties which are not fully homozygous for resistance becomes reduced under sèvere field conditions, and at soil temperatures at or above 22° C. the commercially resistant Wisconsin Hollander variety may become quite severely attacked; 100 per cent. of disease was obtained at a soil temperature of 24°. But the homozygous resistant variety, Wisconsin Ballhead, remained quite immune at 22°.

Many of the cases of this kind that have been recorded are most easily interpreted as resulting from the action of the environment on accessory factors for the character modified.

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Chapter VI

MORBID ANATOMY AND HISTOLOGY

THE powers of regeneration and wound repair possessed by the plant are considerably greater than those of the animal body. To understand the part that they play in plant pathology it is necessary first to consider the processes of tissue protection and repair in the absence of disease. Some of those that will be mentioned have been observed under the specialised conditions of laboratory experiments, but they illustrate the potentialities of plant tissues in striking fashion.

REGENERATION

In many cases the tissues of growing parts of plants, destroyed by traumatic action, can be regenerated from tissues of different origin. The conventionally distinct derivatives of the dermatogen can be replaced from those of the periblem and those of both the dermatogen and the periblem can be reconstituted from those of the plerome. Exposed young cortex cells may directly become epidermal by thickening their outer wall, acquiring a cuticle and even forming hairs or stomata. This occurs quite normally in citrus leaves, in which during development the upper epidermis splits and is replaced from the mesophyll, sometimes after a few cell divisions. Still more remarkable is the replacement that has been effected under suitable conditions of the entire lost halves of a young tuber, stem or petiole, in form and structure very like the normal. A perforating wound involving the central cylinder may become surrounded, not only by a cork layer, but by a deeply seated cambium which may cut off xylem on the side away from the wound and phloem towards it, the part between phloem and cork coming to resemble a cortex. In perforating needle wounds vertically down to the pith in young sunflower heads the cavity can become lined with a complete vascular ring, and bundles of the hard bast type develop on the side of the phloem towards the cavity; then follows a thin-walled parenchyma resembling a cortex, and finally, lining the cavity, an epidermis forms, sometimes with hairs like those on the surface of the plant (Fig. 147). Even natural hollows in the stem of *Brassica oleracea* can develop a cambium with phloem next the cavity and xylem outside, and the injection of certain chemicals (e.g. monobasic ammonium phosphate) has caused the same condition in the hollows of young internodes of *Ricinus*. The restoration of lost parts, however, is ordinarily preceded by the development of repair tissue on the denuded surface.

WOUND REPAIR

The commonest precursor of the replacement of lost tissues under moist conditions, especially in herbaceous plants, is callus formation. Callus can be

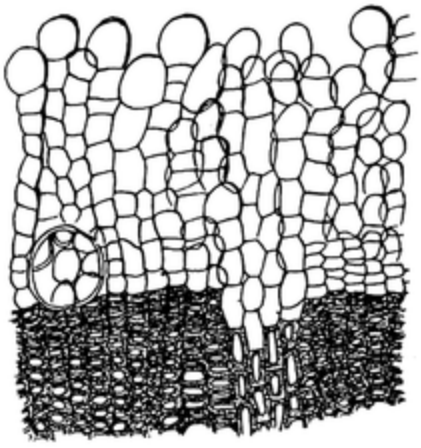


FIG. 131.—Transverse section of stem of *Robinia* showing formation of callus tissue from the cambium (after Frank)

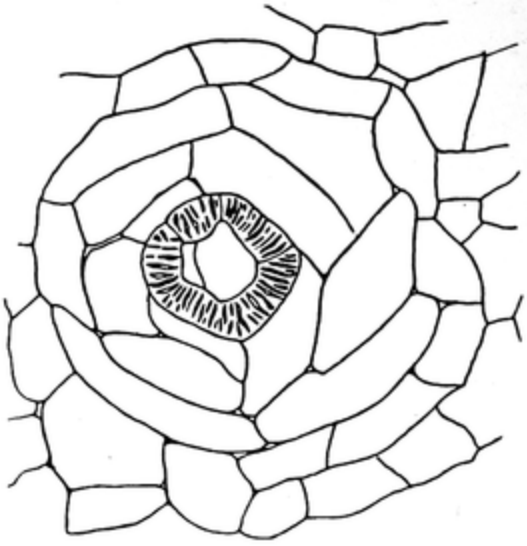


FIG. 132.—Tracheid formation in the callus from a root of *Beta* (after Kuster. Butler, *Ann. App. Biol.*)

formed in suitable circumstances from any living cell or tissue, and the subsequent changes that it undergoes are independent of its origin.

The living cells bounding the wound below those that have been killed in wounding grow out, multiply, and form a tissue of dividing thin-walled parenchyma (Fig. 131). Sooner or later differentiation occurs in this tissue. Its outer margin may become corky by the development of a cork cambium or may consist of a loosely united mass of cells, resembling the so-called hyperhydric tissue. This may arise from a meristem which cuts off roundish, loosely attached cells outside and thin-walled callus-like cells inside, or it may grow without the intervention of a meristem, since callus cells far removed from the level of the wound may continue to divide and form ever-increasing masses of thin-walled parenchyma. In the deeper part of the callus the first differentiation often is the direct transformation of a callus cell, derived from any tissue, by reticulate thickening and lignification of its walls, into a tracheid-like element (Fig. 132). This is one of the key processes in wound repair. Several of these tracheids often develop close together, forming a little island of woody tissue. Neighbouring groups may unite to form irregular strands; or an island may become wholly or in part surrounded by a meristem which forms tracheids and fibres on its inner side (cf. Fig. 146 D). At a later stage a procambial strand, forming wood and phloem, may develop in the cells bordering the tracheid groups or strands, and may join up with vascular outgrowths from the original conducting system of the organ. A similar process, without the formation of callus, can restore a broken conducting tract — say, in a leaf — cells of the mesophyll in which become changed into short tracheids which can bridge the gap and which, later, can become bordered by a procambial strand forming phloem and xylem (Fig. 133).

Very often a large transverse cambium develops below the free surface of a callus and cuts off wood on its inner side and, less frequently, phloem and secondary cortex-like tissue on its outer side. Occasionally a layer resembling an epidermis clothes the surface, especially when a petiole or stem is split longitudinally. In

such a case a central cylinder may be reconstituted by a cambium developing in the callus and joining up with that of the uninjured half; this forms not only phloem but a more or less normal cortex and epidermis outside.

Finally a callus may form shoots exogenously from a mother cell or cell group at or near the surface of the callus. This may happen even in a callus derived from the pith. Shoot formation seems to be stimulated by excessive or unbalanced accumulation of food or of growth-promoting substances, for in uncongenial grafts the upper side, where the imperfect union holds back the passage of material from the scion, may develop large callus-like masses, rich in shoots. In the decapitated swollen hypocotyl tuber of cyclamen there is a formation of adventitious cotyledonary leaves. The vascular system which supplies these arises from the division of one or more of the normal cortical cells, and this process extends back to a bundle. There may be a central mass of xylem surrounded by phloem in these adventitious leaf traces, or xylem and phloem may pursue a wavy course, not necessarily close together, towards the vascular ring (Fig. 134). In callus buds the vascular connection may be established in a similar manner. Roots are very often formed in callus but, unlike shoots, they always come from the deeper layers, usually from the neighbourhood of the pericycle but also sometimes from the medullary rays and the external region of the pith. As the horticulturist well knows, different species vary greatly in their readiness to develop roots from the callus of a cutting. Good callus formation does not necessarily mean good root production. It would take us too far to follow the extensive studies that have been carried out during recent years of the part played by phytohormones in the regeneration of roots and shoots and of the practical application of these studies in horticulture.

A second type of wound healing is common and is, indeed, the normal process in woody tissues exposed to ordinary air. In this the repair resembles the normal growth in thickness in that it is the result of the division of cambial cells, the products of which, as usual, soon lose the

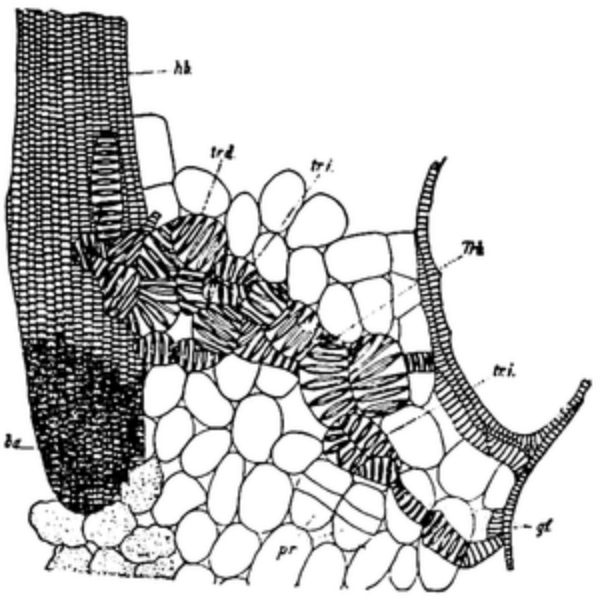


FIG. 133.—Replacement of vascular connection destroyed by wounding in a leaf of *Impatiens*; tracheids are formed directly from mesophyll cells (*tr.d*), or after division of latter; a procambial strand is beginning to form at *pr*. (from Kuster, after Freundlich. Butler, *Ann. App. Biol.*)



FIG. 134.—Portion of trace of adventitious leaf of *Cyclamen*, cut longitudinally, *ph*, phloem (after Boodle. Butler, *Ann. App. Biol.*)

aptitude to divide, becoming differentiated. The tissues thus formed are sometimes termed wound wood and wound cortex.

If the wound is an open one extending beyond the cambium, or is the result of an amputation as in pruning twigs or lopping branches, the first stage in the process of healing is the blocking of the tissues by the deposition of suberin in the cells, intercellular spaces and cell walls of parenchymatous tissues and an accumulation of an insoluble gummy substance in the cavities of the conducting elements of the wood. The position of maximum formation of this blocking layer is marked by a brown or dark-green band situated at a variable distance below the surface of the wound, near the surface in the cortex, deeper in the pith. Its rate of development also varies with the stage of activity of the tissues : when growth is active as in the spring and summer it appears in ten days to a few weeks, but in wounds made in the late autumn or winter the completion of blocking is delayed until the spring ; young woody tissues are also more rapidly blocked than old. The chemical nature of the blocking substance is not clear. In large parenchymatous masses of tissues it is a product of fatty acids but in woody tissues it is a viscous or resinous substance, usually termed ' wound gum ' though it is not a true gum. Sometimes a ' gum zone ' forms in the pith and wood of an amputating wound and a band of cork at the same level in the cortex. When wound gum forms in ray or wood parenchyma cells containing starch, the starch first disappears, and it has been suggested that wound gum may be formed from products derived from the starch together with resinous and ligneous substances and pectin. When micro-organisms invade the cut tissues they may stimulate the formation of wound gum. Gum barriers, whether formed normally as a result of wounding or as a reaction of the living cells of woody tissues to invasion by a parasite, are considered to play an important part in checking the invasion of parasitic fungi through wounds. The readiness with which they form differs in different trees. In the red gum (*Liquidambar styraciflua*) they develop rapidly under ' fire scars ' in several of the layers of the outer sapwood, and resist the attack of *Polystictus pergamenus* and *Polyporus gilvus* when the unprotected sapwood is heavily decayed. In oak, ash, and *Celtis* similar fire scars result in early infection by various fungi.

The formation of the blocking layer is followed by an overgrowth of callus mainly derived from the exposed cambium but by no means entirely so. The relative parts played by the cambium and by the other living (usually parenchymatous) tissues in re-clothing the exposed surface of a stem wound depend to a great extent upon the nature of the wound. When this is a tangential one caused by removing a slice or layer as, for instance, when a portion of the bark is peeled off a stem or a shaving cut out, perhaps including some of the outer wood, there is extensive destruction of the cambium, and regeneration of the bark is affected mainly by outgrowths of callus cells from such exposed living parenchymatous cells as those of the medullary rays and wood parenchyma ; only at a later period does cambial activity play a part. The process has been carefully studied in Malaya in its bearing on the ' stripping ' method of treating brown bast disease of the rubber tree, where large areas of the affected and surrounding bark are peeled off and the new bark may develop free from the disease. In *Hibiscus* the

peeled stem surface begins to show outgrowths of callus cells of relatively enormous size in about three days, none coming at this stage from the cambium which is largely destroyed by the stripping. At the central part, medullary ray callus plays the largest part; at the margins, cells of all the tissues external to the cambium, rays, phloem parenchyma and cortex, are involved. The callus cells divide and come into contact with one another laterally, so that a continuous clothing of callus forms over the wound. In about another three days, under Malayan conditions, the phellogen from the uninjured bark extends inwards into the cortical callus, and phellogen forms in a continuous layer across the mass of basal callus (derived mainly from the wood) which clothes the major part of the stripped surface. Beneath the cork thus formed the callus cells are preserved from desiccation, but for a time there is no further differentiation of tissues in them. Then, at the level of the severed marginal cambium surrounding the wound, a level which is now, owing to growth in thickness of the uninjured wood, somewhat above the bottom of the callus clothing the stripped surface, a new cambium forms in the callus. This comes mainly from an extension of cambial divisions inward from the regenerated cells of the cut margin of the original cambial ring, involving the callus cells next in contact with these. The process extends right across the wound, so that the continuity of the cambial cylinder surrounding the stem is fully restored. Thenceforward, the wound covering follows the normal course of growth in thickness of a stem. In this type of repair the wound wood and wound cortex closely resemble the normal tissues.

In an amputating wound, however, especially in older woody stems, there is less scope for the activity of callus-forming cells in the wood, and the bulk of the closing tissue comes from the cut ends of the original cambium and the tissues in its neighbourhood. An annular cushion is formed which gradually spreads over the exposed wood, often leaving pockets of necrosed tissue buried at the level of the wound. In this case cambium formation is less regular than in the last, and isolated tracheid groups may form, each bordered by its own separately developed cambium, cutting off wound wood.

The most noticeable masses of wound wood arise when the original cambium, without being exposed, is stimulated into activity by injury to some of its cells or to the bordering tissues, from frost or the attack of parasites. Cankers are largely due to an excessive development of wound wood and are a familiar result of frost and of certain types of insect and fungal injury. The pressure from below caused by this deep-seated growth ruptures the cortex, leaving exposed the newly formed wood with sometimes a dead patch at the base. Frost cankers are more fully discussed in Part II, p. 919. Wound wood may also develop in callus tissue, from cambiums formed, as already mentioned, in any part of the mass. These may cut off only wood, without phloem. When the meristem develops, as is not uncommon, around a tracheid group or a mass of sclerenchyma or a localised injury, the xylem may be produced either on its outer or its inner side. In the former type there may be a small phloem group in the centre of the nodule, in the latter, either phloem or parenchyma develops outside the cambium. Structures of the same kind are also found in wound bast and even occasionally around the tracheid groups that form in leaf calluses. In woody plants they may produce

knobby isolated masses of wound wood, such as are found in the secondary cortex of the apple and other trees; while the woody nodules ('burrs'), which are the most serious concomitant of the 'brown bast' physiological disorder of rubber in Eastern plantations, are composed of xylem developed on the inner side of a cambium surrounding a little island of necrosed tissue in the phloem. Very often the wound wood is developed in vortex-like masses of fibres and vessels, probably as a result of pressure from unevenly developing meristems or uneven growth at the junction of normal and wound wood. Woody galls often show beautifully grained examples of whorls of this nature (cf. Fig. 149).

Wound wood differs histologically from normal wood in having less regular rows, composed of shorter and generally thinner-walled elements, in the usually greater proportion of wood parenchyma and fewer fibres, and in wider medullary rays (Fig. 135). Its less solid construction renders it more liable to damage; thus the new wood in frost cankers is readily again injured by frost, and that

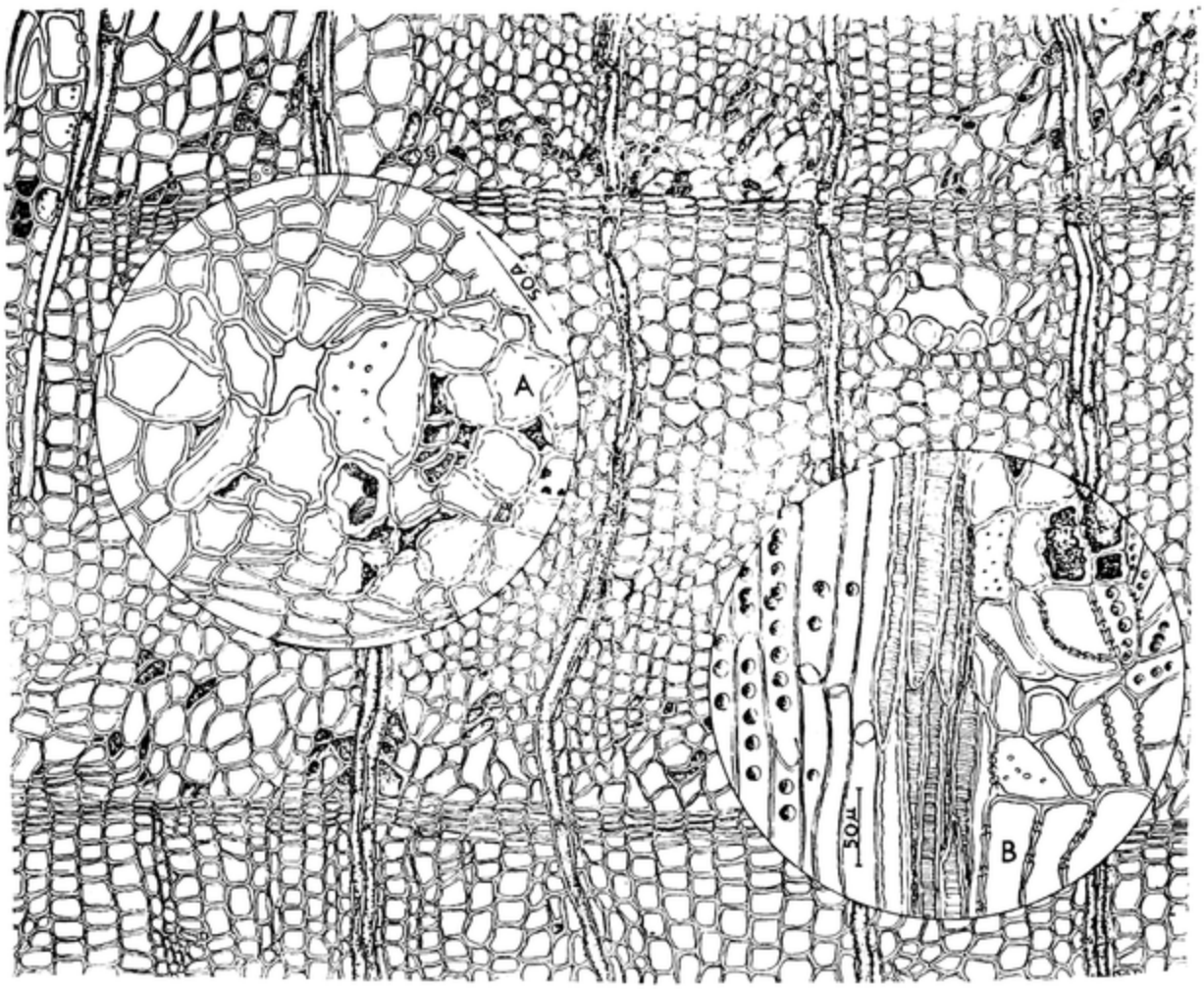


FIG. 135.—Abnormal wood in the stem of larch. The abnormal tissues are in the spring increments outside the narrow increments of autumn wood. The abnormal cells may or may not retain living contents, or become lignified, forming isodiametric elements with simple pits, as shown, inset, in *A* and *B*. Note the gradual restoration to the normal wood in the later-formed spring increments. (Such features of abnormality in the wood of larch and other trees may result from frost action or from disease, or from both)

resulting from woolly aphis in apples and *Helopeltis* in tea is very prone to the attacks of fungi.

Instead of callus or wound wood forming, a wound may be cicatrised by wound cork (cf. Fig. 139). This commonly occurs in all organs of plants in dry air. The cork tends to form parallel to the surface of the wound and often differs from normal cork in having larger cells and thinner walls. When a leaf or a potato tuber is cut, the living cells nearest the wound may enlarge, sometimes with a few divisions parallel to the surface, and their walls become metacutinised or impregnated with a deposit of suberin or of a lignin-like substance or of wound gum. Below this 'blocking layer' the cells elongate and divide transversely, the middle divisions corresponding to a phellogen which cuts off cork cells in rows to the outside and phelloderm rows below. All this part is without intercellular spaces and, in the leaf, the distinction between palisade and spongy parenchyma is lost (Fig. 136 A). Every living tissue from epidermis to cambium or pith may join in forming the cork layer, which always tends to unite at its margin with the surface covering of the sound part, epidermis or normal cork as the case may be. When the epidermis takes part in the process it divides into cells resembling those of the other tissues of reaction, while collenchyma and even young sclerenchyma cells lose their thickening and react like the rest.

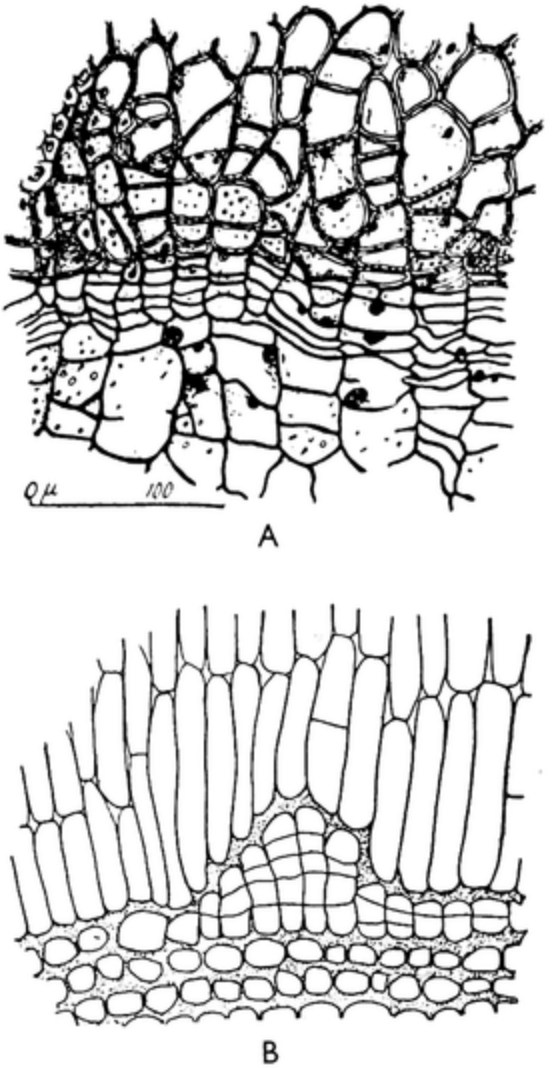


FIG. 136.—Wound cork. *A*, edge of cavity in leaf of holly, mined by *Phytomyza aquifolii*, showing elongation of the mesophyll cells and their division by cross walls to form a phellogen; intercellular spaces absent (after Kerling). *B*, abnormal histological changes from non-traumatic or non-parasitic causes. Hypertrophy and hyperplasy of collenchyma of *Clerodendron* twig after vaselining (from Kuster after Schilling. Butler, *Ann. App. Biol.*)

In superficial wounds in roots and submerged aquatic stems, apparently correlated with the presence of a well-developed endodermis, there may be no other reaction than the formation of the blocking layer, without any division of the cells or development of meristems. The endoderm is believed to act as a barrier to the passage from the central cylinder of the solutes that are necessary for meristem formation.

These various processes of healing are not always sharply divided, and more than one of them may sometimes be seen in different parts of the same wound.

Abnormal Histological Changes from Non-traumatic Causes

Besides the processes that result from traumatic action there is another group of modifications in cell or tissue growth arising from inner conditions in which no gross injury is involved. Only a few examples need be given.

Changes, which may sometimes be deep-seated, can be induced in the cells and tissues of plants merely by the action of environmental conditions, such as moisture or light, or by nutritional factors. It is convenient to distinguish the main basic modifications that may result by the terms *hypoplasy* or under-development, *hypertrophy* or swelling without cell division, and *hyperplasy*, an actual increase in the number of cells (Fig. 136 B). When hypoplasy is total and the organ or tissue does not develop at all, the term *atrophy* is used. These are not the only modifications that can be caused by physiological factors, for the cell membranes and structures such as stomata also respond in varying degree, as for instance in etiolated shoots, sun and shade or submerged and aerial leaves, and over- or under-nourished plants.

Excessive moisture, especially if transpiration is checked, often causes on green stems or leaves the formation of ' intumescences ', such as are commonly seen on tomato and other plants in glasshouses. These are usually white protrusions formed of rows of radially elongated, thin-walled parenchyma derived from the epidermis, and mesophyll or primary cortex. Collenchyma can participate in the process, the cells elongating and forming thin dividing membranes (Fig. 136 B).

Large hyperplastic modifications have been induced in some of the biennial *Brassicae* with swollen stems, such as kohlrabi, by preventing flowering (Fig. 137). Both cortex and conducting tissue became much enlarged, the xylem of the main leaf trace especially so; this was accompanied by an increase in, and broadening of, the medullary rays, so that a number of narrow bundles were formed. Each of these bundles tended to become surrounded by a cambium developed in the ray cells and deeper part of the wood parenchyma. This cambium formed phloem on the ray side and xylem towards the bundle. Small inverse bundles also formed in the enlarged rays or in the protoxylem region; in these bundles there was a nest of phloem surrounded by a cambium producing a little xylem on the outside. Concentric inverse bundles have also been found to develop in the thickened roots and leaf base cushions of similarly

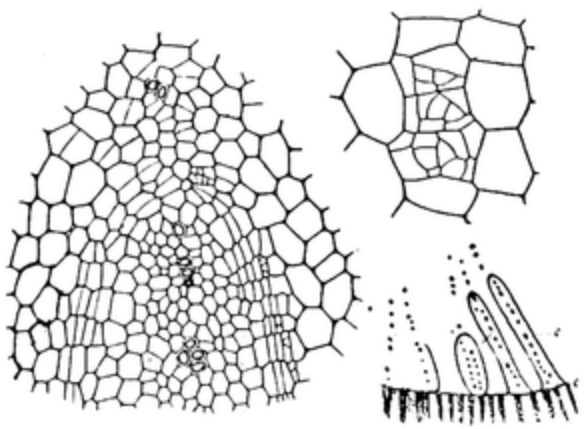


Fig. 137.—Swollen leaf-base cushion of kohlrabi, prevented from flowering by removal of flower buds. Lower right-hand figure, showing new cambium formation in enlarged medullary rays, tending to encircle one or two rows of vessels but leaving the innermost outside the ring. Left-hand figure, detail of inner part of a row of vessels with lateral cambiums uniting in the protoxylem, and a few phloem elements forming on the outer side; cell division active around the innermost protoxylem vessels. Top right-hand figure, a phloem nest in the vascular region of a primary bundle (after Vöchting. Butler, *Ann. App. Biol.*)

treated savoy cabbage, being in this case situated in the collenchymatous tissue of the inner xylem. In the abnormal tissues that have been thus induced to form, there is a noticeable scarcity of fibres and, where the thickening is due largely to cambial activity, as in swollen side shoots of kohlrabi sometimes, the new tissue on the xylem side consists of thin-walled, rather uniform cells.

Petioles have been successfully rooted, and also have been grafted into tubers, when they grew into abnormally thick organs. In such cases the normally flattened collateral bundle of the petiole tends to become concentric, and the petiole comes to resemble a stem in structure and function. The change is due to the formation of new cambium by division in cells of the ground parenchyma, either extending out from the edges of the original cambium and curving round till the two sides unite, or beginning in groups of parenchyma cells opposite the central part of the collateral bundle, the process then spreading laterally and curving down to join the original cambium on each side. Xylem forms on the inside of the new cambium and phloem outside. Ultimately a compact woody cylinder forms, which may be twenty times the area in transverse section of the normal petiole bundle. A small-celled phloem surrounds the wood, and all the elements that are formed outside the original bundle are short, as they are limited by the diameter of the ground tissue cells from which they are derived.

It has, indeed, been shown experimentally that all living plant tissues have potentialities of hypertrophic and hyperplastic reaction to ' physiological ' stimuli in the absence of wounding or of any parasite. In the hyperplastic tissues differentiation to other tissue forms can follow, but there is a tendency for them, at first at least, to show less differentiation in the elements of which they are composed than is found in similarly situated normal tissues. Naturally, cells that have not finished growth and differentiation react the more readily to such stimuli, but old cells can also be affected, either directly or after first dividing and thereby becoming, so to speak, rejuvenated. Even cells that have developed thickened walls, as collenchyma, can enlarge and divide, or those in the early stages of lignification can undergo delignification and resume activity. In the hyperplastic tissue a cell, perhaps only one of a group formed from a single mother-cell, may become a thick-walled element such as a sclereid, or develop into a little island of phloem or a tracheid; or a meristem may appear, with all its powers of tissue production. Similarly hypoplastic modifications can result from under-development brought about by inadequate or unbalanced nutrition or other unfavourable conditions.

Tissue Modifications and Galls caused by Parasites

The tissue-changes that may result from parasitic attack on plants, remarkable though they may sometimes be, resemble in many respects those that have been described above, allowance being made for the fact that the stimulus may be more localised and may continue for a longer time than that caused, for instance, by wounding. It seems very doubtful whether there is any essential difference between the modifications of tissues and organs found in galls due to parasites and those that may occur in wound calluses or the various other types of reaction to wound injury or physiological causes. The tissue and organ changes due to virus

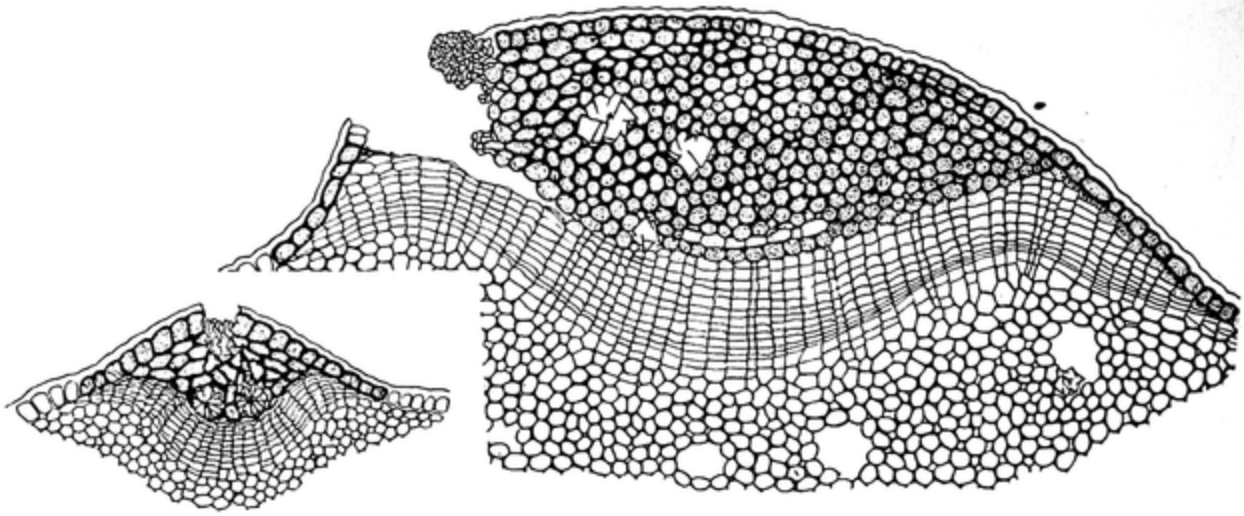


FIG. 138.—Reaction of almond twigs to infection by *Clasterosporium carpophilum*, showing small and large scabs, and commencement of tearing away in larger scab (× 87) (after Samuel, *Ann. Bot.*)

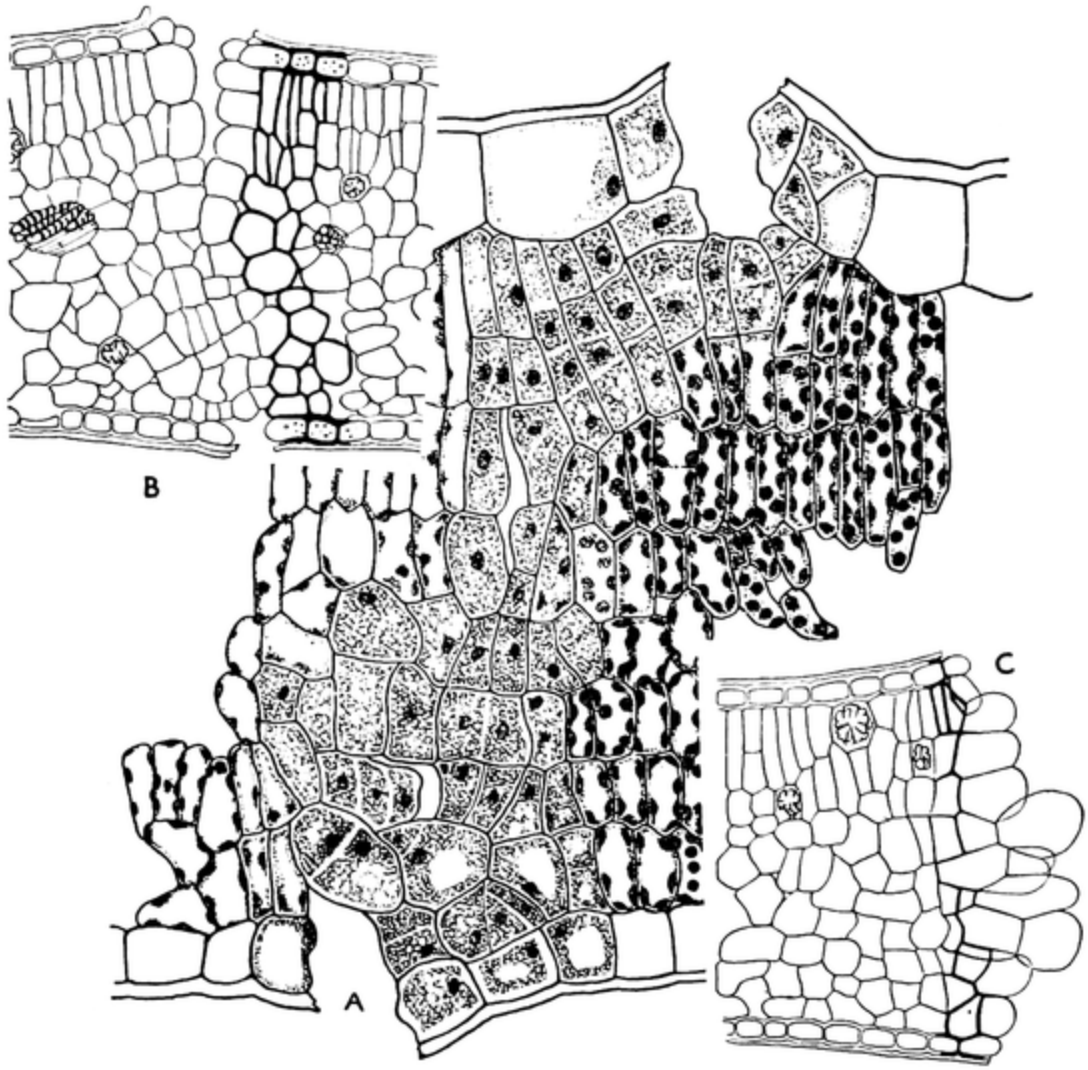


FIG. 139.—' Shot-hole ' effect caused by *Clasterosporium carpophilum*. *A*, cross-section of almond leaf showing formation of the abscis line ; infected tissue is on the right ; the occluded zone, with lignified zone towards the injured tissue. *B*, *C*, two stages in the process of abscission in cherry-laurel leaves ; lignification shown by heavy outlines ; the lignified zone drops away with the injured tissue when abscission is complete (× 190) (after Samuel, *Ann. Bot.*)

diseases are separately dealt with in Chapter VIII.

Hypoplastic modifications are seen in the sterility caused by some parasites, in the small size of the leaves in trees attacked by certain virus and other diseases (spike disease of sandalwood) and in inhibition of tissue differentiation as a result of parasitic attack. An interesting example of the latter is the prevention of the formation of an abscis layer in cherry leaves attacked by *Gnomonia erythrostoma*, with the result that they do not fall in the autumn but remain hanging on the tree and serve to infect the new leaves in the following spring (see Part II, p. 768).

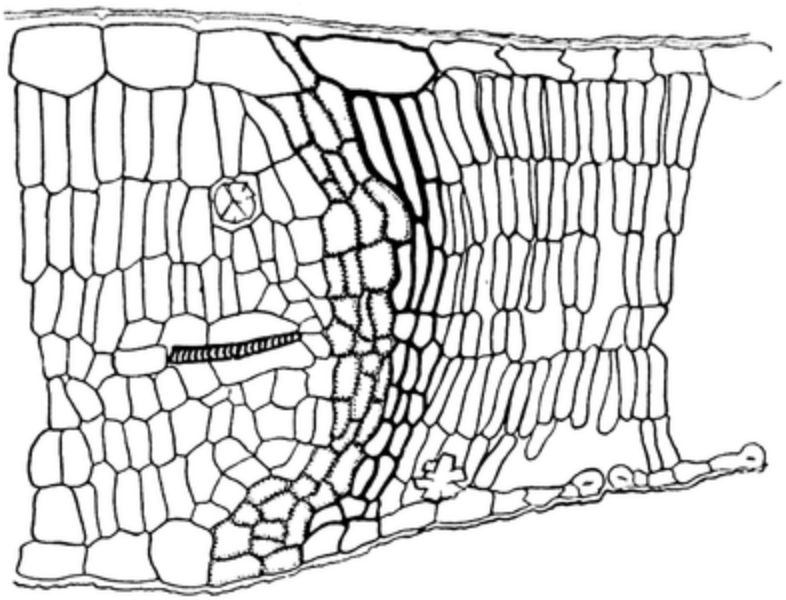


FIG. 140.—Section of almond leaf, illustrating the layer of wound cork isolating the infected tissue in a case where abscission did not occur ; suberised cells are dotted round the margins ; lignified cells are heavily outlined (× 250) (after Samuel, *Ann. Bot.*)

Host-tissue reactions to parasitic attack by the formation of wound gum in woody tissues and wound cork in all living parts resemble those due to simple wounding in their main characteristics (Fig. 138). Various examples of them are given in Chapters III and IV. The band of reactionary cork formed around a leaf infection can sometimes be seen in thin leaves as a narrow dark circle on holding them up to the light. The cork may be effective in checking the parasite or may fail. A common result of its formation in leaves is the necrosis and dropping out of the surrounded area, producing what is known as a ' shot-hole ' effect (Figs. 139, 140, 395).



FIG. 141.—Galls involving the modification of organs. Tumours of maize smut caused by *Ustilago zeae*. An affected cob (after Butler)

It has been already mentioned that there is a tendency for the tissues of reaction to non-parasitic stimuli to consist at first of elements resembling one another, as, for instance, in calluses and intumescences, in the repaired epidermis, palisade and spongy paren-

chyma at the edge of a leaf wound, or in the thin-walled derivates of the cambium on the xylem side in the thickened shoots of kohlrabi prevented from flowering in the second year. This tendency is still more marked in many galls and has led to the use of the term 'gall' or 'tumour' tissue to characterise the masses of soft rounded cells, often with few or no intercellular spaces, that may develop as a result of the presence of a parasite.

Galls are swellings on plant organs due to a stimulating or irritant action of parasites such as fungi, bacteria or insects on the tissues. In a few cases (stripe disease of narcissus, Fiji disease of sugar cane) they are caused by viruses. They may vary from tiny swellings involving a few cells of the epidermis as in the early stages of wart disease of potato (*Synchytrium endobioticum* (Figs. 249, 250)) to the soft rounded tumours several inches across of maize smut (Fig. 141), or the large woody masses formed sometimes in crown gall (Figs. 142, 375) or in certain rusts on *Acacia*, (*Haplophragmium*, *Uromycladium*). The term gall is sometimes extended to include organic outgrowths or proliferations, such as the leafy organs formed in cereal ears attacked by *Sclerospora* and also to cover cases of uniform enlargement of an organ, as in flowers of some *Cruciferae* infected by *Cystopus candidus*, or the 'bladder plums' produced by *Taphrina pruni*.

Galls due to Tissue Swellings : 'Histoid' Galls

Some of the simplest galls are caused by species of *Synchytrium*. The fungus may be confined to a single epidermal cell which generally becomes much enlarged. Neighbouring cells also react, usually hyperplastically, and the host cell becomes surrounded by a sheath which, as in *S. taraxaci* on the dandelion, may be derived from both epidermal and subepidermal cells. The bordering cells in this little gall elongate tangentially to the host cell and divide by walls at right angles to the latter. A small-celled sheath of two or three layers in depth is thus formed around the enlarged cell containing the fungus. No intercellular spaces occur in the sheath nor is there any detectable difference between the derivatives of the epidermis and mesophyll that constitute it; there appears to be also no tendency to form cork, perhaps because of the short duration of active stimulation. Another very simple epidermal gall is caused on poplar leaves by *Taphrina populina*, syn. *T. aurea*, the mycelium of which is situated between the cuticle and epidermis and for a short distance down between the cells of the latter. These cells elongate to two or three times the normal length and may divide once or twice, the result of their uneven growth in the developing leaf being the formation of a small bulge on one side of the leaf and a corresponding depression on the other.

Another leaf gall of a simple type is that formed on apple leaves by the aecidial stage of *Gymnosporangium juniperi-virginianae* in America. The affected part of the leaf is swollen to two or three times its normal thickness by a great increase in height of the spongy mesophyll cells, the palisade being little altered. The elongated cells divide by transverse or oblique walls and the intercellular spaces are lost. In the thickened leaves produced on the shepherd's purse (*Capsella bursa-pastoris*) by infection with *Cystopus candidus*, the mesophyll is composed wholly of large round cells, distinction between palisade and spongy cells being

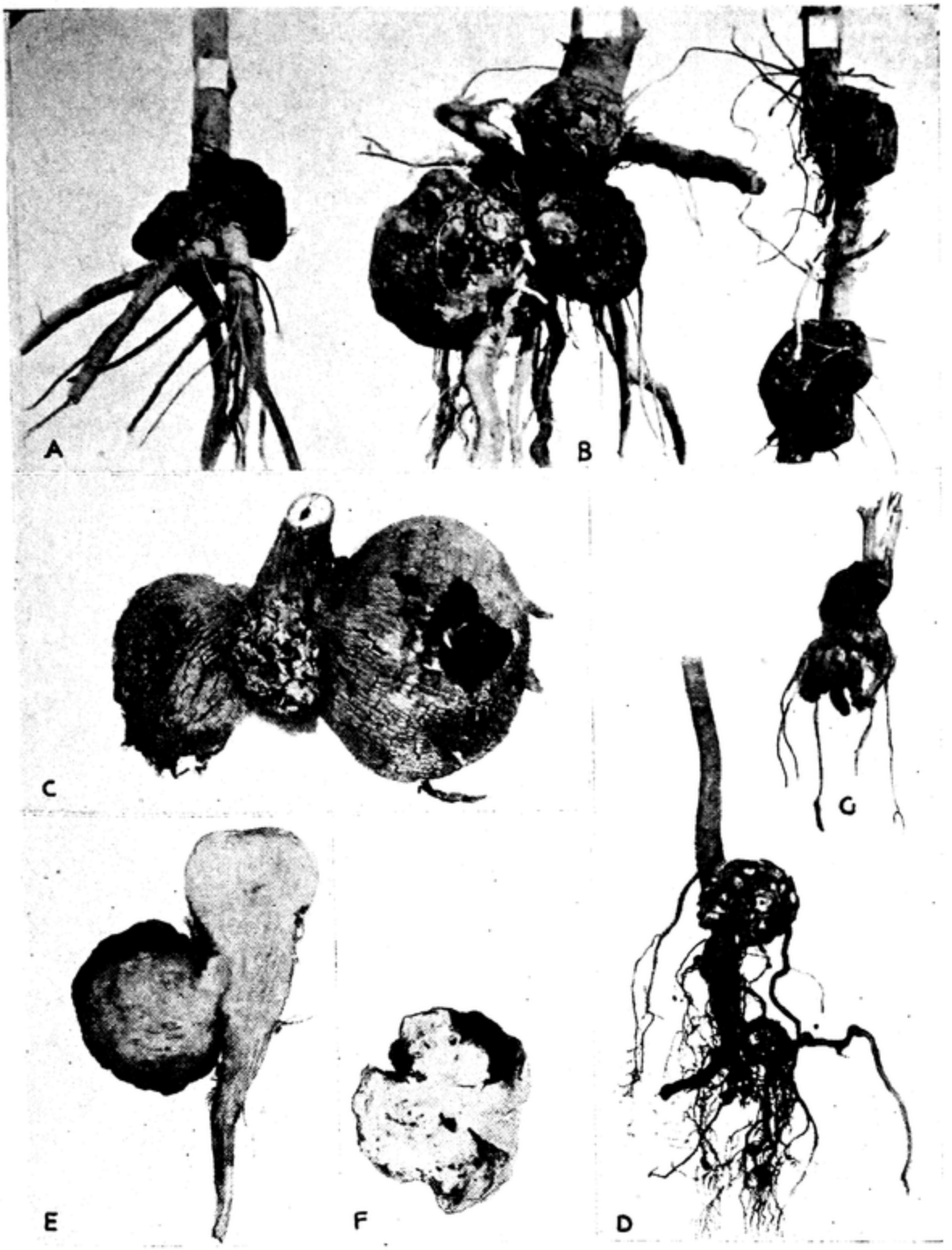


FIG. 142.—Galls and gall formation. Crown gall (*Bacterium tumefaciens*) on various hosts. *A*, on roots of one-year-old budded peach tree. *B*, on one-year-old cherry (budded tree) on Mazzard roots (photos of *A* and *B* by Bur. Plant Indus., U.S. Dept. Agric. By permission of E. A. Siegler). *C*, on tubers of dahlia (photo by Dennis). *D*, on apple (photo by Wormald). *E*, on beet. *F*, cross-section of same (photos by Scott Wylie). *G*, club root on swede (photo by Scott Wylie)

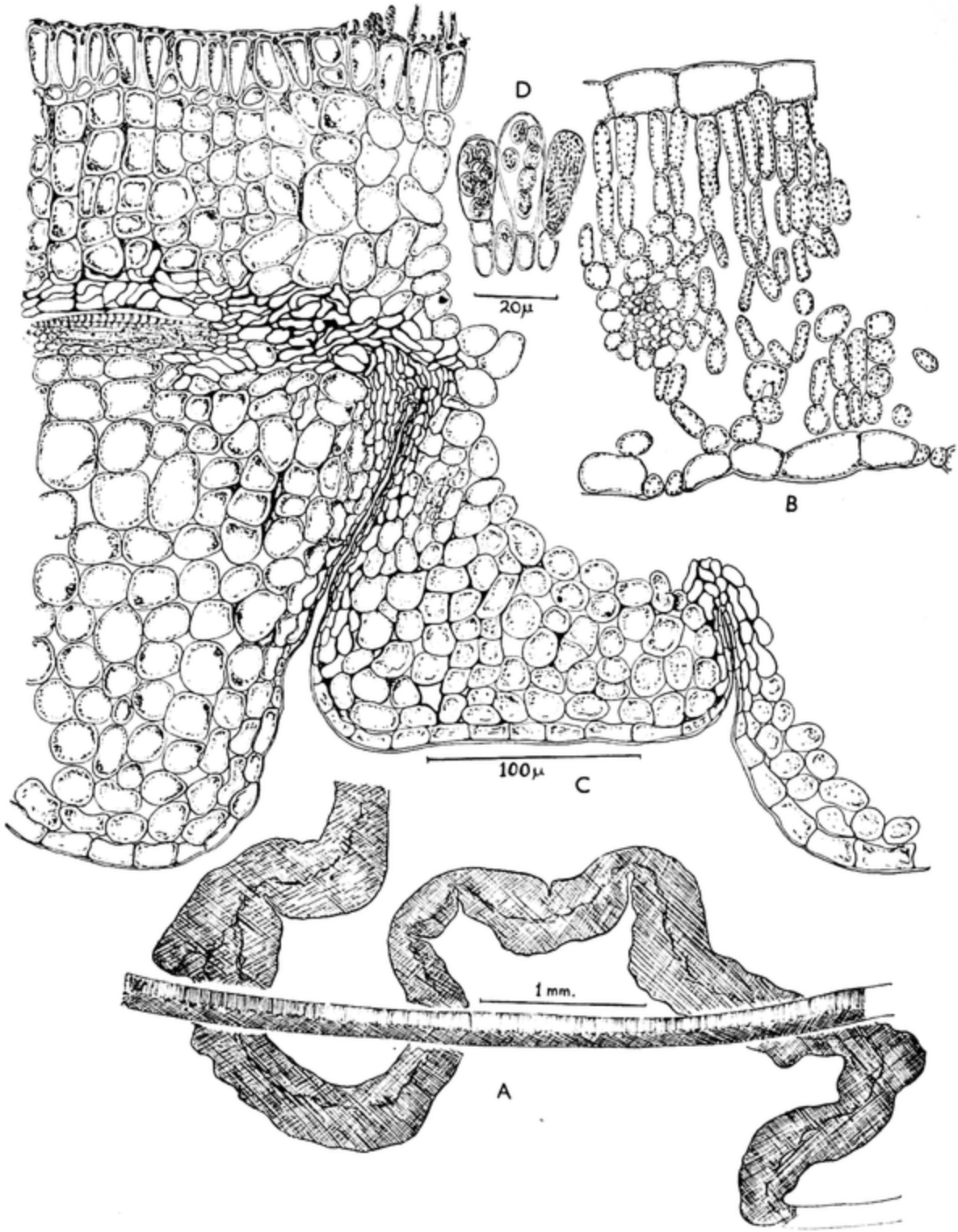


FIG. 143.—Leaf galls. Peach-leaf curl (*Taphrina deformans*). *A*, transverse section of swollen part of infected leaf, much distorted, compared with section of a healthy leaf. *B*, section of a healthy leaf. *C*, section of the swollen leaf showing the much thickened distorted lamina in which distinction between palisade and spongy tissues is lost; cell division has occurred in the palisade cells which have also lost their normal shape, becoming almost isodiametric in the outer layers; note considerable increase of cells in spongy tissue, with reduction of intercellular spaces (drawn to same scale as B). *D*, the sub-cuticular asci, two containing ascospores, and the other budded 'conidia' or secondary spores

lost. *Taphrina deformans* (Fig. 143), also thickens the peach leaves that it attacks, and causes the palisade cells to multiply and lose their normal shape, becoming almost isodiametric in the outer layer, where they are hard to distinguish from epidermal cells. *Exobasidium oxycocci* makes all the mesophyll into a uniform type of parenchyma without intercellular spaces in *Vaccinium macrocarpon*, one of the cranberries, but in the thickening caused by *E. vaccinii* in *V. vitis-idaea* leaves, the palisade is little altered and the swelling is due to the development of roundish or polygonal cells with no intercellular spaces in the spongy parenchyma. A simple stem gall is the common one on goutweed or bishopweed (*Aegopodium podagrariae*) caused by *Protomyces macrosporus*, where there is a hyperplasy of the outer cortical cells, restricted to those in the neighbourhood of the intercellular hyphae. The easiest way to locate the hyphae of this fungus in the early stages of infection is to look for a fully formed cortical cell in process of division.

Somewhat more complex are the small corky swellings that constitute the disease known as scab on the leaves and twigs of citrus plants. The parasite, a conidial stage of the fungus *Elsinoe fawcetti*, causes in attacks on the under surface of the leaf a necrosis of a few superficial cells, and provokes a hyperplasy of the underlying spongy parenchyma. The new cell walls are roughly parallel to the necrosed surface, and the reaction may extend through the leaf and cause some of the palisade cells to divide. Cells near the lesion elongate considerably and divide by several cross walls. A band of the new cells thus formed becomes a cork cambium. This extends up to the epidermis all round the lesion, and cuts off cork on the side next the latter and a little phelloderm towards the sound tissues. All the primary cell walls of the hyperplastic area are thickened, and intercellular spaces are much reduced.

A good example of a gall restricted to the vascular region is that produced by *Sorosphaera veronicae* on *Veronica chamaedrys*, the germander speedwell [2]. The swelling starts in the procambial region of the shoot, from the point behind the apical meristem where the xylem is just beginning to form, and extends backwards without involving the cortex or pith, and sometimes outwards along a young leaf bundle. Spiral vessels can be found throughout its length but do not occupy any fixed position in relation to the mass of gall tissue, being sometimes on its inside towards the pith, sometimes on its outside towards the cortex, sometimes in the middle. This shows that the gall tissue is formed not only from the cambium, but from cambial derivatives lying around the protoxylem vessels. It consists of more or less polygonal cells of a very uniform type, the only other elements found in the swollen part being the few xylem elements that were already differentiated before the parasite reached the area, and some scattered groups of elongated cells. The parasite, an intracellular one, does not appear to occur in all the tumour cells, but the hypertrophy (which is sometimes marked) and hyperplasy are general throughout the region involved. The cortex and pith usually undergo no histological modifications, though there are indications that, in later stages, the parasite may reach the inner cortex by passage from within through the endodermal layer.

In the finger-and-toe or club-root disease of turnips (Fig. 144), cabbage and other crucifers, the parasite *Plasmodiophora brassicae* (like the last, one of the Plasmodiophorales) is again mainly confined to the central cylinder. In

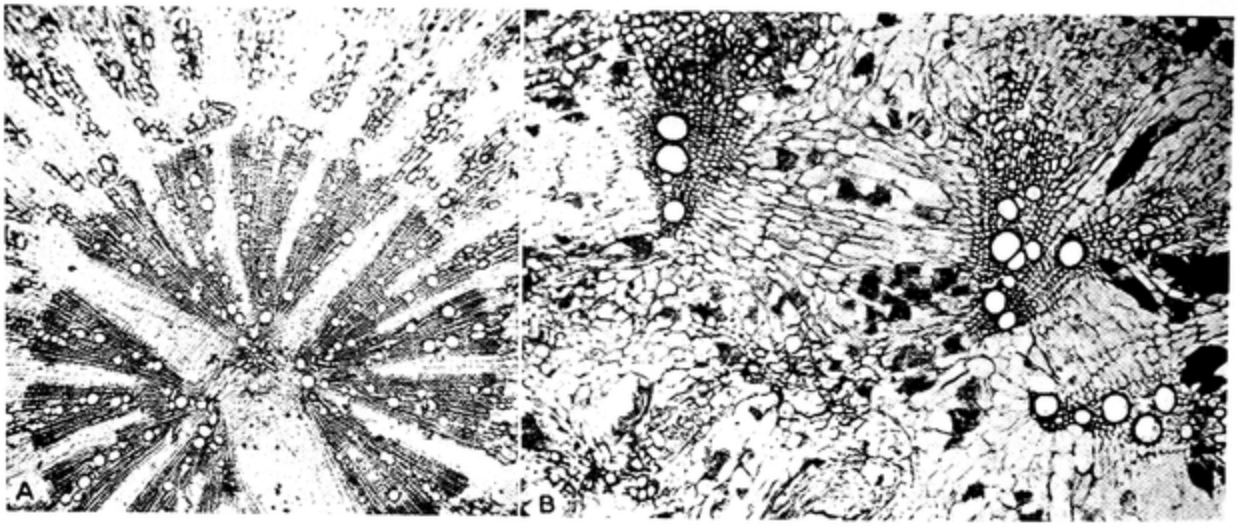


FIG. 144.—Galls involving the vascular region. Club root of cabbage. Part of central cylinder of the stem, inoculated with *Plasmodiophora brassicae*. *A*, enlargement of medullary rays. *B*, showing a later stage of disruption in the wood following proliferation in the rays (after Kunkel, *J. Agric Res.*)

inoculations on cabbage stems the fungus reaches the cambium fairly soon and induces a marked hyperplasy in the cells in this region. The elements cut off on the xylem side remain undifferentiated and again rapidly divide. In older stems the medullary rays are chiefly infected and show great hypertrophy and hyperplasy. Except at the point of original invasion, the phloem and secondary cortex only become infected outwards from the cambium. In roots the bulk of the hyperplasy is in the cambium and the phloem initials adjoining it ; the new cells in the position of the phloem are thin-walled and accumulate starch. The increase in size of the medullary rays may divide the vascular ring into widely separated bundles which ultimately may become much distorted by the uncontrolled growth in the rays. New tracheidal tissue develops in the rays. The changes that occur in the vascular region in this disease show a certain similarity to those already mentioned as occurring in allied plants as a result of physiological processes consequent on the prevention of flowering (Figs. 147, 277, 278).

In various other types of gall, insect as well as fungal, the tendency already mentioned for the cambium, when stimulated to abnormal activity, to cut off non-lignified parenchyma without fibres and with fewer and thinner-walled tracheids on the xylem side, and secondary parenchymatous phloem outside is strongly marked. In some of those that cause the medullary rays to enlarge, so as to separate the vascular ring into isolated bundles, these tend to become bordered by a cambium forming in the ray cells and in the region of the protoxylem, very much as was found in the thickening due to prevention of flowering in kohlrabi. The underlying causes of these processes are not known, but the similarities are sufficient to suggest that whether the stimulus is a fungus or an insect or purely ' physiological ', the response follows along certain broadly defined lines.

In some fungal and bacterial galls all or nearly all the tissues may become involved in the swelling. The thickened axial shoots that are the most prominent feature of the destructive witches' broom disease of cacao in the West Indies and South America, due to *Marasmius perniciosus* (Figs. 99, 145), are good examples

of this. As many as a thousand of these may be found on a tree. The swelling may involve every tissue from epidermis to pith, sometimes almost equally, each being two or three times the normal thickness, sometimes mainly in the cortex or the xylem. Most of the thickening in the xylem seems to be from cambial activity, but in the other tissues it is mainly due to division of parenchyma *in situ*. The pericyclic region and deeper tissues may proliferate to such a degree that the cortex is crushed, or may become so parenchymatous that it is almost impossible to define the inner limits of the cortex, though scattered groups of pericyclic fibres can still be distinguished. All the parenchyma cells, except those nearest the epidermis, assume the same shape, elongated radially and with thin walls. The xylem vessels are broader and thinner than usual and the part of the xylem near the cambium is largely parenchymatous. The fibres become at first septate, then reduced in numbers, and finally are not formed. As the thickened shoot grows in length, tissue differentiation is decreased. In the swollen leaves the distinction between palisade and spongy parenchyma is lost. The 'brooms' are short lived (some six weeks) and remain soft and fleshy, cork being rarely found in them.

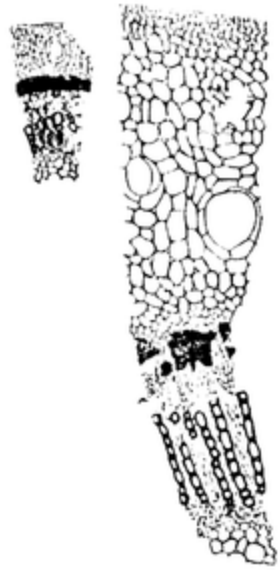


FIG. 145.—Comparison of normal and swollen midribs of leaves from cacao, to show the effect of *Marasmius perniciosus* (after Went. Butler, *Ann. App. Biol.*)

The huge *Uromycladium* galls formed on species of *Acacia* in Australasia (that caused by *U. notabile* may be over a foot in diameter) start usually by a proliferation in the phloem, infection beginning generally before cork has developed. The outer tissues are deformed and ruptured by pressure from within, due both to expansion of the phloem and pericycle and to the production of wound wood composed of unlignified rows of tracheids, with a few wide vessels and an excess of parenchyma. The identity of the original cambium is lost at an early stage, and later it becomes impossible to distinguish clearly between phloem, cambium and outer xylem. Cork may form in the pericyclic region or the outer cells may continue to proliferate as hyperhydric tissue. Tracheids may develop in any of the tissues that take part in the formation of the gall.

No plant gall has been so completely studied as crown gall caused by *Bacterium tumefaciens* (Fig. 142 A-F), because of its intrinsic interest, its wide range of host plants, some of considerable economic importance, and (perhaps mainly) because of suggested analogies between it and cancerous growths in man and animals.

The organism which causes crown gall is one which is not ordinarily provided with enzymes capable of dissolving the polysaccharides of cell walls or starch, so that it does not form cavities or penetrate into the interior of the plant cells or induce soft rotting. On the other hand, it has marked powers of stimulating the cells in its immediate vicinity to divide and form hyperplastic masses of tumour tissue. When it is inoculated into the tissues of herbaceous stems that are still growing, it may occur in the intercellular spaces of considerable lengths of stem formed subsequently to the inoculation and it may also spread actively or passively in the fully grown internodes, along the xylem or where, as in the pith or cortex,

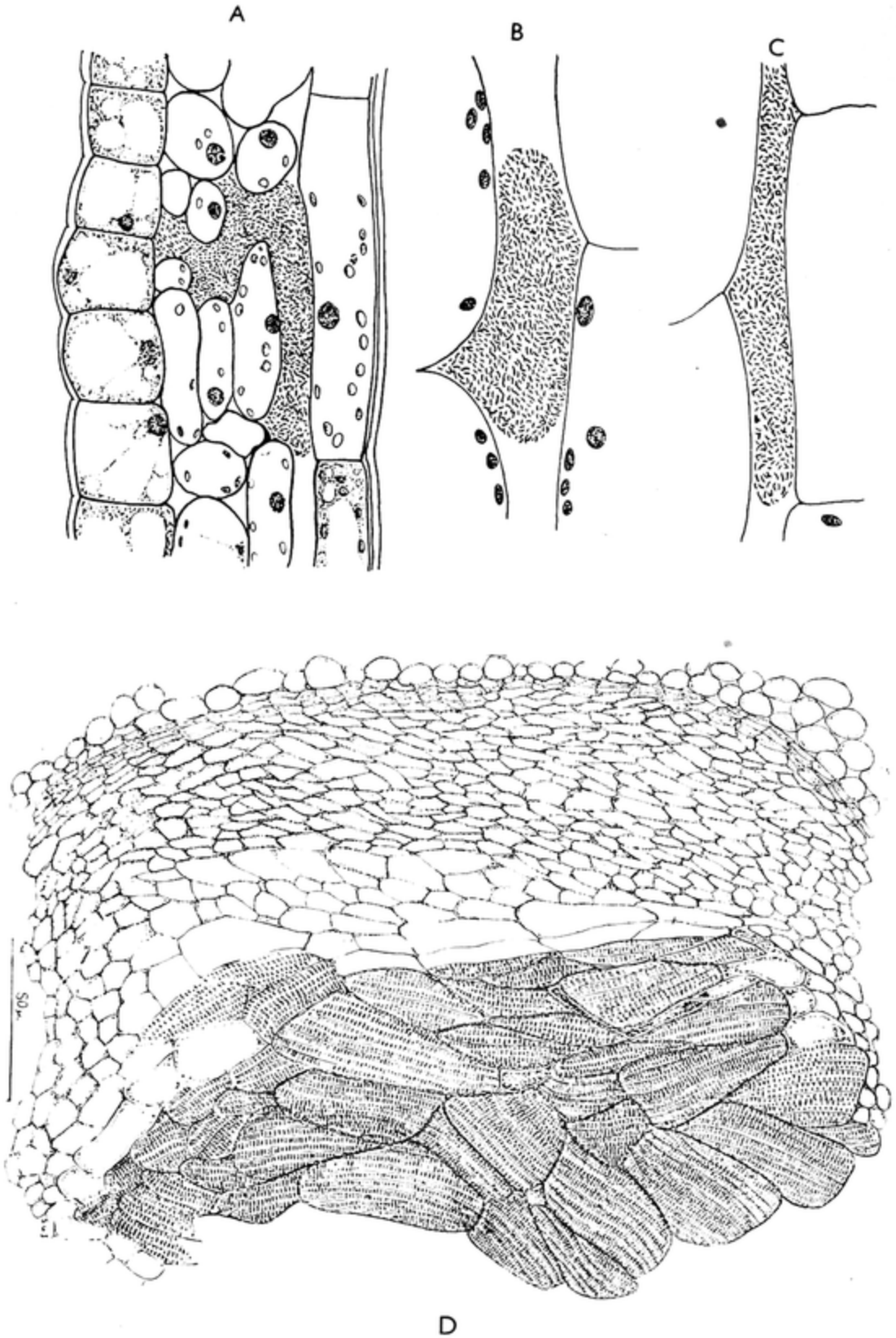


FIG. 146.—Crown gall (*Bacterium tumefaciens*). *A*, radial longitudinal section through outer portion of stem of tomato showing the relation of the bacterial zoogloeae to the intercellular spaces of the sub-epidermal tissues. *B*, a tangential section where the zoogloea has reached its present location by moving in a radial direction from an adjoining space, the zoogloea advancing in both directions. *C*, a longitudinal section through central portion of the pith of a young tomato stem (× 800) (after Hill, *Phytopath.*). *D*, a section of portion of a sub-aerial gall on the cane of red raspberry, showing the formation of tracheids from the gall parenchyma; note evidence of meristematic activity at periphery

intercellular spaces are in vertical communication with one another for some distance longitudinally. In these cases it may give rise to continuous strands of tumour tissue formed by the division of bordering cells, or it may only cause visible hyperplasy (' secondary tumours ') here and there along the course of its extension. It is not uncommon to find, following inoculations of this type towards the top of the stem, closed vascular rings, spheres or cylinders in the pith and cortex. When these are found in the pith the orientation of the tissues is frequently inverted, the phloem being inside and the xylem outside the cambium (Fig. 147). In the cortex and petiole the orientation is normal, and in the latter situation the stimulation from the parasite may lead to the formation of a closed concentric bundle by division of the ground cells of the petiole outside the leaf trace, just as occurs when a petiole is rooted as described above. The centre of the new formation is a mass of tumour tissue in which cells of the ground parenchyma not transformed into tumour cells may sometimes be seen. In some of these infected petioles the organism has been found to occur in the protoxylem, and the meristematic divisions in the ground tissue begin opposite this point.

Tumour strands in the outer cortex resulting from inoculations of *Bact. tumefaciens* in this region have been found to arise from extension of the bacteria along intercellular spaces running longitudinally in the stem or drawn out by rapid elongation of young parts, the cells bordering the infected spaces being stimulated to active division (Fig. 146). A secondary gall has been found to develop at some distance from the point of inoculation, and in this a closed vascular sphere may occur, having no vascular connection with the central cylinder of the stem. In some pith strands the cells bordering the first-formed tumour cells may show only hypertrophy but in others a vascular cylinder develops, with xylem outside. The vascular tissue may form around a core of tumour cells in a part only of these strands ; in strands in the pith and cortex it develops independently of the original cambium and usually at some distance from it. The inverse arrangement of the bundles thus formed in the pith resembles that found normally in many families of plants possessing medullary bundles. Sometimes also, both in normal pith and in the protoxylem of inoculated plants, only a little nest of phloem occurs without cambium or xylem, and it is interesting to note that a similar formation has been found in the middle part of the protoxylem parenchyma in kohlrabi plants prevented from flowering. The exciting cause of these inverted normal or induced formations appears to be unknown, though attempts have been made to explain them.

When crown gall results from inoculation into the deeper layers of a herbaceous stem the tumour tissue may appear to burst through the central cylinder and cortex to the outside. This appearance is due to a progressive hyperplasy of the ray and cortex cells, the outer layer of the latter being sometimes crushed by pressure from below. In such erupting galls it is easy to follow the conversion of the normal cells into gall tissue by multiple division. In the tomato, the gall cells thus formed become smaller and smaller, by division, for some weeks, then enlarge somewhat ; an average normal cell of the cortex may be nearly 5000 sq. μ in area, and this may fall by division to 166 μ after about a month and rise to over 400 μ in about two months.

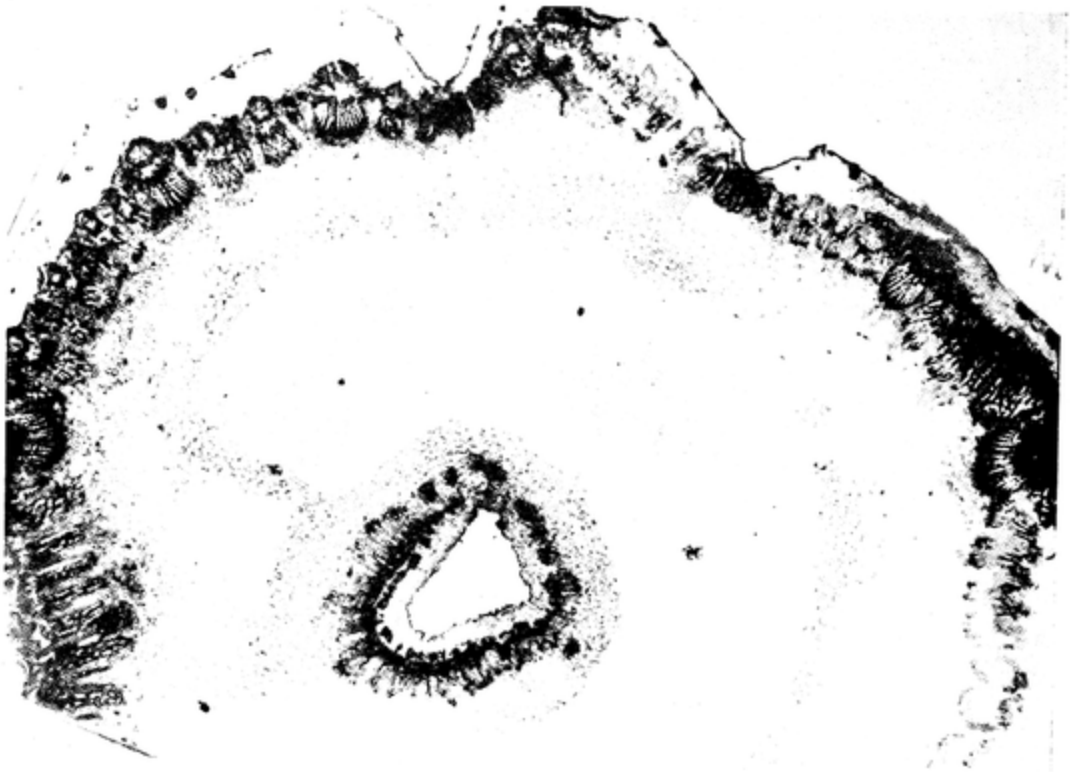


FIG. 147.—Cross-section sunflower stem inoculated with crown gall (*Bacterium tumefaciens*) by a needle wound in the young flower head, showing vascular ring, cortex, and epidermis formed around the wound, at the centre; in these pith bundles the orientation of the tissues is inverted, the phloem being inside and the xylem outside the cambium; a few glandular hairs line the cavity, these resembling the normal epidermal hairs (× *circa* 5) (after Smith, *Mem. Nat. Acad. Sci.*, 22, No. 4)

The tumour tissue from secondary tumours in herbaceous plants appears to be able to continue growth after removal from the infected plant. It still goes on growing when, after several ' generations ', bacteria cannot be isolated from the mass of tumour cells and the ground-up mass is incapable of infecting healthy plants. The apparently sterile tumour mass can be implanted by grafting into a healthy sunflower and will continue to grow into a tumour resembling a typical crown gall on the plant, reaching sometimes a diameter of 1 cm. after seven weeks. Work at East Malling has shown that similar tumours can be produced by the growth-promoting substance indolebutyric acid, while in America it was found that indoleacetic acid causes both primary and secondary tumours, the latter at some distance from the treated site. Though it has been found that *B. tumefaciens* can form indoleacetic acid in media containing tryptophane or other nitrogen compounds of high molecular weight, and the accumulated evidence indicates that a diffusible substance is concerned in the development of the tumours, it is not yet clear what the substance is or whether it is a product of the host cells or of the metabolism of the bacteria, or even if it is one of the recognised growth-promoting substances, such as indoleacetic acid, at all.

In woody stems such as those of the raspberry or blackberry, in which crown gall is not uncommon, the galls may appear as multiple outgrowths. In these infections the swelling may at first be confined to the region of the pericycle outside the fibre bundles (Figs. 148, 149). There may be no nests of

tumour tissue, as in the herbaceous stem galls, the swelling consisting of thin-walled radially elongated parenchyma in rows and with scanty contents. The multiplying cells lie below the cork, in the pericycle, which form in layers (the so-called polyderm) in the neighbourhood of the endodermal region. At this stage the new tissue may be regarded as a parenchymatous derivation of the actively dividing pericycle, but the reaction soon extends down between the fibre bundles and involves the tissues between them and the outer part of the phloem. The growth in this position bends up the pericyclic fibres and separates groups of them widely from one another. Here and there, usually not over the whole arc underlying the gall, the cambium is stimulated to form wound wood, which is sharply differentiated from the normal xylem by a scarcity of fibres, the thinner walls and wider lumina of the tracheids, and the absence of large vessels. In the tumour mass outside the cambium single tracheids or tracheid groups are formed by differentiation in individual tumour cells, just

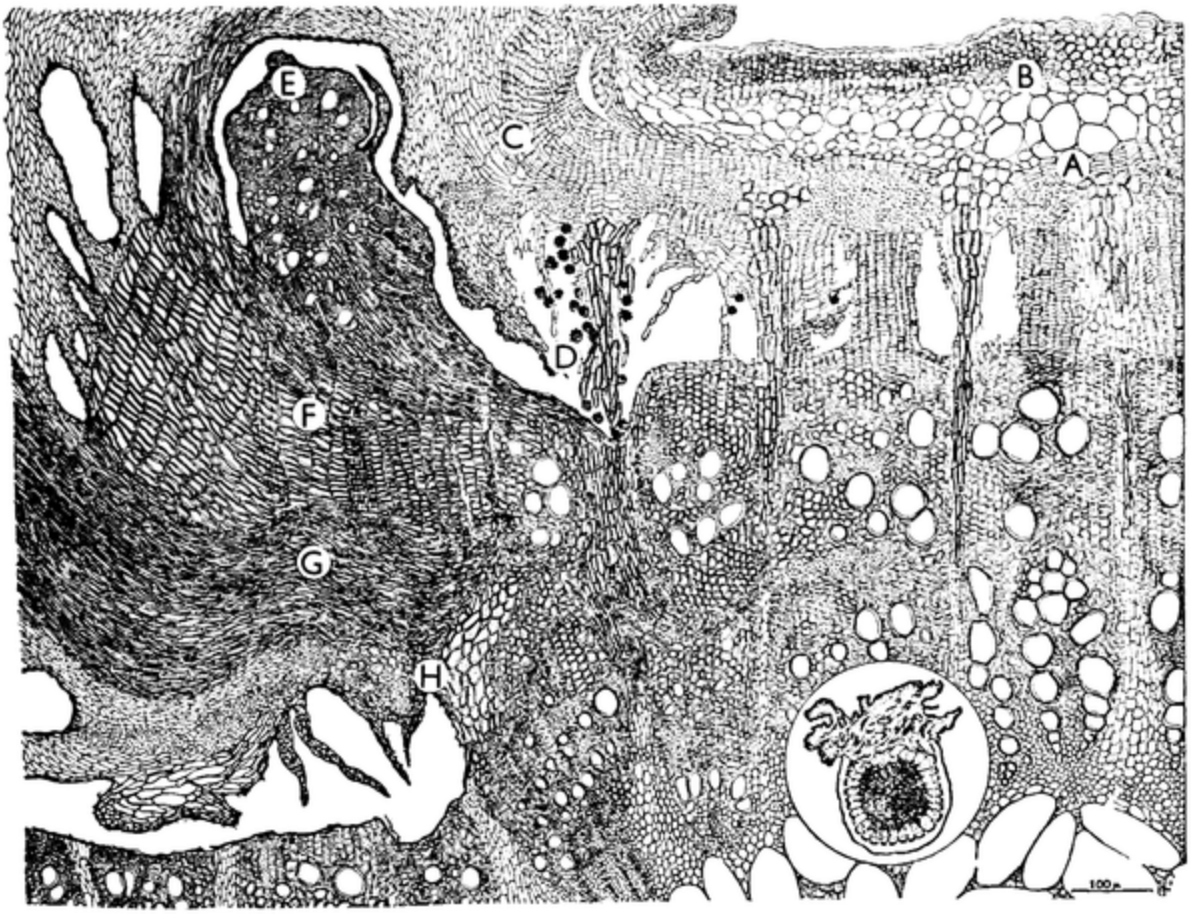


FIG. 148.—Transverse section of stem of Himalaya giant blackberry affected with crown gall, showing general plan of host and gall tissues. Inset, in circle, section of cane showing entire gall in relation to the axis. The large-scale drawing is taken from right-hand portion, at junction of gall and stem. *A*, the outer layers of the pericycle, becoming meristematic. *B*, the cortical host tissues. *C*, the outer pericyclic cambium fanning out into the gall to form secondary parenchyma. *D*, the thick-walled tracheids of medullary ray, added to from above by the pericyclic cambium; note the torn phloem and cavities, and crystals; the deep dent in the surface of the axial cylinder opposite the medullary ray. *E*, displaced vascular bundle. *F*, *G*, tracheids and fibres, developed probably from medullary ray and cambium, sweeping out into the base of the gall in Fig. 149. *H*, intrusive parenchyma (after Jones, *Phytopath.*)

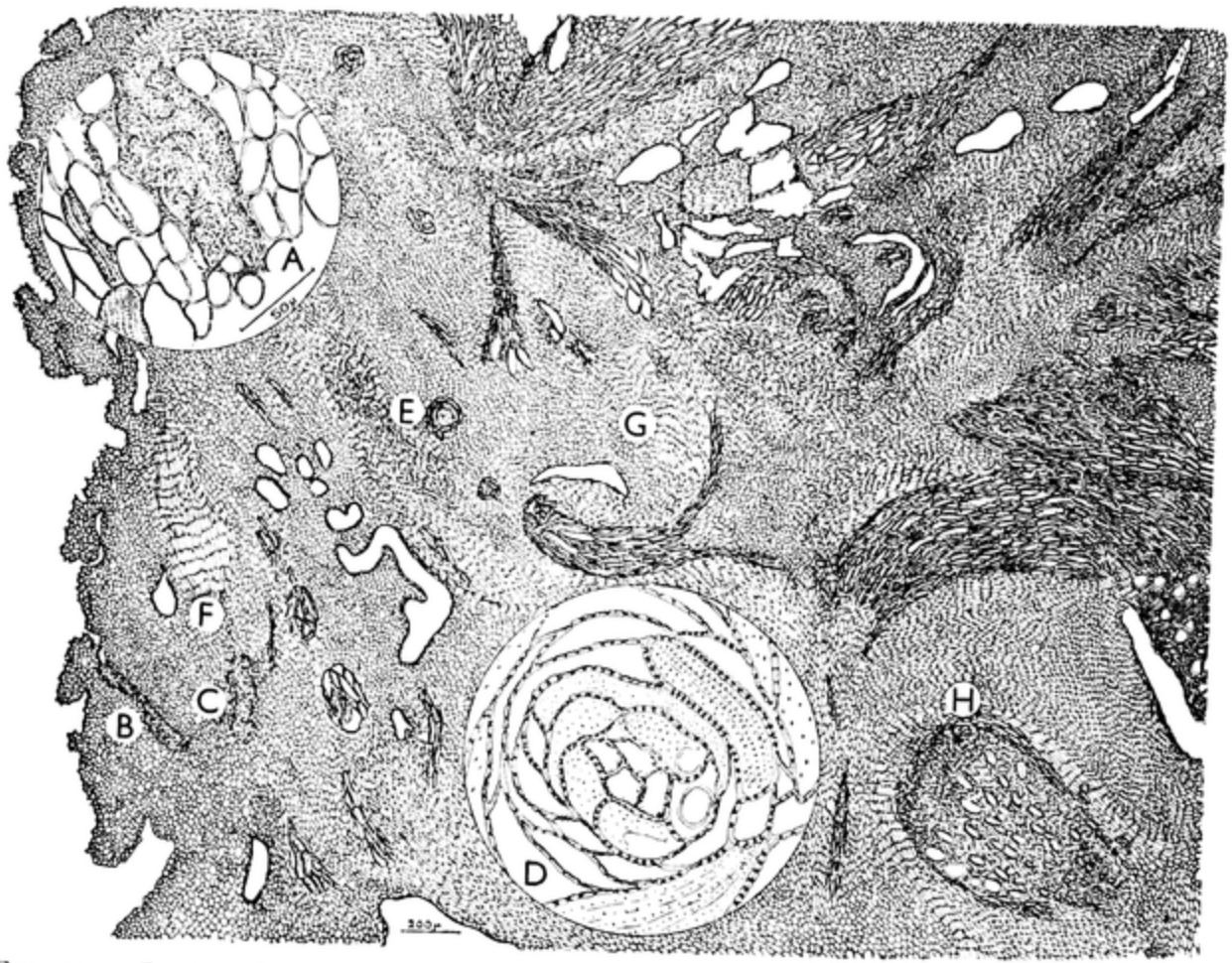


FIG. 149.—Section of a portion of a gall showing general structure (see Fig. 148). *A*, the bacteria in a pocket at surface of gall, as at *B* and *C*. At *C*, *E*, *F*, *G*, *H*, and other places, note portions of meristem or cambial arcs forming secondary parenchyma. Note the variously shaped groups of lignified tracheids, some spherical as at *E*, others V- or Y-shaped. *D*, details of a spherical group of tracheids formed around a small group of parenchyma. Just below *H* is a leaf trace pushed out into the gall and subsequently invested by a cambial meristem which has formed a few lignified tracheids towards the leaf trace, together with secondary parenchyma towards the gall. Note the crushing and tearing of gall parenchyma, with cavities, due to the contending activities of the numerous cambial arcs (after Jones, *Phytopath.*)

as occurs in an ordinary callus outgrowth and, as in callus, the tracheid islands may become bordered by a meristem producing further tracheids on one side and phloem or gall parenchyma on the other. The gall often becomes highly vascularised in this manner and some of the strands may reach the original central cylinder. The surface of the gall is usually a loose mass of brown collapsed cells with weakly lignified walls, resembling the hyperhydric tissue frequently formed on the surface of a callus. There is, indeed, a fairly close histological analogy between crown gall and wound callus of the type found causing knots on apple grafts.

There is little reason for the supposed analogies between this plant gall and the malignant growths that form animal cancers. As will have been seen, the processes involved in its formation are not essentially different from those found in other plant galls. Similarly, the leafy outgrowths that sometimes occur on the galls, and that have been termed embryomata from a suggested resemblance to one type of cancer, are no doubt to be likened to callus shoots.

Organoid Galls

There is another type of structural modification sometimes produced in plants as a result of the presence of a parasite in the tissues to which, though the changes are morphological rather than histological, the term ' gall ' is often applied. These ' organoid ' galls, as distinct from the ' histoid ' type already discussed, result especially from systemic infections with obligate parasites of the kind that do not kill the invaded plant parts at an early stage of parasitism. They are characterised by changes in the morphological plan of the shoots or other organs affected, or in the rhythm of their development.

Marked changes in symmetry and habit may be found in plants affected by some rusts and mildews. A good example is the change from the normal short unbranched stem, with radical leaves, of *Launea asplenifolia*, a common Indian weed, to an elongated much branched axis, with cauline leaves, when infected with *Puccinia butleri*, a systemic rust. Stem elongation is caused by *Ustilago hypodytis* on *Elymus arenarius* and *Sclerospora sacchari* (oospore stage) on sugar cane [8], and the number of internodes and leaves is increased by various rusts, e.g. *Uromyces pisi* on *Euphorbia cyparissias* and by *Gibberella fujikuroi* in the ' bakanae ' (elongating) disease of rice in Japan, where it has been traced to the production of a crystalline active principle gibberellin ; shortening of internodes (up to 45 per cent.) occurs in rye attacked by *Urocystis occulta*.

The symmetry of the leaves is altered in some diseases. They may be changed from simple to irregularly lobed in *Berberis buxifolia* attacked by *Aecidium jacobsthalii* in South America. In the ' reversion ' disease of black currants (Fig. 389) (Part II, p. 830) one of the most useful aids to diagnosis is the change in the venation and, especially, the lobation of the leaves of affected plants. The reverted leaf may have only three main (palmate) veins, diverging from the top of the petiole to the extremity of three main leaf lobes, instead of the usual five of each. Lateral sub-veins from the middle main vein (the midrib) are reduced to about half the normal number, but they serve to supply nearly all the teeth on the leaf margin, whereas the 10 to 16 sub-veins of the normal leaf do not supply more than about half the teeth of the apical lobe. The reverted leaf, best seen on wall-grown shoots in May and June, is smaller, narrower, flatter at the base, coarser and of a deeper green than normal. An extreme example of the opposite tendency is to be found, as the name implies, in the ' fern leaf ' type of tomato mosaic disease (Fig. 172), while some forms of tobacco virus infection can cause a twisting of the leaf so that the under surface faces upwards. Another remarkable effect of virus infection on leaves is the formation of ' enations ', green cup-shaped or blade-shaped protusions, generally on the under side of the leaf. Distortions due to virus diseases, however, will be more fully discussed in Chapter VIII. The very remarkable leafy outgrowths from the leaves of bracken caused by the fungus *Taphrina laurencia* in Ceylon are probably due to processes similar to those causing enations.

Perhaps the most profound modifications of the type under discussion are caused in inflorescences and single flowers by the action of certain fungi and viruses. One of the commonest of these changes is the inducement of ' phyllody ' or the

transformation of flower parts into leafy organs. This is found in several virus diseases of tomato, sandalwood and other plants, in the flower of the Japanese plum *Prunus mume*, infected by *Caeoma makinoi*, and in certain crucifers attacked by *Cystopus candidus*. Single flowers may be doubled by the development of additional petals (*Viola sylvestris* infected by *Puccinia violae*) or their symmetry may be changed from the regular (actinomorphic) to the irregular (zygomorphic) plan and vice versa; in *Matricaria inodora* attacked by *Peronospora radii* ligulate flowers may be found in the centre of the disc and tubular flowers at the margin). The rudimentary stamens in pistillate flowers of *Lychnis dioica* may become fully developed when attacked by *Ustilago violacea*, except that the pollen is replaced by spores, while in the contrary direction the maize ' tassel ' (male inflorescence) when infected by *U. zeae* may bear female or bisexual flowers, or even normal grain at its base. Some rusts prevent flowering of the host when there is a perennial mycelium extending from the root-stock to the growing point but permit normal flowering when the infection is local [6].

The effects produced on the inflorescence of certain crucifers by *Cystopus candidus* may be very intense (Part II, p. 635). The flower stalk and axis of the inflorescence may be enormously thickened (up to 12 to 15 times the normal diameter), while the floral parts are wholly or in part fleshy, green or violet and persist instead of falling early. The petals may resemble sepals and the stamens become leaf-like or occasionally like carpels. The latter may be open instead of united into an ovary, while the ovules and pollen grains may be atrophied, thus causing sterility; sometimes the pistil may be much swollen into a conical thick-walled sac containing sterile ovules while the anthers may have supplementary pollen sacs or become like carpels by the development of stigma-like structures at the tip and rudimentary ovules along the margin. Occasionally the flowers revert to the primitive spiral insertion of their parts. Chlorophyll and starch may be copious even in the petals and stamens; hairs may develop on the anthers, and stomata in the inner epidermis of the enlarged ovary. In this case the tendency to a lessening of differentiation in the tissues of the swollen parts, which is marked, is carried further to a tendency to reduce the differentiation of organs.

One of the most striking cases of virescence of the inflorescence accompanied by leafy modifications of the floral parts is the ' green ear ' disease of millets in India, Africa, Japan, the United States, and elsewhere, caused by *Sclerospora graminicola*. As the name suggests, the chief symptom of the disease in the commonly affected bullrush or pearl millet, *Pennisetum typhoideum*, is the transformation of the solid spicate ear wholly or in part into a loose green head, composed of a mass of small twisted leaves. This is due to the replacement of the upper part of the axis of each floret, which normally forms the grain, by a short leafy shoot. The bristles below the glumes are enlarged and contorted and the fertile glumes enlarged and sometimes turned green. The stamens are often converted into minute leaf-like organs with a distinct division between blade and sheath. When not so transformed, they may occur as brown pointed bodies with no trace of the anthers, or if the anther is formed, its size relative to that of the filament may be much altered and pollen may be absent. The pistil often undergoes median proliferation, being replaced by a small leafy shoot, but sometimes

by a minute branched axis with undeveloped buds, or by a simple, hollow, horn-like growth, which may be a leaf united along its edges. Sometimes two spikelets arise from the pedicel instead of the usual one, and the number of florets on a spikelet in infected ears may be increased from two to three or four. On the other hand, oats infected by *Sclerospora macrospora* in Mississippi bore only a single seed-like structure in the glumes instead of the normal two close together. The median floral proliferation, which is the main characteristic of the 'green ear' cereal diseases, occurs also in certain virus diseases.

A remarkable instance of structural modification is caused by an organism first isolated in the United States from fasciated sweet peas. Later, this organism, *Bacterium* (or *Corynebacterium*) *fascians*, was found to be parasitic in a number of garden and other plants in England and to cause leafy and fasciated outgrowths from the buds at the crown of the root-stock and at the stem nodes, when inoculated in these sites. In some plants the leafy shoots from the base are so numerous as to appear like moss. Bud inoculations on seedlings (e.g. French bean) cause galls at the growing point and in leaf axils, with a complete check of normal growth; in other plants fasciated or cauliflower-like shoots are produced. Proliferation is confined to the bud tissues, inoculations elsewhere on leaves, stems and roots producing no effect. Amongst the 25 genera from which *B. fascians* has been isolated are peas, beans, strawberries, chrysanthemums, petunias, and asparagus. In strawberries a high proportion of inoculated seedlings of the St.-Jean variety developed leafy and floral abnormalities resembling those caused by cauliflower disease, but the inoculation of 'runner' plants gave inconclusive results.

The term witches' broom is applied by the country people in some parts of Europe to irregular tufts of crowded twigs found in trees and shrubs. They have, as indeed have several of the 'organoid' type of galls above-mentioned (including those in the last paragraph), some of the characters of 'histoid' galls, for the tissues that take part in their formation are often swollen. Their chief character, however, is the production of short twigs in irregular groups, often all turned upwards and with small leaves. When due to fungi, rusts and species of *Exoascaceae* are mainly concerned; the fungus passes into the new shoots produced each year, and often fructifies on them. When spore production is finished, the leaves and sometimes the younger twigs die off. Well-known examples are the brooms produced by *Taphrina cerasi* on cherries and those on silver fir (*Abies pectinata*) due to *Peridermium elatinum*. Mites and insects sometimes cause similar growths, and some brooms on pine and spruce are not caused by parasites, but appear to be due to an innate heritable tendency of the tree.

In galls, whether 'histoid' or 'organoid', with rare exceptions, there is nothing new in the tissues or organs of reaction, but merely a disorganisation or an intensification or inhibition of normal processes of tissue and organ formation and differentiation. The parasite can do no more than call out or suppress powers which the cells and tissues already possess, powers which plants can ordinarily make use of in dealing with such accidents as wounding or in meeting extremes of environmental or nutritional or growth modifying conditions. Indeed, most of the modifications in tissues and organs caused by parasites can be paralleled by those brought about by physiological processes without the intervention of a

parasite, or by examples selected from the extensive literature of plant teratology. In the ' quercina ' virus disease of the thorn apple *Datura stramonium* the spines on the capsules are eliminated, the stigmatic surface of the pistil is elongated, the tubular corolla becomes one with separate petals, and the leaf margins are eroded. All these modifications have been recently found in a hereditary mutation of the same host [1]. In speculating on the causes that may underlie some of these modifications, especially those of the type found in organoid galls, it is impossible to avoid the suggestion that they may be due to dislocation in the production or movement of growth controlling substances, much light on the existence and properties of which has been obtained in recent years. Little knowledge, however, is as yet available as to the part they play in stimulating gall formation though, as already mentioned, they seem to be concerned in the production of crown-gall tumours. It is worth mentioning that phytohormones are formed in abundance by some fungi (yeasts, *Rhizopus*, *Glomerella cingulata*) and may have a similar effect to those in a crude lentil extract in promoting growth in *Nematospora* and fructification in many fungi [7, 11]. It is not only the meristems that have the ability to respond to stimulation by the development of new tissues or by modifications in the products of their activity : other living tissues can play a part not less important. No doubt meristems respond very readily, and there may be indications that the pericyclic region is easily stimulated into activity in some plants, but the examples given above show that this sensitiveness must not be given undue importance. The generalisation has been made that in all living plant cells there slumbers the potentiality for the development of all histological characters that appertain to the particular species. The study of galls gives point to this remark.

Effects on Single Cells and Cell Membranes

So far the pathological modifications discussed have been chiefly those affecting tissues and organs of the plant. The changes in single cells and cell membranes brought about by the presence of a parasite have been only incidentally referred to both in this chapter and in earlier ones, but it will have been inferred that they fall into certain main groups according as the parasite is a cause of cellular necrosis or is one which stimulates the cell to divide and become more or less meristematic, or even lives in equilibrium with the host, causing no appreciable change in the earlier stages of its development.

Cell Contents

The cell may be killed without reacting against the attack. There may be no increase in size or change in the nature of the contents, except such as results from their death and consumption by the parasite. The whole cell contents may collect towards the centre in a grumous mass, which turns brown apparently from the oxidation of substances like tannin present in the cell, and breaks up into amorphous granules often with the formation of refractive brown globules ; these have been mistaken for parasites (*Pseudocomis*) on several occasions. Sometimes the liquid of the cell is rapidly removed, and the ordinary effect of wilting from drought (plasmolysis) is produced. Starch may be hydrolysed, or may

be left unaltered especially when the cell has been rapidly dried out. Several of the potato tuber rotting species of *Fusarium* can hydrolyse gelatinous starch, but have no effect on the unbroken starch grains of the tuber. Chlorophyll may be reduced—leading to pallor of the infected area (*Peronosporaceae*)—or destroyed, when browning usually follows. Or it may be increased (reversion disease of black currants) or preserved to form green islands after the rest of the green tissue withers (many rusts, *Rhytisma acerinum* on the leaves of sycamore, *Septoria apii* on celery, apple scab on leaves sometimes). In some rusts there is evidence that not only is the chlorophyll preserved but the chloroplasts are stimulated to increased activity by the fungus [10]. A development of red or purple pigment due to the formation of anthocyanin often results from infection by rusts, *Exoascaceae* and other fungi, especially on the side towards the sun. This is also a frequent occurrence in the abnormal stems, petioles and leaves produced by systemic rusts and other parasites such as *Bacterium fascians*. The common *Oidium euonymi-japonici* causes large red blotches from this cause on *Euonymus* leaves in the spring, which extend beyond the area bearing the haustoria of the fungus. The nucleus may be enlarged or become lobed or even fragmented, as in cells of the shepherd's purse attacked by *Cystopus candidus*. Calcium oxalate crystals may be increased in apple scab, or reduced in buckthorn infected with the aecidial stage of *Puccinia coronata*. Infection is reported often to stimulate the activity of the mitochondria, and also to cause an accumulation of phenolic compounds in the cell vacuoles. These have been thought to exercise a protective action against parasites. The distastefulness of uncongenial or resistant hosts to some fungi is probably due to the presence of substances in the cells; but it is a local reaction and not comparable with the formation of true antibodies such as play so important a part in the protection of the animal body against disease.

Cell Membranes

Action by the parasite and reaction by the host are more obvious in the cell membranes than in the cell contents. It has already been mentioned that the parasite may penetrate cell walls by pressure or by the solvent action of enzymes, pressure being chiefly used against cutin and suberin but also sometimes against internal walls devoid of these substances, while the pectin and cellulose membranes of the softer interior tissues are usually dissolved by enzymic action. Action on cellulose membranes has been, perhaps, most fully studied in certain leaf and twig parasites which occupy chiefly the subcuticular and epidermal walls (Fig. 150) and in fungi which attack woody tissue; action on the lignin (which is responsible for the most widespread of the secondary modifications which the primary membranes undergo) is best seen in woody tissues; while the soft rot which results from pectin dissolution has been most closely studied in tubers, fruits and other storage organs [9].

Cellulose is the most important constituent of plant membranes, forming the bulk of the walls of the softer tissues and of all young parts that have passed the pectic stage. Most parasitic endophytes are capable of dissolving cellulose by means of cellulose enzymes. Sometimes the power is restricted to certain organs

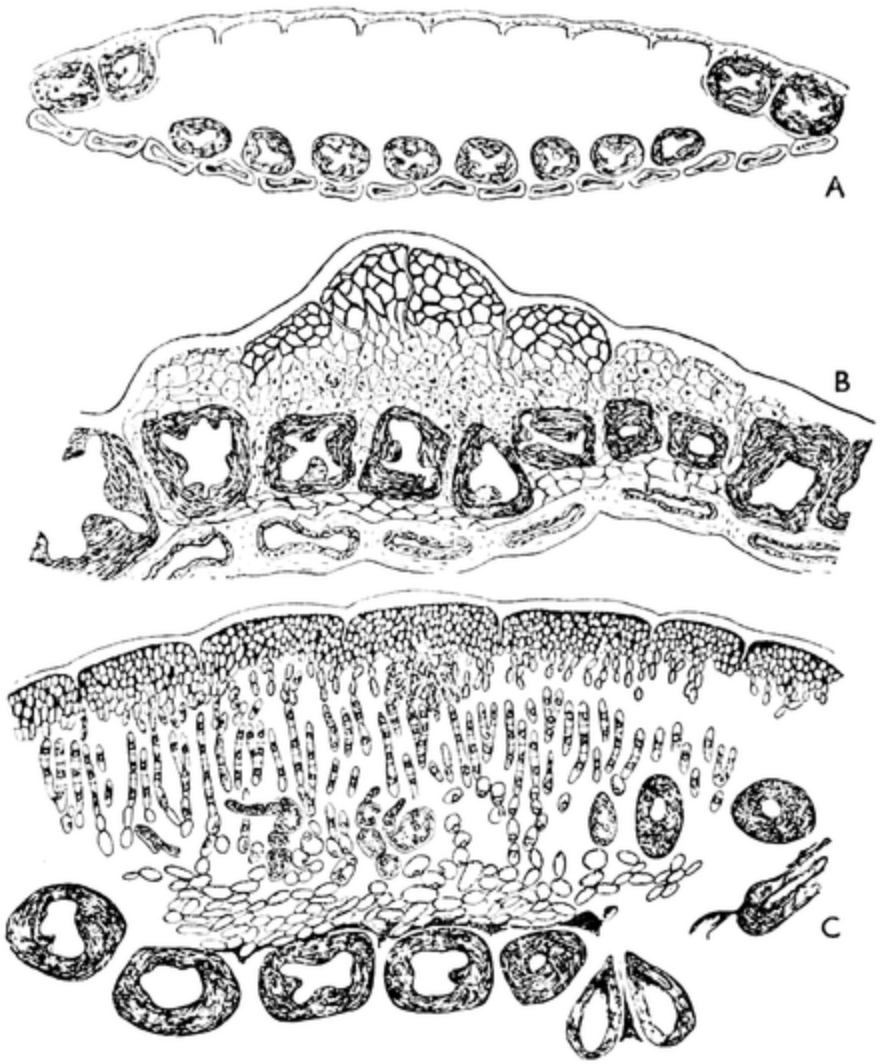


FIG. 150.—Action of *Lophodermium pinastri* in the leaf tissues of Scots pine. The fungus dissolves the middle lamella of the vertical walls of the epidermal cells. The sclerosed thickenings in the latter are thus removed to make room for the apothecia, at the base of which they collect, as in *B*, being pushed down by the developing fungus. *C*, an early stage of apothecial development showing ascogenous hyphae, and formation of a thickened roof to the apothecium (after Jones, *Ann. Bot.*)

or stages in development of the parasite, as in a number of *Peronosporaceae*, smuts, and the like that are intracellular in or near the epidermis in the early stages of penetration but are subsequently confined to the intercellular spaces; pressure, however, may be involved also in some of these last. Cellulose and other membranes may be penetrated by a hole which does not subsequently enlarge, presumably because in such cases the enzymes involved are released only from the tips of the hyphae.

In the subcuticular parasites whose vegetative thallus is mainly just below the cuticle or in the outer surface layers of the epidermal cell walls, entry into the host is effected by a fine infection hypha; this penetrates the cuticle and enlarges below into a mycelium which does not gain access to the lumen of the underlying epidermal cell. Some seem to dissolve their way in the cellulose part of the wall (apple scab) (Fig. 151), others to force a passage by mechanical means (*Guignardia bidwellii*). The epidermal walls are often impregnated with cutin, cutinised walls

sometimes extending to the radial and inner sides of the cells; mechanical force may be required in passing along these, since cutin-dissolving enzymes have not been found. But in most fungi of this type there are evident signs of dissolution of the cellulose and little doubt that the fungus gets part of its carbohydrate nutrition from this source. In practically all subcuticular fungi there are, at the same time, signs of depletion of the underlying cell contents, modifications in the chloroplasts, desiccation and browning, often accompanied by a formation of wound cork deeper in, so that there is evidently a passage of substances (enzymes, toxins, or the like) from the fungus to the host cell and, presumably, a transfer of nutrient in the reverse direction. The vegetative thallus, which often extends down to some extent in the radial walls of the epidermis, eventually is able to complete its development and form spores, set free by rupture of the cuticle.

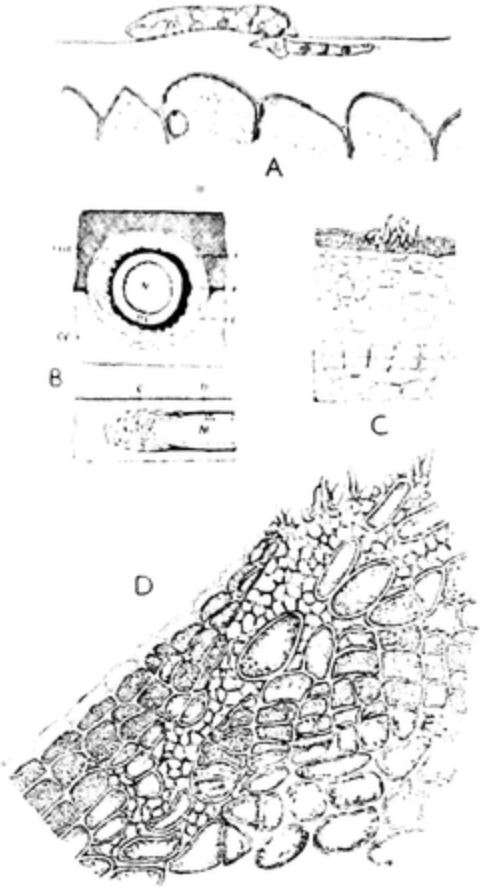


FIG. 151.—*A*, infection hypha of *Venturia inaequalis* entering cuticle of apple fruit. *B*, diagram of digestive action of a hypha on the outer membrane, in transverse and longitudinal section: *M*, hyphal cavity; *m*, hypha wall; *b*, brown products of digestion; *c*, zone of digestion; *cut*, cutinised part, and *ce*, cellulose part of membrane. *C*, formation of reactionary cork in apple twig attacked by *Venturia inaequalis*. *D*, penetration of the reactionary cork formed in pear twig attacked by *Venturia pirina* (*A*, after Wiltshire; *B–D*, after Ducomet. From Butler's *Fungi and Disease in Plants*)

Another group of fungi which consume the cellulose of the cell membranes comprises those responsible for the brown rots of wood. This is a large group containing both parasites and saprophytes but all characterised by attack restricted more or less completely to the cellulose, pentosans, and hexosans of the wood, and all increasing the acidity and the solubility in alkali of the products of decay. Even in extremely decayed wood, however, all the cellulose is not necessarily destroyed, and part of the pentosans may also be left (*Fomes roseus*). Lignin is little affected in this type of rot, the enzymes concerned in which seem to be those which effect hydrolysis (see p. 71). All the fungi involved can use cellulose as a source of carbohydrate nutrition; tests at the Forest Products Research Laboratory at Princes Risborough have shown that *Trametes serialis* can live with chemically pure Sitka spruce cellulose as its sole carbohydrate nutrient. The cellulose is usually converted into glucose before consumption. All the fungi in this group can also use the hexose and pentose sugars that result from the hydrolysis of hexosans and pentosans. Easily hydrolysable hexosans such as mannan and galactan may be amongst the earliest substances to be consumed, though more usually the pentosans are the first to disappear.

Amongst the saprophytic species affecting wood in this way the best known is

Merulius lacrymans, the chief cause of dry rot in structural timber in temperate climates (Fig. 18). This fungus dissolves much of the cellulose in six months, during which time it can cause a loss of 45 per cent. by weight of the wood, rising to about 70 per cent. in two years. Like many other wood destroyers it forms much water from the wood, which drips away (hence the name *lacrymans*) and also provides the moisture necessary for its further progress into dry timber. There is little or no delignification, the residue consisting of a rather homogeneous lignin body apparently similar to that yielded by sound wood on treatment with caustic alkali under pressure. A certain amount of the other constituents of the wood, more or less modified (oxycellulose, pentosans, etc.), are also left. The methoxyl content of the decayed wood may be increased by as much as 77 per cent. and uronic acid is also increased. The final product of the decay is considered to be closely allied to humus. *Coniophora cerebella* (*C. puteana*) can also reduce the weight of the timber by about 70 per cent. and leaves a humus-like residue. In the brown rot caused by *Poria vaillantii* the loss of weight in Scots pine sapwood may be about 55 per cent. after two years and in spruce wood attacked by this fungus all the cellulose may be gone in six months.

Trametes serialis is one of the most important sources of decay of coniferous timber in western North America, causing a cubical rot of redwood (*Sequoia sempervirens*) and a butt rot of Sitka spruce, Douglas fir, and other softwoods. It causes an acid hydrolysis of Sitka spruce which alters the chemical composition, increases alkali solubility and reduces the strength of the wood before any loss of weight can be detected. The loss in mechanical strength may exceed 15 per cent. in the first fortnight, whereas it may take four or five weeks before any significant loss of weight occurs. The wood is composed, as to about 99·1 per cent. of its weight, of cellulose, lignin, pentosans not in the cellulose, and water-soluble material, and this fraction was found to lose only about 4 per cent. in three or four months, the loss falling on the cellulose and pentosans. The rise in alkali solubility runs parallel to loss of strength, which may increase steadily to about 80 per cent. ; then it slows down. In the early stage there is little change in the appearance of the wood, and aeroplane timber which has failed under test may show, on microscopic examination, hyphae well in advance of any visible sign of decay. With *Polyporus schweinitzii* (see p. 943) it causes the condition known as ' dote ' in Sitka spruce, one of the chief timbers used in aeroplane construction.

Other active brown rot fungi which have little effect on lignin include *Fomes pinicola*, which is sometimes parasitic, but very rare in Britain, *Lenzites trabea* and *L. abietina*. *L. sepiaria*, which is a fairly common cause of decay of pine timbers, is found in pit props, can reduce the wood of *Pinus taeda* and *P. caribaea* to a brown crumbling mass and has an exceptional armament of enzymes, and *Lentinus lepideus*, which eventually may cause a slight loss of lignin owing to hydrolysis of the methoxyl groups in it, and which may, like *F. pinicola*, cause large holes in the wall when it passes from cell to cell.

The other main group of fungi causing decay in standing (p. 941) or fallen timber consists of those responsible for the so-called white rots. The white rots are more important to the forester as a rule than the brown rots, since they

comprise a number of the most serious parasites of the wood of trees (see p. 945). Their action is not limited to hydrolysis for they possess also oxidising enzymes, in contrast to the brown rots, many of which show reductase activity. They attack all the major components of the cell walls of the wood, but there is no uniformity of sequence or proportion in their destruction of these. Some attack the chief components of the wood simultaneously, others cause delignification before the cellulose is depleted, others again show a delayed delignification. The variations are in part due to differences in the time when one or another type of enzyme (hydrolytic or oxidising) becomes active, and the course and speed of the secondary reactions. Where delignification precedes cellulose decomposition, it has been suggested in certain cases that an oxidase from the fungus acts first on lignin and pentosans to produce acid and that the oxidase with acid can complete the process involving the depletion of the cellulose. The lignin destroying fungi can digest lignin presumably after decomposing the aromatic content and the aliphatic carbohydrates that constitute its two major groups of compounds.

Polystictus versicolor is a very good example of the white rotting fungi, for it can so completely destroy beech wood as to leave only a fraction of 1 per cent. after two years. It attacks the ray cells and wood parenchyma first, reducing the lignin, pentosans not in the cellulose, cold-water soluble and 1 per cent. alkali soluble compounds, before the cellulose and its associated pentosans and the hot-water soluble fractions are diminished. It also bleaches the wall pigments. Like many other white rotting fungi, the 1 per cent. caustic soda solubility of the pentosans not in the cellulose diminishes as the decay progresses, so that there is ultimately no marked increase in the total alkali solubility as compared with sound wood. Loss in strength exceeds loss in weight.

Trametes pini (Figs. 152 B, 434 1) causes a red rot in some hosts, but the white or pocket-rot type of decay is eventually found and is due to a progressive delignification working outwards from the tertiary layer of the membrane to the middle lamella; cellulose is attacked later. Like *Fomes annosus* (p. 915) it may dissolve the middle lamella, causing the cells to fall apart, before the cellulose is completely destroyed (Figs. 152 A, 424). *Trametes suaveolens* follows the same course from within outward in its action on the wall in willows and poplars, but cellulose is depleted before lignin. *Ganoderma curtisii* (regarded by some as a synonym of *G. lucidum*) causes a white basal rot of hardwoods in the south-east of the United States, destroying the middle lamellae of the vessels and large ray cells, delignifying these and corroding the bordered pits into large holes. *Stereum frustulatum* (Figs. 433 G, 435 B) causes

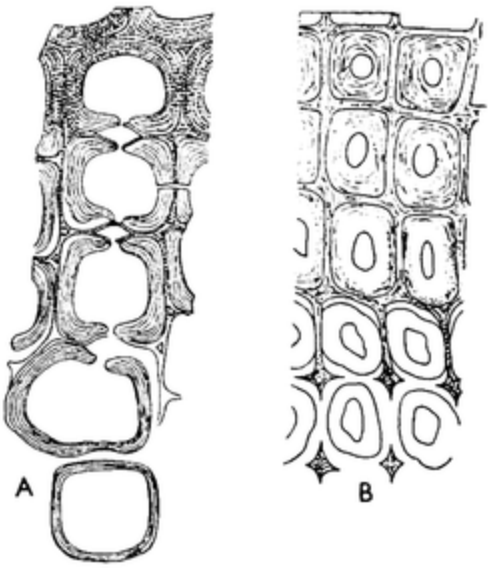


FIG. 152.—*A*, action of *Fomes annosus*, and in *B*, of *Trametes pini*, on wood of pine and spruce respectively. The lignified walls (dotted) are gradually delignified, and the middle lamella dissolved, leaving isolated cells below, with walls of cellulose (after Hartig and v. Schrenk)

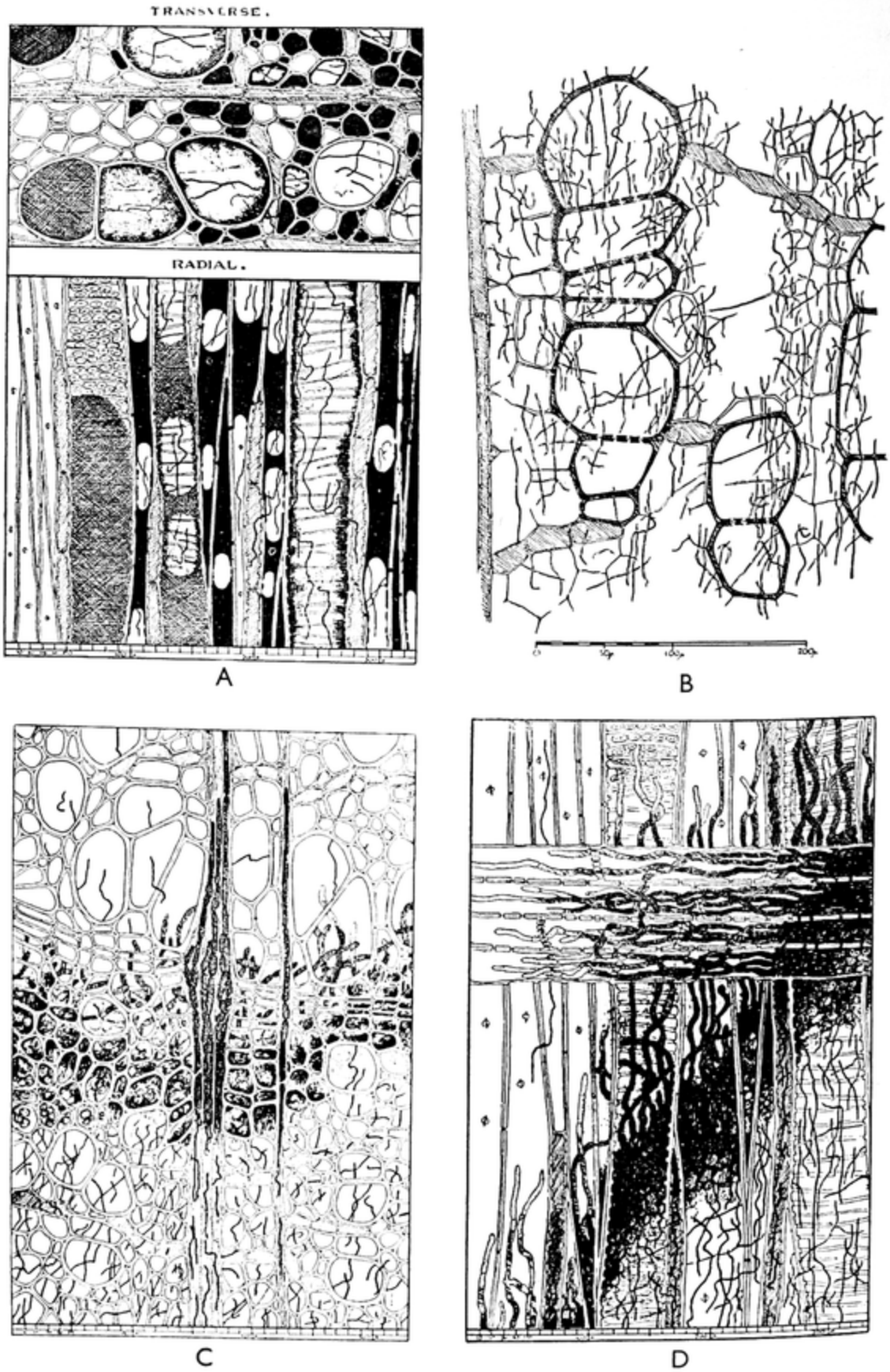


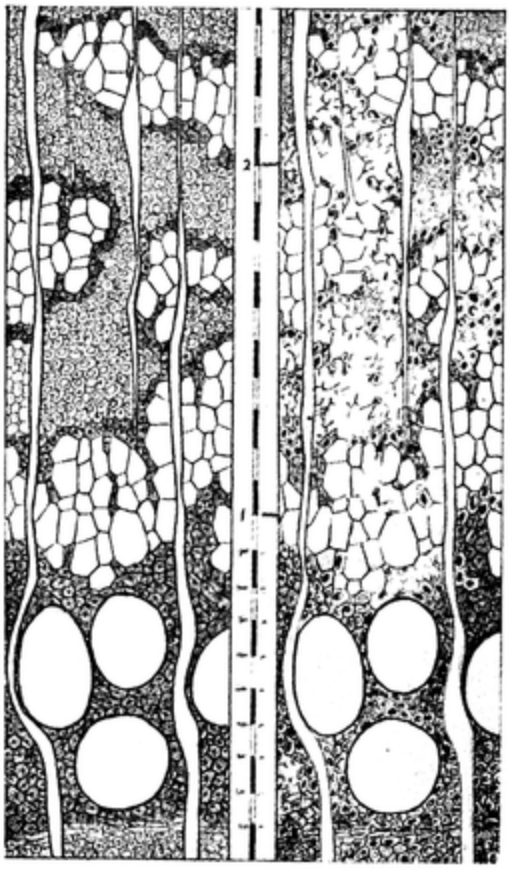
FIG. 153.—Wood of the lime attacked by *Ustulina vulgaris* (*U. deusta*). *A*, transverse and radial sections through the timber in the wound-gum region (stained with Cartwright's stain); the products in the vessels stain blue (cross-hatched), in the tracheids red (full black). *B*, transverse section of a very diseased part of the timber; decay is more marked on the autumn than on the spring wood of the annual rings; the large vessels of the spring wood appear to be unaffected; vessels, rays, and wood parenchyma show no sign of disintegration, but the

white pocket-rot of oak by an early concentration of delignifying activity in oval areas separated by firm woody tissue. The pockets at first contain cellulose with some hyphae but are eventually empty. All the wood-decaying species of *Stereum* act in much the same way, the white areas being merely places where the lignin decomposition has proceeded most rapidly so that a higher proportion of white cellulose is left, but some of the cellulose is consumed early and eventually most of it disappears so that there is no real selective action on lignin apart from the time factor.

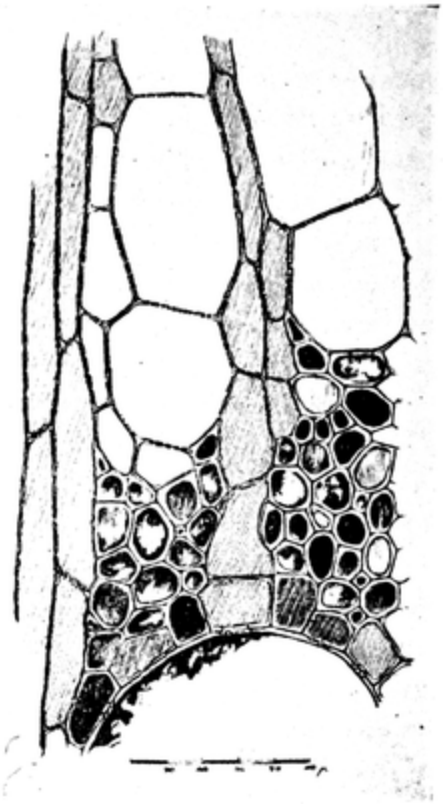
Armillaria mellea causes a decay which has some of the characters of both the brown and the white rots but can best be included with the latter, with which it shares the capacity to produce both hydrolysing and oxidising enzymes and failure to increase appreciably the total alkali solubility of the wood; at all stages this solubility is greater in the brown than in the white rots. *A. mellea* begins by attacking the cellulose and associated pentosans but it depletes the lignin also, causing a soft, wet white rot (in citrus), while in advanced stages of decay it may cause coniferous wood to turn light yellow or white, (e.g. in Douglas fir). *Polystictus abietinus* is a sapwood destroyer and can evidently assimilate the cell contents of the wood parenchyma as well as the membranes. It destroys cellulose more actively than lignin, though early attack on both has been reported. It has an exceptional enzyme armament, 15 hydrolysing and 4 oxidising enzymes having been detected. It can assimilate tannin, the phenolic groups of which are probably first oxidised by the laccase of the fungus.

Phellinus cryptarum gained a wide notoriety some twenty years ago as the agent chiefly responsible for the destruction by white rotting of the oak beams in the old Louis XIII wing of the Palace of Versailles. It is, in fact, a not uncommon cause of the type of decay of structural oak in England which is usually attributed to the 'death-watch' beetle, *Xestobium rufovillosum*. There is little doubt that a similar attribution would have been made in Versailles had the palace happened to be (like Westminster Hall) in this country, the more so since the beetle was found in some of the beams. Work at Princes Risborough has shown, however, that the death-watch beetle colonises chiefly wood that has already been softened by fungal decay, various types of which may be involved, and that it is almost impossible to get its first-stage larvae established in sound wood. This does not appear to be due to specific modification of any particular organic component of the wood, all the main compounds in which may be decomposed simultaneously, but to mechanical softening which facilitates boring by the beetle in conformity with the extent to which decay has proceeded. The life-cycle of the beetle is shortened according to the extent of decay, so that the speed of its depredations is increased. This effect, which may be considerable, is apparently associated with the nitrogenous metabolism of the insect, which is affected by the content and distribution of the nitrogen present in the decayed wood. While it would be difficult to assess the relative parts played by insect and fungus in bringing about final collapse of the structure, it seems safe to ascribe the origin of the trouble to

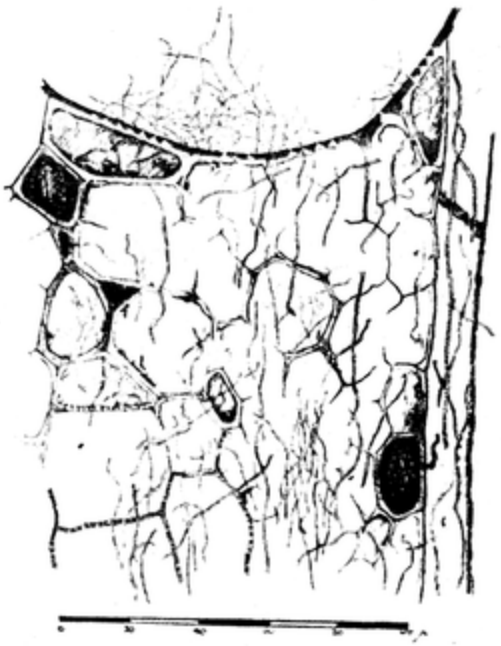
tracheids become completely disorganised, so that only the middle lamella is left, and finally the tracheids disappear entirely, leaving an empty space surrounded by the non-disintegrated elements. *C*, a transverse section of the junction between the diseased and the discoloured wood, showing the black line and the relative distribution of the hyphae. *D*, a longitudinal section through the same region as shown in *C* (after Wilkins, *Trans. Brit. Myc. Soc.*)



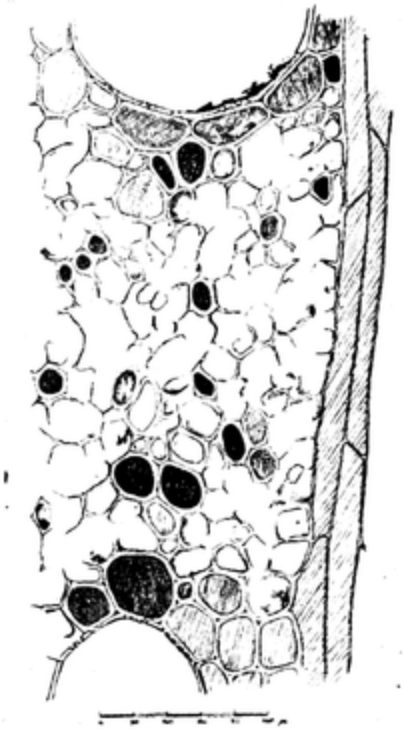
A



B



C



D

FIG. 154.—White rot of elm caused by *Ustulina vulgaris*. *A*, comparison of sound wood (left), and diseased wood (right); note, in latter, in general, the ring vessels of the spring wood, together with the rays, resist disintegration, but all the tissues in between, that is, the vessels, fibres, and wood parenchyma, sooner or later disintegrate. Progress of decay takes place in two stages, the first, shown in *B*, is the occlusion of the fibres and wood parenchyma by infiltration products which, at this stage of decay, stain red with Cartwright's stain, suggesting some kind

fungal decay, the prevention of which, by means that are quite well known, assumes an enhanced importance from the knowledge now available of its influence on the biology of the death-watch beetle.

The Ascomycete *Ustulina vulgaris* (*U. deusta*) is parasitic on the lime (Fig. 153), in the wood of which it destroys at the most advanced stage of decay about one-third of the original cellulose and one-fourth of the lignin. There is a red zone between the sound and decayed parts in which about 4·6 per cent. by weight is lost, almost all of it cellulose. It is also a parasite of elm heartwood (Fig. 154) in which it disintegrates the fibres, wood parenchyma, and vessels of the summer wood, leaving the medullary rays and large vessels of the spring wood almost unharmed. On *Acer* in America a brittle white cone of decay is caused, but eventually little is left but the black zones in the wood; these are further discussed below (p. 219). *Polyporus subacidus* has a similar selective action on the summer wood of spruce, but *Lenzites sepiaria* seems to prefer the spring wood in the early stages of attack. Marked differences also occur in ability to attack heart- and sapwood, as in *Trametes pini* which seems unable at first to attack sapwood, *Polyporus squamosus* which is much more easily established in heart- than in sapwood, and *Polyporus shoreae* which may be closely restricted to the latter. The fact that the sapwood may be an effective barrier to the outward extension of fungi decaying the heartwood may sometimes be very important in preserving the life of the tree.

It has recently been claimed from a study at the Yale School of Forestry of the white and brown rots of various coniferous trees decayed by *Fomes annosus*, *T. pini*, *Polyporus schweinitzii*, *Poria weirii*, *Lenzites trabea* and *Trametes serialis* that penetration of the walls of the wood is effected wholly by enzyme action. This is stated to cause local total dissolution of the membrane in advance of passage of the hyphae, which need not actually come into contact with the wall except perhaps at the site of the earliest stage in penetration when contact may stimulate enzyme secretion from the hyphal tip. Similarly it has been stated that the cellulose, lignin, and pectin of the wood of *Abies alba* are dissolved by the parasite *Fomes hartigii* in Austria entirely by enzymic action, without need for the mycelium to touch the wall. If these exo-enzymes were produced in quantities approximating that of the pectinase of *Botrytis cinerea*, there might result a weakening of the woody tissue along the course of the vessels above the area actually invaded by the fungus; so far, however, no evidence of this seems to be available.

Many other cells besides those of the xylem may become lignified, and many fungi other than those causing the brown rots of trees and timber find difficulty in penetrating or dissolving lignified walls (*Pythium*, *Sclerospora*, some rusts and smuts, etc.). The same seems to be true of *Xanthomonas stewarti*, by which a good many vessels of resistant varieties of maize may be penetrated while they are seedlings, and moderately susceptible varieties show a development of heavily lignified parenchyma around the infected protoxylem which is absent from the

of lignin complex; the second stage, shown in *C*, a cross-section from the summer wood (towards the end farthest from the spring wood), appears to be the gradual disappearance of the infiltration products, and disintegration of the fibres. The final stage of decay, in *D*, shows that continued disintegration leads to an almost complete disappearance of the fibres and wood parenchyma, as well as considerable breakdown of the vessels of the summer wood (after Wilkins, *Trans. Brit. Myc. Soc.*)

most susceptible varieties. Lignification is at once the commonest and, perhaps, the most rapidly brought about of the secondary modifications that cell membranes undergo. The walls of cells lying in the path of an invading mycelium sometimes become lignified, and this reaction can be regarded as a defensive one even though it does not necessarily check the progress of the parasite (chestnut attacked by *Endothia parasitica*) (Fig. 155). In this respect lignification is even less universally efficient as a barrier than the suberisation of the wall which often follows it or the development of wound cork, which as already mentioned is far from being invariably successful in the defensive reaction of the host. For this reason some find a difficulty in accepting such processes as defensive reactions at all; there is no doubt, however, that they are often highly efficacious, as against the parasites that cause diseases of leaves and twigs, in inverse proportion to the rate at which the invaded organ forms defensive barriers. In the long course of evolution it is not unreasonable to conceive a stage during which the defensive mechanism of the host held the upper hand, only to be broken through at a later period as certain of the aggressors acquired a speed of penetration too great to allow of secondary reactions in the membranes, or the facility of secreting lignin-dissolving enzymes (like the fungi causing the white rots of wood), or of bursting a path through corky barriers (*Armillaria mellea*) or turning their flanks (pear scab, Fig. 151 D). Nothing is more evident than that these 'defensive' reactions are now often blindly called into play without regard to their effectiveness, but this does not reduce their success against certain forms of attack, or lessen the possibility that they were once of considerable evolutionary value.

The soft rots due largely to the dissolving action of pectinase on the middle lamella of the cell membranes have already been discussed in an earlier chapter. They are responsible for much injury to storage organs, on which they act in the early stages by dissolving the middle lamellae and setting free the still living cells of the ground tissue. Eventually the cell walls and the contents are also broken down, but some, such as *Botrytis cinerea*, leave the lignified part of the membranes unaffected. Several bacteria can cause rotting of this type, the commonest in Great Britain being *B. carotovorum* (Fig. 274 F, G), a cause of soft rot in many vegetables and other plants. A good many Phycomycetes, such as *Pythium*, *Phytophthora*, *Rhizopus*, and the like, often produce a soft or watery rot in which pectic decomposition is a prominent feature. In certain diseases which ordinarily end in wet rot the original parasite causes a dry type of necrosis but secondary organisms following in its wake soon lead to a moist type of decay (*Xanthomonas campestris*, *Phytophthora infestans*). In most rots of this type there is little or no attempt at a visible defensive reaction on the part of the host.

Tyloses

When woody tissues react to parasitic and other types of injury by the secretion of wound gum, a second reaction is frequently seen which appears to have a similar objective. This is the development of tyloses in the vessels of the wood, especially that of trees (Fig. 156). Tyloses are outgrowths from the living cells of the wood parenchyma and medullary rays, which pass through the pits in the vessel wall and swell up into thin-walled bladders in the lumen of the vessel. In the Dutch

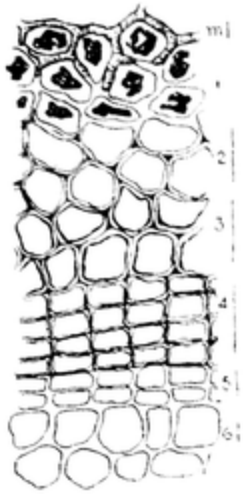


FIG. 155.—Scheme of defensive mechanism. Reaction of cells. *m*, mycelium; 1, cells dead, without reaction; 2, angular lignification; 3, ligno-suberisation; 4, cork; 5, meristem; 6, normal tissue (after Ducomet)

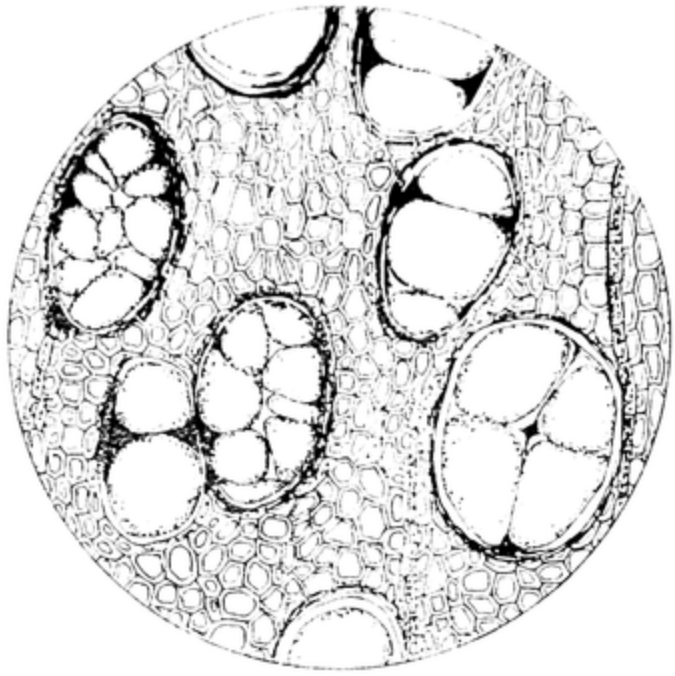


FIG. 156.—Tyloses in watermark disease of the willow, showing vessels completely blocked with tyloses (adapted, from Oxford For. Mem., No. 3, by permission of Day)

elm disease tyloses and gum are stated to come from the same cells and to be formed simultaneously from identical causes. In the watermark disease of the cricket-bat willow, the causal organism (*Bacterium salicis*) becomes active in the spring following infection of the outer layers of the new annual ring in the late summer, and oil accumulates in the ray cells of these layers, while tyloses appear in their vessels whether bacteria are present in the same vessel or not. These tyloses undergo a similar oily degeneration and the oil stains them and the contents of the ray cells brown. The brown stain has been suggested to result from the oxidation of catechol tannins or their breakdown products. Tyloses can be induced to form in elm, oak, lime, and other trees by injecting chemicals into the wood, but the degree of reaction to these various stimuli differs widely in different hosts. They seem seldom to be effective in blocking the way to parasites or preventing loss from other types of injury.

Black Lines

As already mentioned in the first chapter, various parasitic and saprophytic fungi which decay woody tissue produce sharply defined black lines. These are well seen in *Armillaria mellea* where they tend to mark the boundary between sound and decayed wood (Figs. 20, 21). The black line is the edge of a continuous band of brown bladder-like cells developed from hyaline hyphae in the lumina of the vessels, fibres, medullary ray and other parenchymatous cells. After a time the bladder hyphae collapse and their contents stain the walls of the enclosing cells and fill all gaps with an impermeable layer. This layer surrounds completely an area of host tissue colonised by the fungus, and it has been suggested that it, as well as the black layer formed by *Polyporus squamosus*, constitutes

an organ of preservation of the fungus to which the name ' pseudosclerotium ' has been given. Quite similar black lines are visible in wood infected by *Ustulina vulgaris* (Fig. 153 C, D) and in that containing the mycelium of saprophytic species of *Xylaria*, *Hypoxylon*, etc. (3, 4, 5, 13, 14). Another more diffused type of black line is caused by the production of the blocking layer of wound gum mentioned earlier in this chapter, and may be seen bordering the lesions caused by *A. mellea* in some hosts, demarcating the limit of the rot due to *Ganoderma applanatum* in many hosts and the decayed islands caused by *Fomes annosus* in the heartwood of the larch (p. 915); dark zones may also form as a result of the antagonism of two fungi along the line where the mycelia meet.

Resin and Gum Flux

Several parasites of conifers cause an outflow of resin, which is sometimes inimical to the progress of the mycelium; spruce produces resin less freely than pine and, possibly for this reason, suffers greater injury from certain parasites. Gum is exuded from many trees, especially citrus and the stone fruits, both from parasitic and non-parasitic causes. *Armillaria mellea* not infrequently causes a resin flux from the base of trees like Douglas fir and it is sometimes a cause of gummosis in citrus, walnut and various species of *Prunus*. Citrus gummosis (' mal di gomma ') was one of the earliest symptoms of disease to be recognised in this host. It may result from the attack of almost any bacterial or fungal parasite of the stem or collar but is most prevalent in infections by the brown-rotting species of *Phytophthora*. It can also result from chemical injuries caused by spraying or fumigation; is one of the symptoms of the copper deficiency disease known as exanthema; and sometimes develops without apparent external cause. There is a formation of clear watery gum in the living cells of the cambial region, which may collect into pockets between the bark and the wood and finally escapes to the surface through ruptures in the bark. In *Prunus* and *Acacia* also gummosis may accompany the attack of parasites or result from non-parasitic causes. In neither of these hosts is it a reliable symptom of the presence of any specific disease. The etiology of these fluxes is not well understood. They are quite distinct from the exudation of bacterial slime found in herbaceous plants (e.g. cotton affected by *Xanthomonas malvacearum*) or trees (poplar canker).

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Chapter VII

THE PRINCIPLES OF THE CONTROL OF PLANT DISEASE

In attempting to check diseases of plants, knowledge is usually required of the cause of the disease, of the life-history of the parasite, and of the circumstances which influence the establishment of parasitic relations between it and the host. Knowledge of the cause is of advantage, largely because it often allows of previous experience with the same or closely allied diseases being applied to specific cases; treatment may thus be possible as soon as a diagnosis is made. Knowledge of the life-history of the parasite is clearly essential before all the methods of checking it can be tried; such knowledge is available for most parasitic diseases of temperate plants but less often for those in the tropics. Knowledge of the circumstances which increase or diminish the power to injure of the parasite, and the resistance to attack of the host, has greatly increased during the past twenty years; these circumstances, however, are so linked with small differences in climate and other external conditions, with the crop varieties grown or the strains of the parasite involved, with local agricultural practices and the like, that it is seldom possible to utilise rigidly the experience with them of workers in other localities in combating a particular disease.

Considered broadly, it is impossible to assign a greater degree of importance to any one of these requisites than to the others. The cause of malaria in man was known for a considerable time before the life-history of the parasite had been followed out; and the value of the use of quinine in the disease, as manifested by its destruction of the parasite in the blood, has not been lessened by the subsequent discovery of the origin of the latter from mosquito bites, though this added to the methods of fighting it. The causes which lead to alterations in the severity of an attack are not likely, even when completely known, to reduce the significance of the earlier discoveries. Each step is dependent on what went before, and none can be said to be the most important. Viewed in this light, the perennial controversy between those who believe that the parasite is the important factor and those who consider that attention should be concentrated on the condition or treatment of the host loses much of its meaning: a knowledge of each is equally important when the study of the factors influencing the severity of a disease is taken up and its control is attempted.

While it is convenient to deal separately on paper with the three main phases of plant disease control, the attack on the parasite, the strengthening of the host, and the modification of the environment, in practice all must be taken together; a rational system of control to be applied to almost any parasitic disease will be based on considerations affecting the parasite, the host, and the cultural and climatic conditions in which the crop is grown. Very often these last are such



as to make the survival of the crop precarious ; for political or economic reasons the plant pathologist is posed with the problem of fighting disease in areas where nature has loaded the dice against him. The simple answer may not be given that the local conditions are unsuitable for the crop ; and it is left to the pathologist, in alliance with the plant breeder, to show how failure may be turned into success.

MEASURES DIRECTED AGAINST THE PARASITE

Methods of control based on combating the parasite include such aims as: (*a*) the exclusion of the parasite from contact with the host either wholly or during only temporary periods which carry special risk of infection, (*b*) field sanitation and the reduction of inoculum by attack on the sources of spores or other organs of infection, (*c*) protective applications to the host of substances that kill the inoculum before penetration is effective, and (*d*) curative measures intended to check the parasite after it becomes established.

(*a*) Exclusion of the Parasite from the Host

Quarantine and Prohibitions

Many of the most formidable outbreaks of plant diseases of which records exist have been due to the introduction of a crop parasite from some other part of the world. Whence the parasite came and how it entered are not always known, but there is sufficient evidence to show that trade in diseased plants is one of the greatest sources of danger. Against this menace most States have established plant quarantines.

Sometimes a complete embargo is placed on the importation of living plants or parts of plants of a particular species or group of species either from all countries or from only such as are regarded as dangerous owing to the occurrence in them of certain diseases that it is desired to exclude ; importation under Government licence may at times be allowed in mitigation of an embargo. Occasionally plant produce such as timber, or other substances such as honey, may be barred on the ground that they may introduce disease, while packings are often strictly controlled for the same reason. Frequently only plant material intended for planting is covered by the embargo, that intended for consumption being admitted, while special regulations may be applied to seeds. Importation of the controlled material through the post or by air may be totally prohibited or allowed under restrictions.

Sometimes planting material is admitted subject to detention so as to allow time for the detection of any disease that it may carry ; special quarantine stations, usually insect proof, are established for growing the plants under observation.

Sometimes the material is admitted subject to disinfection on entry. Disinfection is most frequently prescribed for seeds, as it is difficult to apply effectively to other plant parts ; it is obviously of little value against internal parasites (though heating is sometimes effective against these), but is useful for cleansing seeds from parasites or their spores occurring as surface contaminants.

Sometimes reliance is placed on inspection at the point of entry, the plant

quarantine administrative staff usually having expert advice available in the examination of doubtful consignments.

In the larger countries similar measures are sometimes taken to prevent the passage of diseases from State to State or Province to Province. They are not easy to apply and it has been found preferable in a good many countries to quarantine specified infected areas and prohibit the movement from them of diseased plants or other dangerous material. In Great Britain, for instance, an embargo was until recently placed on the supply of seed potatoes from wart-infected to wart-free districts, though it is now restricted to the wart-free areas near the Wash; in the United States there is an embargo on the movement of all elm material from certain areas in which the Dutch elm disease is known to exist, in the States of New York, Connecticut, New Jersey, and Pennsylvania, into other States free from the disease.

If imported material that may introduce plant diseases be divided into seeds, other planting or propagating material, and commercial produce, it may be concluded that disinfection is valuable for seeds in a good many cases but has a limited use against risk from the other categories; detention is valuable for planting and propagating material only; embargo may be necessary against commercial produce but is not often required against seeds or propagation material except from particular areas; while inspection has little value against danger from any of these categories. Commercial produce is often the most difficult to control and is the chief obstacle to an ideal quarantine system.

Quarantines have unquestionably been effective in preventing the entry of many alien diseases and of delaying the entry of still more. In spite of them, however, scarcely a year passes without its record of some new extension of a plant disease into some important area previously free from it. The United States have probably as stringent and as strictly applied plant quarantines as any country in the world, yet diseases of major importance have slipped through them several times during recent years. Two examples may be mentioned: the Dutch elm disease was imported in logs from Holland, probably on various occasions from 1928 onward, and reached several of the eastern States before a concerted effort was made, at the cost up to date of some £500,000 in direct Federal contribution alone, to check the spread; ten years later the ring-spot disease of potatoes caused by *Bacterium sepedonicum* was first seen in Maine, whence it spread with extraordinary rapidity through the potato-growing parts of the country. Most other countries have had similar experiences. They may be traced to various causes: the inherent difficulty in recognising disease in a plant as compared with an animal or man; the speeding up of transport so that material arrives fresh and viable even when it is only a bit of mycelium; the increasing commerce in plants by trade and 'fancier' interests always on the look-out for novelties from distant lands, and sometimes also the similar introductions by scientific persons or institutes in need of fresh material for breeding or study; ignorance of the distribution of many diseases, especially in the less developed countries whence many of the plant novelties are derived; and ignorance of the means by which particular diseases may be carried.

The prohibited imports in Great Britain include plants of the genus *Ulmus*

with a view to checking further introductions of the Dutch elm disease, and sugar beet and mangold plants (other than seeds) as a protection against virus diseases of these crops.

Certification

The dislocation of trade caused by plant quarantine is such that strenuous efforts have been made to secure their relaxation by measures directed to ensure that only healthy plant material is offered by the exporting countries. This entails a close supervision by accredited persons of the growing crop intended for export, and the issue of certificates of freedom from disease or from certain specified diseases by competent authority in a form acceptable to the importing country.

In Great Britain, for example, all living plants (including all parts of plants except seeds) that are intended for planting are prohibited ingress unless accompanied by an official certificate in a prescribed form from the phytopathological service of the country in which they were grown. The certificate must vouch that the living plants (or representative samples of them) were thoroughly examined on a date which must be not more than fourteen days prior to shipment and were found to be healthy, and must also state, unless the consignment consists wholly of potatoes, that no plant of *Ulmus* or of *Beta vulgaris* is included. For all potatoes an additional certificate is required that no case of wart disease has occurred on the farm or holding on which they were grown nor within two kilometres of it. Provision is made for the inspection of any consignment and for its treatment, destruction, or return if required by the inspector ; this regulation applies equally to packing materials and containers. In particular cases licences to import plants covered by these regulations may be issued by the Departments of Agriculture in England or Scotland. In Canada measures are taken to prevent the export of diseased or insect-infested consignments of certain plants.

Notification of Plant Diseases

Many countries list certain plant diseases, the occurrence of which must be notified by the owner or grower of the crop to the authorities. This is usually prescribed only for such diseases as have a limited distribution within the country, or against which restrictive measures are in force (e.g. potato wart and onion smut in Great Britain), and is intended to facilitate measures to prevent their further spread (e.g. the growing of immune varieties or treatment in a prescribed manner). Apart from such compulsory notification, plant pathologists, conscious of their ignorance of the world distribution of many important plant diseases, have urged the necessity of phytopathological surveys in all countries, and have often done their best to ensure that knowledge of the occurrence of particular diseases in their areas is made available to their colleagues in other countries. Experience has shown, however, that these voluntary efforts do not go far enough, and that there is need of the extension of the principle of compulsory notification to the international sphere.

Eradication

Attempts have been made in several countries to exterminate destructive plant diseases of limited occurrence within the country by the eradication of

all existing cases and quarantine measures to prevent new introductions. Some of these are described in the section on virus diseases (Chapter VIII), others have been applied against diseases caused by bacteria and fungi. Amongst the more successful have been the campaigns against citrus canker (*Xanthomonas citri*) in the United States and South Africa. This disease was probably introduced into the United States from Japan in 1911, and was well established in Florida and other Gulf States when recognised some years later to constitute a serious menace to the great citrus industry of that region. Eradication was vigorously pursued, a total of not far short of 4,000,000 grove and nursery trees being destroyed. Florida was cleared by 1927 and the other commercial areas shortly afterwards, so that it was reported in 1935 that complete success had been achieved, at a cost of more than $2,500,000. Subsequently, however, cases continued to occur in non-commercial areas in Texas and Louisiana, and an intensive effort was made with relief labour to destroy this source of danger. This increased the number of citrus trees destroyed by another 13,600,000 to the end of 1936. From 1937 to 1940, 1,144 diseased trees were found in the two States, but in 1940 only one infected property in each State could be seen, while all the commercial areas in the United States maintained their freedom from the disease. In South Africa the disease probably also came from Japan, and in the first year of eradication, 1917-18, 11,700 infected trees were found; after 1920, however, few cases occurred and in 1939 it was reported that none had been seen in the Union for nine years. The total cost up to 1927 was £56,193, but this included some other services.

In areas where *Puccinia graminis* cannot, for climatic reasons, persist on cereals or grasses throughout the year, barberry eradication, enforced by law in some countries, has proved a powerful means of reducing losses from black rust. Indeed, barberry eradication laws had been enacted in Europe and America in the sixteenth and seventeenth centuries because of the obvious part played by the shrub in the blasting of the corn. In several northern European countries barberries are not allowed to grow within a certain distance of cultivated land (the limit is 200 metres in some regions), while in others the barberry has been outlawed from arable land. In parts of Australia (where the barberry is not indigenous) every owner and occupier of land is required to destroy any barberries on it. In the United States it was estimated in 1934 that the barberry eradication campaign in thirteen of the north-central grain-producing States between 1918 and 1933 had led to the destruction of 6,000,000 bushes and a saving of $20,000,000 annually to the grain-growers. In the intensified campaign subsequently carried out no less than 68,500,000 bushes are stated to have been destroyed in the United States in the single year 1936.

Less happy has been the experience in the attempt to limit the destruction of the immensely valuable stands of white pine and other five-needled pines in North America caused by the blister rust, *Cronartium ribicola* (Part II, p. 929). This rust reached the United States in the last century and was for a long time confined to the east. Voluntary, State, and Federal effort combined to prevent its spread to the west and to reduce its ravages by the eradication of the alternate hosts, species of *Ribes*. Large areas were cleared of *Ribes*, the highly susceptible black

currant being practically outlawed in some districts. Spread continued, however, so that by 1938 the rust was reported to have become established in most of the commercially valuable pine-producing areas of the United States, and the inter-State quarantines against the movement of diseased plants were withdrawn from all but eleven States and a part of California. The established fact that infection can spread from pine to *Ribes* over a distance of many miles is one of the main reasons for the failure to check spread in this case, but though the measures taken did not protect the western States, *Ribes* eradication has been continued for the lessening of blister rust damage; in 1936 alone, 196,211,187 *Ribes* bushes were eradicated from 3,829,890 acres, of which 85,385 were cultivated black currants in the Lake States. The standard practice of eradicating the alternate hosts within 900 feet of the pine plantation borders affords fairly efficient protection of the latter.

In New Zealand the control of fireblight (*Bacterium amylovorum*) in pear and apple trees has been materially aided by the Act passed in 1922 which enabled the Government to prescribe the removal of hawthorn bushes, which are a dangerous source of infection, from the vicinity of the orchards in commercial fruit-growing districts. The planting of maize in certain places in Queensland has been prohibited because it was found to serve as a reservoir for *Sclerospora sacchari* on sugar-cane [8]. Similarly in Czechoslovakia the owner or tenant of land may be required to destroy wild hop bines as a protection against hop downy mildew (*Pseudoperonospora humuli*) (Part II, p. 879).

In England an attempt has been made to limit the losses caused by the water-mark disease (*Bacterium salicis*) of the cricket-bat willow by an order compelling the felling of infected trees in the county of Essex, the chief endemic centre of the disease.

Occasionally the elimination of all the diseased individuals of some particular host plant is prescribed even where there is no expectation of total eradication of the disease, so that the interests of the general body of growers may be safeguarded. This is the justification of the Silver Leaf Order in England which enforces the destruction of dead plum and apple trees and their wood infected by *Stereum purpureum* by a certain date, though this fungus has too many hosts for complete eradication to be practicable. In certain parts of Jamaica eradication has had to be abandoned, after the removal over a period of years of some hundreds of thousands of banana plants had given time for a large-scale programme of breeding resistant varieties to be undertaken, with promising results, against Panama disease.

Prevention of Sale of Diseased Plants

Many countries aim at preventing the sale of diseased nursery stock or other planting material. In England, for instance, it is a punishable offence to offer for sale any fruit trees with canker, silver leaf, or American gooseberry mildew, any brassicas with club-root, or seed potatoes with powdery scab; in Scotland onion smut is substituted for the last-named. Some countries go farther and aim at the prevention of the sale of diseased plants by controlling the nurseries in which they are grown. Thus, in parts of Germany there is a regular official inspection of nurseries and other horticultural establishments in which Douglas firs are grown, in order to detect the presence of certain diseases, especially that caused by

Rhabdocline pseudotsugae; if found, the destruction of the infected plants within fourteen days may be ordered.

(*b*) Field Sanitation and the Reduction of Inoculum

Compulsory Treatment of Plant Diseases

Not only is the sale of diseased planting material dangerous but the presence of disease in commercial plantations carries a risk of infecting neighbouring holdings. To obviate this, powers are taken to inspect orchards and the like, and to prescribe appropriate treatment. In England such powers are granted by the Ministry on the application of the local authority, usually the County Council. Many of the counties have used the Fruit Tree Pests Orders to empower their officers to inspect orchards, and, if such diseases as scab, canker, the *Sclerotinia* brown rots, and the like are not reasonably controlled, to prescribe treatments by serving an order on the occupier. Similarly in Germany it is possible for the local inspector to prescribe such a treatment as the cutting out and burning of cherry wood affected by *Sclerotinia laxa* before a certain date. Other countries have similar regulations. Furthermore, steps are sometimes taken to prevent the carry-over of a disease of annual recurrence from one season to the next by enforcing a close season during which plants of the crop concerned are not allowed to be grown. In New South Wales the owner or occupier of land on which tobacco was growing on the 31st May in any year had, after the disastrous outbreak of blue mould (*Peronospora tabacina*) in 1934, to uproot and burn all tobacco plants not later than the following 30th June. Similar local regulations have been enforced in Germany to prevent the over-wintering of asparagus rust, and in Southern Rhodesia against certain tobacco diseases.

Sometimes powers are taken to enforce the growing of certain varieties of a crop that are resistant to a particular disease, as is done in England and other countries in areas infected by potato wart disease, or to prohibit the growing of particularly susceptible varieties, as of sugar-cane in Queensland in areas infected with downy mildew (*Sclerospora sacchari*) or gumming disease (*Xanthomonas vasculorum*). In this State of the Commonwealth the control of sugar-cane diseases is vested in the Director of the Sugar Experiment Stations. The State is divided into ten quarantine areas, removal of sugar-cane from one to another of which is prohibited. An area may be declared an infected area and a local Control Board, financed by a levy, is set up to undertake the responsibility of carrying out control measures by inspection and treatment or destruction of diseased canes.

In Kenya the Governor can prohibit the movement within the colony of any plant or seed in a diseased condition or likely to spread disease, or the cultivation of any crop liable to impede the proper control of a disease.

The measures so far considered are those undertaken by administrative authority in the national interest or the interest of the general body of growers. They are not applied in the interest of the individual grower. The individual is bound to regard disease control as it affects his own pocket; measures that may

be imposed for the general good may be quite uneconomic when applied to a single holding. Costs and yield increases then become dominant considerations in the fight against the parasites, which is mainly carried on by field sanitation and the use of chemicals.

General Field Sanitation

The object of most sanitary measures is to reduce the sources of infection by micro-organisms. The best way of doing this is the removal of all infected individuals before they have time to spread the disease. This is the purpose of the large-scale eradication campaigns mentioned above as undertaken by administrative action where there is a prospect of exterminating a disease or at least of checking its extension.

In parasitic plant disease the sources of infection are most often the spores of fungi. In temperate climates their production is usually seasonal and restricted to the period from spring to autumn. The persistence of the parasite during the winter, when plant growth is dormant, is frequently secured by the production of resistant or resting spore forms in the late summer and autumn, or of resting or perennial mycelium which resumes activity and produces spores in the spring. A fuller discussion of this phase of the life-history of temperate parasites is given in Chapter II. Very often the winter spores are produced on the dead plants or parts of plants killed by the disease in the preceding summer and autumn. The removal and destruction by burning of these sources of infection is, therefore, a first principle in the sanitary supervision of orchard and plantation crops, and is sometimes applicable also to market garden and seed-bed crops, though usually not practicable in ordinary field cultivations. Pruning out and burning diseased woody parts is recommended for the control of such diseases as canker, scab, and mildew of apple, blossom-wilt and brown rot of fruit trees, leaf blight of quince, *Fomes* disease and die-back of plums, witches' brooms, bacterial shoot wilt of plums, leaf-curl, and scab of peach, anthracnose of vine, etc.

Dead leaves are sometimes an equally prolific source of inoculum. Where ascospores are important sources of apple and pear scab attacks in the spring it is frequently recommended to plough under the fallen leaves in the autumn or to treat the diseased leaves with a fungicidal spray either while still on the tree or after they have fallen. For treatment on the ground, not only are the common sprays such as Bordeaux mixture and lime-sulphur effective but chemicals such as sulphate of ammonia and chloride of potash have been found to check maturation of the ascospores. In the United States an eradicant spray of sodium dinitrocresylate has been recommended both for fallen apple leaves with scab and for cherry leaves with *Coccomyces hiemalis* leaf spot.

Ploughing in of the leaves has also been advocated in vineyards as a means of preventing aerial or rain splash dissemination in the spring of the winter spores or the products of their germination from leaves containing oospores of *Plasmopara viticola* or apothecia of *Pseudopeziza tracheiphila*. Early ploughing of the stubble of cereals infected by *Ophiobolus graminis* gives a better chance of destruction of the resting mycelium before the next susceptible crop is sown. Deep ploughing under (to more than 3 inches) of leaves and the like that carry the sclerotia of

various species of *Sclerotinia* or of the fruit 'mummies' from which apothecia also develop may cause the sclerotia to give only sterile stipes in the spring. It has been sometimes recommended against the species that attack fruit trees and against *Sclerotinia sclerotiorum*; but it is difficult to bury all the sclerotia, and, besides, the species attacking orchard fruits in the British Isles rarely develop the ascigerous stage, so that this measure has a limited value against them. Against *S. sclerotiorum* on celery in Florida, flooding for six to eight weeks in the summer or applications of calcium cyanamide after harvest or before planting were found equally and highly effective in destroying the sclerotia, and gave better control than attempting to bury them.

In tropical countries, where the dormant season may be in the hot dry weather rather than the winter, or where growth may be more or less active all the year round, much may still be done by the removal of dead or dying parts of infected woody plants, as these are often potent sources of inoculum.

Other important means of reducing inoculum in parasitic diseases of crops are seed disinfection and crop rotation, but these can be more conveniently considered later.

(*c*) Protective Applications of Substances to prevent Infection by Parasites

The application to the surface of the host plant of substances that prevent the germination of spores or kill the germ-tubes before they can enter the tissues is still one of the most widely used means of attack on plant parasites. The above-ground parts of plants are sprayed or dusted with chemical compounds or mixtures, wounds are protected by applications in paste or other convenient forms, seeds are disinfected by immersion in solutions of chemicals or by dusting or sprinkling with the compound or mixture. In some of these treatments, especially the last, the object is not so much the protection of the host plant as the reduction of inoculum by killing or preventing from germination the spores and other agents of infection present on the surface; but they mostly have some protective action, and their inclusion here is convenient. Soil treatment may also be included, for though theoretically their object may be to reduce inoculum by destroying spores or other infective material throughout the soil in which the crop is to be grown, in practice it is frequently impossible to do more than surround the roots with a fungicide or bactericide which will preserve them from infection. The use of heat as a disinfecting or sterilising agent may also conveniently be considered here, though it is definitely curative and not protective.

Spraying and dusting have a limited application in that they are both troublesome and rather expensive. The increased yield must, therefore, be of substantial value to outweigh the trouble and cost. They find their chief use in orchard and plantation crops, vineyards, potatoes, and market garden vegetables and flowers. The ordinary run of arable crops — cereals, many roots, pulse, brassica and other fodder crops, and so forth — are seldom sprayed or dusted against fungal or bacterial diseases.

Omitting chemical applications against the first spring infections from overwintered spores or mycelium, where protection is the object aimed at, there are

two main classes of parasites that can be usefully attacked by fungicidal dusts or sprays, but where it is difficult to separate the protective from the curative action. In the first place, ectophytic parasites, passing as they do the greater part of their lives on the surface of the host, may easily be reached by the superficial application of a poison before they can attain safety by burying themselves in the tissues. In the second place, those endophytic parasites which are disseminated by spores borne in crops or irregularly on infected organs and carried to healthy surfaces may be attacked before or during the sporiferous stage, while the exposed surfaces may be coated with a deposit which inhibits infection. Protective and curative functions of the treatment cannot readily be separated in either case.

To the first class belong the conidial stages of the *Erysiphaceae* or powdery mildews, the spores of which are borne on superficial hyphae and only haustoria (which cannot reconstitute the mycelium) ordinarily enter the tissues. Suitable fungicides not only protect uninfected surfaces but destroy the sources of infection. In the second class are a large number of fungi which live within the plant and whose mycelium, therefore, is out of the reach of superficial poisons. They are, however, obliged to come to the surface to reproduce, and their spores are formed on hyphae exposed to the air. They are vulnerable at this stage, and at the same time a fungicidal layer may be deposited on healthy surfaces of susceptible plants within reach, so as to prevent the germination or kill the germ-tubes of those spores which may fall on these surfaces. Most other fungicidal applications are protective, their curative action, if any, being of minor importance.

A good fungicide should be toxic to the parasite or inhibit the germination of its spores, without unduly injuring the host: in other words, it should be fungicidal and not phytocidal. It should be reasonably easy to prepare and not too expensive. It should be capable of even distribution from the spraying or dusting machines on to the surfaces to be covered, should tend to remain on the surface without running off too freely (initial retention), and should have good 'sticking' properties (tenacity) so that it is not easily removed after drying. If a liquid, it should have good wetting properties so that it does not tend to run together into large drops or to allow air pockets to form and keep it from contact with the surface to be covered; retention on the leaf is largely correlated with wetting properties, whereas tenacity is equivalent to ability to resist weathering.

Compounds and mixtures of sulphur and of copper with lime have remained for many years the standard fungicides fulfilling the above requirements, except for seed disinfection, where mercurials, formalin, and other substances have often proved more suitable.

Sulphur

Sulphur is one of the oldest of the fungicides, having been used in English glasshouses for the control of powdery mildew of the vine in 1846, because it was then well known as an effective remedy for peach mildew. Great quantities of sulphur are still employed for the same purpose in the vineyards of southern and central Europe and in North Africa, where three or four applications of a sulphur dust, totalling altogether from 60 to 75 lb. sulphur per acre, are most frequently given.

The sulphurs available for dusting are the following :

(1) Ground sulphur prepared in grinding mills and composed of crystals of various sizes from about 4 to 250 μ, not aggregating into masses. Good samples may have 98 per cent. of pure sulphur.

(2) Sublimed sulphur (' flowers of sulphur ') prepared by condensation from the gaseous form. The unit particles are from 8 to 30 μ in diameter but aggregate firmly into chains or plates up to 400 μ in diameter. This type may be 99·5 per cent. pure sulphur, and its lightness tends to make up for its greater cost by weight than ground sulphur, since only about three-quarters as much are required to afford an equal cover.

(3) Precipitated sulphur prepared by chemical precipitation and composed of unit crystals from 2 to 12 μ in diameter, firmly aggregated into masses up to 200 μ in size.

(4) Natural impure sulphurs of the ' ventilato ' or wind-blown type, chiefly got (in Europe) in Italy and containing about 30 per cent. sulphur in an argillaceous matrix or gangue which serves as a filler. A much purer natural volcanic sulphur having over 90 per cent. sulphur in an extremely finely divided form is marketed from Java.

(5) Impure sulphurs obtained in the purification of coal gas. In an American product known as ' flotation ' sulphur, some brands of which contain about 60 per cent., others about 40 per cent. sulphur, the particles are from 2 to 10 μ in diameter. In France the so-called ' black ' sulphurs from gasworks, containing about 40 per cent. sulphur, are popular in some districts.

(6) Various proprietary forms of sulphur characterised largely by the fineness of division of the sulphur particles. As an example, kolodust as used in North America may be mentioned. It is made by grinding a fused mixture of the almost colloidal sodium clay, bentonite, and sulphur and adding this to an ordinary wind-blown sulphur. The result is a fine dust of high fungicidal efficacy.

The fungicidal efficiency of a sulphur dust depends on the fineness of its particles ; a high proportion should pass a 200- or 300-mesh sieve (particles not exceeding 74 and 47 μ, respectively, in diameter), and still finer division is preferable.

Sublimed or ground sulphurs are the forms chiefly used in vineyards, ventilato sulphur being preferred in some parts of Italy and Germany and the black gas sulphurs in places in France and North Africa, as these types are cheaper and said to be more adhesive and less liable to cause scorching than the refined forms. In very hot weather the latter are liable to cause scorching and an admixture of one-fifth to one-half of ground spent lime, gypsum, talc, or other filler is then advisable. In recent years there has been a great improvement in the fineness of commercially available dusting sulphurs which have replaced sublimed sulphur in most countries.

In the intensive and successful campaigns against rubber Oidium in the Dutch East Indies and Ceylon the natural Javanese volcanic sulphur has been largely used. Its tendency to aggregate into larger particles can be prevented by adding unslaked lime in the proportion of 1 to 10. The dust is liberated from

specially designed power machines which throw a dense cloud of sulphur into the air to settle on the trees. A good machine can deal with as much as 300 acres a day (in rough ground this may be reduced to 100 acres), at a cost which even in severely infested cases should not exceed six or seven shillings per acre per annum and may be as low as two or three shillings, exclusive of the cost of the machines; the quantity of dust required during the season varies from 25 to 75 lb. per acre. This is, perhaps, the best example of the successful use of sulphur dusts.

The principal liquid forms in which sulphur is used for spraying are the polysulphides (of which lime-sulphur is by far the commonest) and colloidal sulphur.

Lime-sulphur sprays are ordinarily prepared from concentrated compounds, available commercially, the calcium polysulphide content of which should be stated. In diluting them for use, reliance is frequently placed on the specific gravity as shown by the reading on a Baumé hydrometer, a common reading required for the concentrate being 32° Bé. The dilution is then often made by volume; for instance, one part of the concentrate made up to 40 parts by the addition of water gives a $2\frac{1}{2}$ per cent. solution of the concentrate. A more accurate measure of fungicidal efficiency, however, is given if the percentage by weight of the calcium polysulphide is used. Thus, if 5 gallons of a concentrate containing 20 per cent. by weight of calcium polysulphide is made up to 100 gallons with water, a 1 per cent. lime sulphur is given. Tables are available for calculating the required dilutions.

Lime-sulphur finds its principal use on tree fruits, many standard orchard spraying practices being based on its application either throughout the spraying season or for only certain stages. It is the standard fungicide recommended by the East Malling Research Station for the control of apple scab and is stated to be, at the same time, the most valuable material against apple mildew. Elsewhere Bordeaux mixture is often preferred against apple scab, either wholly or for a part of the spraying programme. The dominant consideration in both cases is the securing of efficient scab control without injury to the trees, and from this point of view neither is wholly satisfactory on all varieties under all conditions. Lime-sulphur, for instance, not only directly injures certain varieties (' sulphur sensitive ') but has an indirect effect in reducing photosynthetic activity in the sprayed green parts even in the absence of scorching.

Various proprietary forms of ' dry wettable ' sulphur, some from by-products of gas purification plants (including flotation sulphur, wettable powder, and paste), are used in place of lime-sulphur in some fruit-growing regions, especially in the United States. Their sulphur content varies, but some forms may have about 90 per cent. They usually cause less injury than lime-sulphur though still liable to damage sulphur-sensitive varieties, and are stated to be useful under English conditions as adjuncts to the main apple-spraying programme where frequent summer applications are practicable. The same applies to the colloidal sulphurs, which are outstanding in the fineness of division of the sulphur particles (mostly below 1 μ). They are extensively used in New Zealand either alone or added to lime-sulphur of which they are said to increase the efficiency against apple scab and mildew, and rust and brown rot of stone fruits; they have been recommended

in England for supplementary applications against apple scab. Such preparations generally find their chief scope when a balance has to be struck between fungicidal efficiency and phytocidal injury to the host plant, or where (as in glasshouses) an odourless and harmless material is advantageous. The cucurbits, however, are highly sulphur sensitive, so that cucumbers and the like are readily injured.

Copper

Though copper sulphate had been tried as a fungicide much earlier, it was not until Millardet accidentally observed its efficiency against the vine downy mildew, *Plasmopara viticola*, in 1882 that its possibilities were recognised. Millardet noticed that grape vines along the roadside in the St.-Julien district of Médoc retained their leaves at the end of October when all the others had been defoliated by mildew. His companion M. David, steward of Mr. Nathaniel Johnston's estate in the neighbourhood, explained that it was the custom to sprinkle the vines near the road with copper sulphate and lime to discourage pilfering. The following year Millardet and David obtained confirmation that this practice checked the disease. Further tests under the supervision of Millardet and Gayon, who were professors at the University of Bordeaux, were carried out near Margeaux; in October 1885 they published illustrations of treated and untreated vines with analytical data, and by 1887 great success was reported in the control of the disease [9, 13] (Part II, p. 833).

The mixture of copper sulphate and lime rapidly became famous under the name 'Bouillie Bordelaise' ('Bordeaux mixture') in the vineyards ravaged by vine mildew. Already in 1885 its use against the allied potato blight was suggested and it was stated to have checked an attack of blight on tomatoes. In 1888 its efficacy against potato blight was established by experiment and other applications followed fast. At first it was applied in vineyards as a paste brushed over the vines with fibre brooms, but by 1885 a sprayer for application in liquid form was described by A. Perrey in Burgundy. Though the properties of the ingredients and the manner of preparation have changed many times, Bordeaux mixture remains the most widely used and the most generally serviceable fungicide to the present day.

The advantages of Bordeaux mixture are: (1) its adhesiveness or tenacity; (2) its effectiveness as a fungicide; in a recent laboratory study of copper fungicides in the United States the authors conclude that after fifty years of experimentation with copper compounds none has been found to equal Bordeaux mixture in fungicidal value; (3) its relative cheapness; (4) its safety to handle; (5) its harmlessness, with certain exceptions, to the sprayed plants; (6) its beneficial effects on some plants in preserving the foliage, apart from its fungicidal actions. This last effect, which is shared by some other copper sprays, especially Burgundy mixture, has been often observed in sprayed potatoes and seems to be due either to a stimulating effect of minute traces of absorbed copper upon the chlorophyll apparatus of the green parts or a slowing of the metabolic processes in the leaf. As against these must be set: (1) its injurious action on some fruit trees — varieties of apples, peaches, plums, etc.; (2) its tendency to delay ripening; (3) the need for taking some trouble in its preparation; (4) the injury it causes to spraying

machines of iron or zinc; (5) the colour it imparts to the sprayed plants, making it unsuitable for use on ornamentals; and (6) a temporary retardation of photosynthesis in sprayed leaves [14].

Bordeaux mixture is ordinarily prepared from the mixture of solutions of copper sulphate and either quicklime or hydrated lime. The latter is available commercially in a more finely divided form of greater purity as a rule than quicklime and has advantages in ease of preparation of the mixture. Air-slaked quicklime is not suitable. In making the mixture, the copper sulphate is dissolved in a part of the water in a vessel not liable to injury by copper such as a wooden tub. The lime may be similarly dissolved in the rest of the water, slaking it gradually if quicklime is used or making it first into a thin paste if in the hydrated form, and the two solutions may be poured simultaneously into a third receptacle. Alternatively, a concentrated stock solution of copper sulphate may be added to the diluted lime solution, a single tank being sufficient in this case. In the 'instant' process the copper sulphate is dissolved in one-quarter of the required volume of water and, while stirring, water is added to the three-quarter mark to secure full solution; then the lime is added in a thin paste and the tank made up with the balance of the water. An agitator should always be fitted in the tank to keep the mixture stirred during making and when supplying to the spraying apparatus. Modifications of these methods of preparation are used in different places, the chief object to avoid being the mixing of a concentrated copper sulphate with a concentrated lime solution.

Bordeaux mixture is used in various degrees of concentration, the tendency having been steadily towards reduction of the high concentration of copper sulphate at first recommended. It is customary in the English-speaking countries to express the concentration in a formula with the copper sulphate first, then the lime, both in lb., and lastly the water in gallons. The 6–4–40 mixture thus means one composed of 6 lb. copper sulphate, 4 lb. lime, and 40 gallons water, or assuming the imperial gallon of water to weigh ten pounds, the mixture contains $1\frac{1}{2}$ lb. copper sulphate and 1 lb. lime in each 100 lb., or $1\frac{1}{2}$ per cent. copper sulphate by weight. In countries following the metric system kilogrammes and litres replace lb. and gallons. As a litre of water weighs a kilogramme, a Bordeaux mixture made with 1 kg. copper sulphate to each 100 litre water contains 1 per cent. copper sulphate. Continental usage, therefore, often gives only the percentage strength of the mixture. As the standard American gallon is smaller than the British imperial gallon and contains only approximately 8·3 lb. water, the concentrations indicated are not the same in the two countries.

The earlier use of excessive proportions of copper to lime did not persist, and for a considerable period the tendency was to use equal amounts, the 4–4–40, 5–5–50 (or 1 per cent.) mixtures long remaining standard general-purpose sprays. The movement still further to reduce the proportion of copper sulphate to lime, however, has continued, especially for use on apple and other fruit trees; three or four times as much lime as copper sulphate is recommended in Nova Scotia and New York against apple scab, and one and a half times against various fruit diseases at East Malling. These trends have been mainly dictated by the desire to find mixtures which will not injure the trees.

With this growing consciousness of the phytocidal action of Bordeaux mixture, great efforts have been made to find a safe copper-containing substitute. Laboratory methods of testing fungicidal efficiency have been perfected and some of the most important properties, as determined, for instance, at Long Ashton, have been found to correspond satisfactorily with those deduced from field trials. Proprietary copper preparations have been developed extensively, especially in the United States and Germany. Even though these are not usually equal as fungicides in field practice to Bordeaux mixture, they often give adequate commercial control, especially when used as supplements in the later applications; the best practice in the vineyards of southern Europe and North Africa now includes dusting with preparations of this kind when the bunches begin to form. Furthermore, as with the sulphurs used in place of lime-sulphur, various proprietary copper preparations find a place when phytocidal injury becomes pronounced. A copper-lime dust in the proportions 20 to 80 has been widely used in North America against potato, tomato, and other diseases but has rather gone out of favour in recent years. Proprietary materials for making liquid copper-containing sprays have become much more extensively used than formerly, but where phytocidal action is not important none has shown evidence of replacing Bordeaux mixture. Cuprous oxide in dust or spray form has been much recommended and has given excellent results against various diseases in America and the British Empire, but on the whole Bordeaux mixture seems to be generally preferred for large-scale work; various proprietary brands of cuprous oxide of the highest standard, such as perenox, sulfocide, etc., are available. Colloidal copper also has its advocates and has sometimes given satisfactory results, especially when staining the foliage has to be avoided. The colloid-like copper preparation 'bouisol' is recommended against *Phytophthora infestans* on glass-house tomatoes by the Cheshunt Research Stations and has won a place for itself in the control of various other diseases of the green parts of plants.

Amongst other copper spray fluids the best known is Burgundy mixture, introduced in 1887 in Burgundy in place of Bordeaux mixture. It is made up like the latter, except that lime is replaced by sodium carbonate crystals which, at that time, were often more easily obtained than good quicklime. A standard formula much used on potatoes in Ireland is 10–12½–50 (2 per cent.), but equal parts of copper sulphate and crystallised sodium carbonate are said to be quite satisfactory. Now that good-quality hydrated or quick lime for spraying is usually easy to get, Burgundy mixture has lost much of its attractions of availability, freedom from grit, ease of storage and preparation; and as it is slightly less effective than Bordeaux, more expensive, and more likely to cause injury on susceptible plants, its use outside Ireland is limited.

Another copper substitute for Bordeaux mixture is the *eau céleste* or cuprammonium spray first applied in French vineyards against *Plasmopara viticola* in 1886. In preparing this, copper carbonate is dissolved in strong ammonia in the proportions of 5 oz. to 3 pints and made up to 50 gallons with water. The preparation is liable to scorch the foliage, is inferior as a fungicide to Bordeaux mixture, and is more expensive than the latter but can be used on ornamental plants or ripening fruits, as it causes little staining of the foliage or fruit. The latter

advantage, however, can be better secured with copper acetate sprays. Copper acetate (*verdet*) has been used for fifty years against vine mildew in some parts of Europe, the basic salt being termed *verdet gris* (verdigris) in France. Both the basic and neutral salt are used. They stain rather less than cuprammonium, cause little injury to grape bunches or foliage in dry weather, and the basic salt adheres well. The 1 per cent. solution is considered in Europe to be equal to 2 per cent. Bordeaux mixture as a fungicide against vine mildew, while for a late spray on the grape bunches 0·5 per cent. has been recommended in the United States and 0·25 per cent. for ornamental or delicate plants.

Fungicides containing sulphur or copper are frequently combined with insecticides. Lime-sulphur by itself has valuable properties in destroying red spider on orchard fruits, and allied mites as well as scale insects on other plants; the addition to it of lead arsenate or lime arsenate materially increases both the fungicidal and insecticidal action of the spray. Bordeaux mixture has little effect on insects. It (but not Burgundy mixture) can be used safely with lead arsenate on plants not liable to injury from summer applications of Bordeaux. Acid lead arsenate and lime arsenate, however, are liable to cause leaf scorching and fruit russeting when water-soluble arsenic forms in the deposit. Nicotine may be safely combined with either lime-sulphur or Bordeaux or Burgundy mixtures for use as a contact insecticide. Petroleum oils are used with Bordeaux mixture to form combined sprays in England [7], and a Bordeaux-oil emulsion is valuable especially in cases when infestation by scale insects is normally kept down by entomogenous fungi as in many citrus groves; the Bordeaux kills the fungi without harming the insects and, unless oil is added or a supplementary application with oil is made, insect injury may become serious. Cuprous oxide is free from this disadvantage.

The Search for Sulphur and Copper Substitutes

Leaving the firm ground provided by the sulphur and copper preparations, one enters a vast field in which testing and research into the fungicidal properties of other inorganic and organic materials for use as sprays or dusts on the green parts of plants have been carried out and are still proceeding. Excluding seed and soil disinfectants, little has been found that has more than a limited application against specific diseases. Amongst metals, compounds of zinc and nickel have been especially tested, but only the former find a real use in the eradication of the branch and twig cankers in which certain fruit-tree pathogens such as *Bacterium amylovorum* and *Xanthomonas pruni* over-winter; when zinc sulphate is used as a spray to counteract zinc deficiency diseases such as citrus mottle leaf and 'little leaf' of stone fruit, it has been found to act also as an effective fungicide against citrus brown rot caused by various species of *Phytophthora* and the peach and plum rust, *Puccinia pruni-spinosae*, a 10–5–50 zinc sulphate lime mixture proving equal to Bordeaux mixture against peach rust in South Africa.

A long series of investigations on the possibility of controlling plant diseases by means of metallic salts absorbed in small amounts through the roots has not yielded results of practical significance. Perhaps the most interesting have been those already mentioned in an earlier chapter (p. 178) which demonstrate that

lithium salts applied to the soil at low concentrations have a more or less marked effect in reducing the susceptibility of celery to *Septoria*, wheat to powdery mildew, and tomato to crown gall. To secure a similar effect on wheat brown rust, however, it was necessary to use a concentration which caused injury. The application of silicic acid to the soil enhances the resistance of rice leaves to blast (*Piricularia oryzae*) by increasing silicification.

Only a few of the many proprietary and other organic spraying and dusting materials that have been recommended against particular diseases can be mentioned. Very favourable results have been obtained with folosan dust (pentachloronitrobenzene) against *Botrytis cinerea* on lettuce, used on or in the top soil of the frames and on the seedlings; the dusting also reduced lettuce mildew (*Bremia lactucae*) and damping-off caused by *Corticium solani*. Turf diseases (Part II, p. 479) caused by *Corticium fuciforme* and *Calonectria graminicola* on golf and bowling greens in Great Britain have been satisfactorily controlled by malachite green applied at a strength of 1 in 10,000 in dilute Bordeaux mixture once a week; for those due to *Corticium solani* and *Sclerotinia homoeocarpa*, recent experiments in the United States show that tetramethyl thiuramdisulphide gives complete control in weekly applications of 4 oz. per 1,000 sq. ft., the dust being mixed with sand for strewing. A form of sodium dinitrocresylate has been widely recommended in the United States under the name of elgetol as an eradicant or dormant fungicide against various fruit pathogens. The salicylanilide preparation known as shirlan, first used in England against moulds on cotton fabrics, has found a wide use as a fungicide against the powdery mildews, tomato leaf mould (*Cladosporium fulvum*), and the like. Shirlan A.G., the brand chiefly used, contains 25 per cent. salicylanilide with agral as a wetting agent. Many alkaloids, aromatic compounds, nitrogenous bases, and so forth have been tested against fungi such as *Phymatotrichum omnivorum*, the cause of Texas root rot of cotton. It would appear that 8-hydroxyquinoline is the most effective fungicide yet discovered against this parasite.

Commercial interests can usually be relied on to keep the claims of fungicides of this nature from being overlooked. Many of them are the result of prolonged experimentation and may find a place in nursery or market-garden practice. It is, however, a wise rule to consult the advisory services as to their value and to test them at first on a small scale before their extended use is attempted. Recently a scheme has been agreed on between the agricultural departments in Great Britain and the representatives of the insecticide and fungicide manufacturers under which official approval can be given to tested proprietary preparations.

Spraying Apparatus

Great strides have been made in the improvement of spraying machines and accessories in recent years. They are most marked in the appliances designed for use in tree fruit orchards and on permanent plantation crops. In the larger fruit-growing districts, especially where the produce is exported, orchard practice has reached or is rapidly reaching the point where spraying for the control of diseases and pests is accepted as part of the normal orchard routine. The small grower is still obliged to rely on hand machines — knapsack and stirrup pumps,



FIG. 157.—Potato spraying, for large areas. Note that the vertical rods between the drills carry the lower nozzles sufficiently low to spray the underside of the foliage, the upper nozzles spraying the tops of the plants (by courtesy of Bayer Products Ltd.)

compressed-air machines, battery outfits, and the like. Many excellent makes are available, the suitability of which for local conditions has been well demonstrated. Information regarding them is readily obtained by the grower and need not be detailed here, the more so as they are subject to constant modification and improvement. For medium and large-scale work, however, the tendency in favour of high-pressure spraying has been steadily growing, and for this purpose the use of power machinery is essential. Power spraying has been especially developed in the United States, Russia, and (for special uses) in other countries, including Great Britain and the British Dominions (Figs. 157-62).

Power spraying may be carried out either with independent power units which use motor power both for the transport of the spraying outfit through the plantation or crop and for delivering the spray fluids through the nozzles, sometimes at pressures of three or four hundred pounds; units capable of spraying through multiple nozzles up to 14 rows of potatoes at a time are in use in the eastern counties. The next step is the replacement of these mobile units by centrally operated plants supplying a permanent series of pipe-lines throughout the plantation. Such stationary spray equipment has been found greatly to reduce the cost of spraying, especially once the capital charges have been written off. Not only is the cost reduced, sometimes by as much as one-half, but working is facilitated and it is possible to carry out effective spraying where this had previously been very difficult [16, 17].



FIG. 158.—The portable 'knapsack' sprayer

A good example of modern practice is given by the very considerable campaign, now being carried out against leaf spot of the banana (*Cercospora musae*) in the Caribbean area. It was recognised soon after the first appear-

ance of the disease in this region in 1934 that the future success of the industry depended upon its control, and a very large expenditure for the purpose has been agreed upon by the Government of Jamaica and the principal fruit companies on the mainland and throughout the affected islands. Tests determined that, on the whole, spraying with Bordeaux mixture was more effective and cheaper than dusting or spraying with preparations of cuprous oxide and the like, and that control was satisfactory if the plants were treated every two or three weeks. Stationary spraying apparatus was at once installed on a huge scale, including power houses, spray-mixing tanks, pumps, and a permanent pipe-line system throughout the plantations, leading to stopcock points to which the actual spraying hoses could be attached. A suitable area for a central unit was found to be 700 or 800 acres, and the scale of the work may be judged from the fact that some 60 square miles on the Ullua River, Honduras, and over 100,000 acres in Central America were under permanent stationary spray plant control in 1941. The pipes are laid upon the ground (in orchard practice they are often buried underground or elevated above ground-level) and the plants (which grow unusually luxuriantly in Honduras) require spraying to a height of 30 to 40 feet, which is just below the tops of the highest leaves.

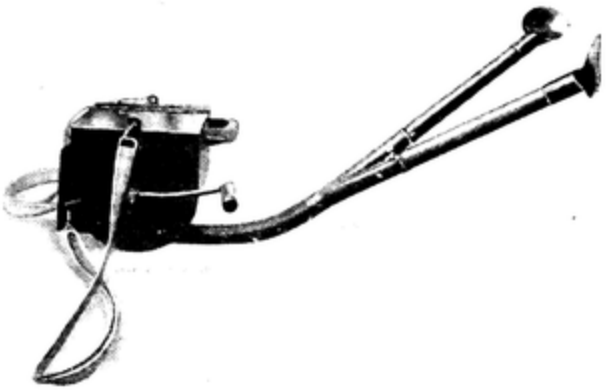


FIG. 159.—The 'Armada' dust gun, Model 2, with flexible tube, lances, and spreaders, for distributing a continuous cloud of powder. It is carried from the shoulder by means of straps, and the fan is worked by a crank handle (by courtesy of Drake & Fletcher Ltd., Maidstone)



FIG. 160.—Power orchard duster. A powerful high-pressure engine-driven powder distributor for use in hops, fruit, citrus, etc. (by courtesy of Drake & Fletcher Ltd., Maidstone)

Fumigation

Fumigation, which has proved a valuable method of applying some insecticides, has been little used against fungi except in glasshouses; sulphur and formalin are sometimes vaporised in warm houses for the control of various leaf mildews and moulds, but treatment by spraying or dusting has largely replaced this practice. A gas treatment, however, has been found the most effective method of controlling the blue mould of tobacco due to *Peronospora tabacina* in Australia and North America. Benzol and paradichlorbenzene are chiefly used, the application being made as soon as the first sign of infection is observed in any of the seed beds, and repeated every three or four days until five or ten have been given. The seed beds should be covered with cloth or the like and the benzol used at night at a rate of 1 sq. inch evaporating surface to 1 sq. foot of bed, or paradichlorbenzene crystals strewn on the cloth at 2 to 3 lb. per 100 sq. yards. The seed beds are opened up in the morning to allow free ventilation.

Soil Sterilisation

A good fungicide for use in the soil is much needed. It should have many of the physical properties of a good fumigant, for it must be able to diffuse into the soil interstices at lethal concentration. It must not be expensive, as considerable quantities have to be used for admixture with even the upper few inches of the soil both to secure reasonably even distribution and to escape the fixing or destructive action of many soils on chemical compounds added to them. It must be reasonably quick in action, because a prolonged sterilisation of the soil might easily do more harm than good by interfering with the normal microbiological activity on

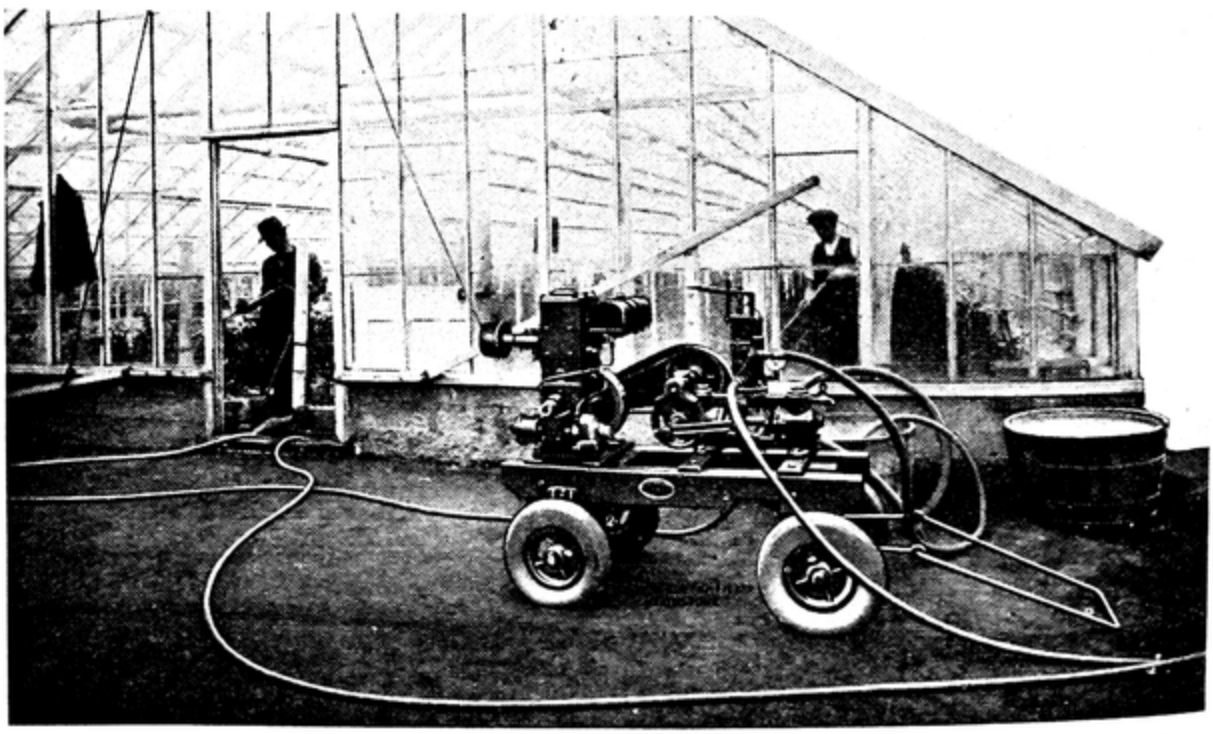


FIG. 161.—Power spraying plant, for working under glass in nurseries. Capacity of 260 gallons per hour; pressure, 300 lb. per square inch (by courtesy of Drake & Fletcher Ltd., Maidstone)

which soil fertility so largely depends. Loss from the surface of the soil is likely to be so high as seriously to reduce the possibility of maintaining a lethal concentration in the upper layers, where it is most needed, except when the soil can be covered. These considerations, and especially the last, tend to limit the use of soil disinfectants to glasshouses, seed beds, and the like, and up to the present time no general purpose soil fungicide has been found that is as affective as heat.

Soil disinfection by the application of steam heat is a common practice in glasshouse cultivation and is often used also in nurseries for seed beds and potting soils. The steam is applied either in bins before the soil is placed in the beds or by inverted pans or perforated pipes delivering live steam to the soil *in situ*. Dry heat is also sometimes used but is definitely inferior to steaming as it spoils the physical texture of the soil and carries a greater risk of injury from overheating. For damping-off in seed beds and the like it is usually ample to provide for sterilisation to a depth of 9 inches, but for some of the wilt-inducing species of *Fusarium* and other soil pathogens a depth of at least double this should be reached. A temperature of 180° F. is adequate against most resistant fungal sclerotia and the like, but in order to secure this at a distance from the steam orifices it is desirable to raise the temperature at the surface of the soil to about 200° to 210° F. and keep it at that point for 15 to 20 minutes.

Chemical soil disinfection often results to some extent from the use of seed disinfectant dusts and the like, as these not only destroy fungal contaminants on the sown seed but form a fungicidal zone around the seed coats and the first-formed organs of germination. Where heat cannot be used, chemical soil disinfection of seed bed and potting soils is often practised. Formaldehyde is the most efficient of the chemicals used for this purpose, as it is a powerful fungicide and penetrates the soil readily both as a liquid in the soil solution and as a gas in the air spaces. It leaves no deleterious residue in the soil, but has no residual protective effect and at the strengths used is injurious to growing plants, so should not be applied once germination has occurred. Dry soils may require 1 gallon of 1 in 100 solution per sq. foot, and wet, half a gallon of 1 in 50 solution, the beds being kept covered for two or three days after treatment. It is a safe rule not to set seed in the treated soil for 14 days after treatment. A special application of formalin is used against onion smut to prevent infection from spores in the soil; a solution of one part of commercial formalin in 128 parts of water is dripped from a tank attached to the seed drill so as to moisten the seed and surrounding soil. The amount of solution used varies with the wetness of the soil from 27 to 130 gallons per acre (5,000 feet of drill), the seed rate being increased to allow for a certain amount of seed injury. Good control of damping-off of tomato seedlings due to various phycomycetes and to *Corticium solani* has been experimentally secured by the incorporation in infested soil of 15 parts of formalin with 85 parts of an inert absorbant such as sawdust or kaolin at the rate of 4 or 5 parts per 1,000 of soil to a depth of a few inches. Sulphuric acid is a favourite remedy for damping-off in forest-tree nurseries. At a strength of 2 oz. of the concentrated acid per gallon of water it has proved superior to all the other chemicals tested for control of *Corticium solani* and *Botrytis cinerea* on seedlings of Sitka spruce and Douglas fir in Northern Ireland. Corrosive sublimate is probably the best soil disinfectant

against club-root, about one-quarter to one-half a pint of a 1 in 2,000 solution being poured into the dibble holes when transplanting into infested soil. Cheshunt Compound, devised at the Cheshunt glasshouse experimental station, has had a wide use in the control of damping-off, and appears to be the best copper-containing compound for the purpose. It is prepared by mixing two parts of powdered copper sulphate with 11 parts of powdered ammonium carbonate and dissolving an ounce of the mixture in two gallons of water, wetting the soil thoroughly with the solution. It is efficient not only against damping-off fungi but also against those causing vascular wilts and various root rots, and does not injure growing plants. Where chloropicrin vapour is used as an insecticide or anthelminthic it has sometimes been found also to possess fungicidal properties. Thus, it has given effective control of damping-off of tomato seedlings and of the tomato wilt due to *Fusarium bulbigenum* var. *lycopersici* in Texas in heavily infested soil, and of black root rot of tobacco (*Thielaviopsis basicola*) at Rostov in the U.S.S.R.

Seed Disinfection

The disinfection of seeds for the control of seed-borne diseases is the most successful of the many applications of chemotherapy in plant pathology. Not only is there a wide range of chemicals available for the purpose, to which frequent additions are made, but the degree of control achieved is often astonishingly high.

Great Britain does not make use of seed disinfectants as freely as some other countries, though official seed-testing stations have been established which undertake the routine examination of cereal and other seed samples for impurities, as well as recording the incidence of seed-borne diseases in general. In Germany, for instance, there is an elaborate system of State testing of fungicides for use on seeds and the like : new remedies may be submitted for testing (with a confidential disclosure of their active ingredients) to a central organisation which, if a *prima facie* case is made out, arranges for extensive regional tests ; lists are then published by the German Phytopathological Service of the purpose for which the disinfectant may be recommended, but not of its composition. A similar system is in force in several other countries, including Denmark and New Zealand, in some of which testing is not restricted to seed disinfectants but covers spray fluids, dusts, and the like. In the United States, Federal and State experiment stations recommend approved materials, and official analyses of these are published from time to time ; as with all therapeutants, the nature and percentage of the active ingredients have to be stated on the label.

A very high proportion of the routine seed-dressing with fungicides is carried out on cereal seed grain. In Holland and parts of the United States farmers can have their seed grain disinfected by contract, and arrangements have been made by which the dusting apparatus is mounted on a motor car or motor cycle trailer and taken round from farm to farm, the seed being treated at a fixed rate per sack. In England the best seedsmen are prepared to supply ready-dressed grain, and it was estimated that in 1938 some 260 centres were in existence for the supply of grain treated by organo-mercury compounds. In Germany special adjustments for dressing the seed are attached to many of the co-operative seed-cleaning plants, or worked by co-operative groups (' rings ') of small-holders.

In 1937 it was reported that large-scale co-operative seed-disinfecting plants numbered 348 in Hanover, 233 in Westphalia and 185 in Schleswig-Holstein, and that Germany was using at least 800 tons of mercurial dusts and 180 tons of liquid mercurial preparations for seed treatment annually. In one co-operative district of Saxony, virtually complete control of wheat bunt, oat smut, barley stripe, and 'Fusarium disease' or 'snow mould' of rye (*Calonectria graminicola*) had been secured by the 1,000 members of the co-operative union by 1932, when 84 per cent. were disinfecting their cereal seed. Co-operative seed-dusting centres have been developed also in America. Almost the only attempt on a comparable scale to combat a seed-borne cereal disease in Great Britain is that which has been applied with a considerable measure of success in Scotland against the leaf spot and seedling blight of oats due to *Helminthosporium avenae* (Fig. 163). As much as 30 per cent. of the seedlings were found to be infected in a large series of examinations in south-west Scotland; seed treatment with an organic mercury dust gave effective control, besides admitting of considerable economy in seed rate. In a recent estimate of the effect of this treatment it is considered that it saved from 40,000 to 63,000 tons of grain in a crop of 633,000 tons in 1938. Satisfactory results from similar treatment have been reported from Northern Ireland and Eire. Taken as a whole, however, the disinfection of cereal seed grain has had its maximum benefit in controlling the bunts of wheat and smuts of oats. These diseases are now seldom of major importance in the more advanced countries, whereas the damage they can do in more backward areas, such as the North-West Frontier of India, has to be seen to be believed. Another cereal smut that can be practically eliminated by seed disinfection is the common smut of sorghum, *Sphacelotheca sorghi*, which was estimated to be responsible for a loss of about £1,000,000 annually in Bombay before treatment was undertaken.

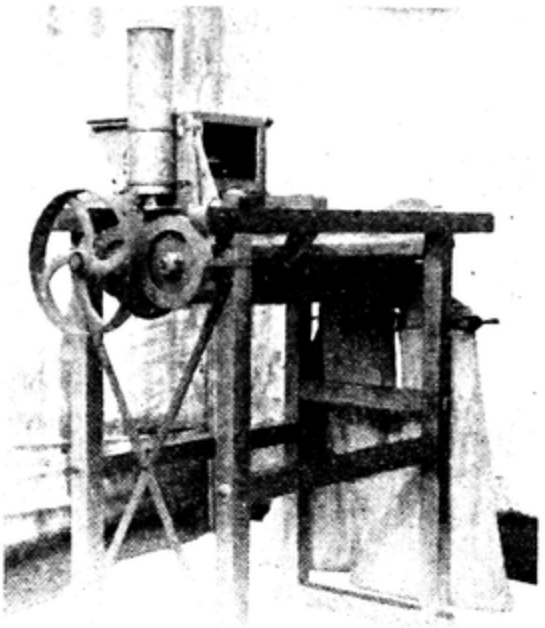


FIG. 162.—Strickland's 'Ceresan' dry seed dresser. The patent powder feed sprays the seed as it passes over a platform, distributing the powder over it before it falls into the mixing drum, the flow being variable to give the correct dose to any given output of corn (Grain Cleaners Ltd. By courtesy of Bayer Products Ltd.)

Although cereal seed grain is the chief subject of seed disinfection, various large-scale applications of the process to other seeds have been used. Thus, it is customary to treat the seed required for sowing the cotton fields of the Gezira, Sudan, where about 200,000 acres are annually grown, against seed-borne infection by *Xanthomonas malvacearum*. The proprietary compound 'abavit B' (mercuric iodide) was used for a time for this purpose, but more recently the best



FIG. 163.—Control of Helminthosporium disease of oats, with mercurial dry seed dressing: top, treated; bottom, untreated grain (photo by courtesy of West Scot. Agric. Coll., Plant Husbandry Dept., Auchincruive)

results have been given by two hours' immersion of the undelinted seed in 1-in-500 solution of mercuric chloride-iodide followed by drying in the sun. In this case, internal infection of the seed has been established in a certain percentage of seeds, so that surface disinfection is not fully efficacious [15]. A still more massive cotton-seed disinfection campaign is carried out in North Carolina, where it was estimated that seed for 600,000 acres was dressed, mainly with ceresan dust, in 1939. A high proportion of the sugar-beet seed used on the Continent is dressed against damping-off and other seedling diseases, and much of that imported from abroad for use in Great Britain in normal times was similarly dressed before importation. Another crop which suffers severely in some areas from seed-borne parasites is flax. The flax diseases due to *Colletotrichum linicola* and *Polyspora lini* have been satisfactorily controlled in Northern Ireland by tetramethyl thiuram-disulphide dust, and over 2,000 tons of flax seed were treated with a preparation of this, known as 'nomersan', in 1941, the rate of application being about 5 oz. per bushel of 56 lb. Peas, beans, celery, cabbage, and other vegetable seeds have been found amply to repay disinfection under certain conditions, and further references o seed treatment will be found under these crops in later pages.

Another great group of diseases which can be controlled by similar methods are those which are commonly carried on seed potato tubers and other plant parts used in vegetative propagation. Steeping in formalin at the rate of 1 pint in 40 galls. water (1 in 320) or 1 pint in 60 galls. (1 in 480) or in mercuric chloride (1-in-1,000 or 1-in-2,000 solution) is useful against the common and powdery scabs and skin spot of potatoes and certain diseases carried on flower bulbs.

Mercuric chloride is more effective than formalin, but it is liable to injure seed potato eyes and sprouts, and is, of course, very poisonous; in New Zealand and Australia it is claimed that acidification with 1 per cent. hydrochloric acid shortens the period of immersion and increases the efficiency of mercuric chloride. In recent tests in the United States acidification by 0·55 per cent. hydrochloric acid or 1·66 per cent. acetic acid, by volume, notably simplified and improved the mercuric chloride tuber treatment. Treatment of these diseases with organo-mercury steeps or dusts appears likely to supplant the older methods, as good results have been obtained in Northern Ireland, Holland, and elsewhere with aretan, ceresan, uspulun, and the like, if applied soon after lifting.

It is probable that seed disinfection marks the earliest attempt at chemo-therapeutical control of plant diseases, for the use of copper sulphate for the purpose dates from about the middle of the eighteenth century. Copper sulphate remained for many years the standard method of 'pickling' cereal seed grain against smuts and the like. Later, formalin came into general use as being at least equally effective and much less liable to depress the germination energy of the seed. Copper carbonate dust was strongly supported in Australia and the west of the United States, but has never found favour in Great Britain, as it appears to do best in drier seed beds than are common in this country. Cuprous oxide has been recommended especially for dressing vegetable seeds, and has given very good results against pre-emergence damping-off of peas in tests at Long Ashton and in practice in the United States. All these and many other chemicals are still used in various parts of the world, but in the more advanced countries the tendency is to replace them by organo-mercury dusts, of which a large range is now available for use on vegetable seeds as well as on cereal seed grain. These dusts are easy to apply, generally harmless at the prescribed dose to the seed, which can be stored without injury for a long time after treatment if kept dry and not overdosed; they are deterrent to rats and sometimes to insects; and they often have a beneficial effect on the seedlings, which appears to be due to their protective action against soil-borne parasites. Most of the dusts in use are proprietary preparations marketed by the large chemical combines. The names given them differ sometimes in different countries and it is, therefore, not always possible to define the nature of the active compound. Mercury chloro- and nitro-phenyl, mercury ethyl acetate, tartrate, chloride and phosphate, mercury tolyl acetate, mercury cresol sodium cyanide, are amongst those widely used in various countries. Of the 13 preparations officially recommended for cereal seed grain disinfection in Germany in 1937, 12 were mercurial dusts and steeps, and only formaldehyde, to be used against *Ustilago avenae*, contained none. In recommending dry mercurial dusts for this purpose in England, the Ministry of Agriculture put the cost in 1941 at 10d. to 1s. 3d. per acre.

Certain seed-borne diseases cannot effectively be eliminated by surface treatment with fungicides, because the pathogen is within the tissues of the seed coats or still farther in. The standard examples of these are the loose smuts of wheat and barley, against which the methods of disinfection discussed above are worthless. For these and certain internally borne diseases of plants propagated from setts, tubers, corms, bulbs, and the like, the only satisfactory method of sterilisation

is by heat. Heat is usually applied as hot water, the methods adopted against the loose smuts being described in the account of these diseases in subsequent pages (Fig. 191). Hot water seems also to be the most effective treatment against primary infection with *Phoma lingam* on swedes and turnips, and is recommended against cabbage black rot (*Xanthomonas campestris*) in various countries. It has the great disadvantage that the margin of safety between the temperature and time required for effective sterilisation, and those liable to injure the seed is generally small, so that the method is not very suitable for general farm practice. Against the obscure sugar-cane disease known as chlorotic streak and attributed in Hawaii to a Chytridiaceous fungus, hot-water treatment of the setts is the only remedy known, a treatment at 52° C. for 20 minutes being recommended. The runners of mint (*Mentha villoso-nervata*) propagation stocks have been effectively freed from rust (*Puccinia menthae*) at Long Ashton by immersing them in water at 105° to 115° F. for ten minutes in mid-February [10].

Wound Protection and Disinfection

In orchard and garden practice and in the care of ornamental and shade trees pruning occupies a very important place in the seasonal routine. It is still more important in tea planting, where it is a most valuable means of securing the desired succession of flushes of the young and tender leafy shoots from which the tea is made. In rubber trees care of the tapping cut is a major consideration of the planter in those areas where diseases of the tapping panel occur, that is, in most of the chief rubber-growing countries. In all these cases there is grave risk of rotting of the exposed bast or wood from a host of parasitic fungi which are normally incapable of penetrating the intact cork of the bark or which sometimes require access to the dead heartwood of the bush or tree before they can cause injury.

In most cases it is sufficient to provide the pruning wound with a cover which will retain its continuity until the healing processes beneath it are sufficiently advanced to reconstitute the corky barrier. For this purpose soft grafting wax made by melting 8 parts of resin, 4 of beeswax, and 1 of tallow, or a white lead paint consisting of 2 lb. white lead paste, two teaspoonfuls each paste drier and linseed oil, and one-quarter of a pint turpentine, has proved satisfactory in preserving plum and other trees from invasion of *Stereum purpureum*. Coal tar, commercial creosote, and asphaltum are other substances much used for similar purposes.

When, however, the cut surface is liable to be contaminated with disease-producing organisms as in the eradication of fruit-tree cankers and the preservation of the rubber-tapping surface from panel diseases, disinfection must precede covering. For this purpose the high-boiling coal-tar carbolineums have been much advocated, but they are very variable in composition and have to be used with caution. Zinc sulphate has already been mentioned as effective against hold-over cankers in fruit trees. A mixture of equal parts of mercuric chloride and mercuric cyanide has been much used against fireblight cankers in the United States, where the exudate from them in the spring is carried by the wind or by flies or rain splashing to the first blossoms and other unprotected parts [2]. In Malaya extensive testing of disinfectants against mouldy rot (*Ceratostomella fimbriata*) led to the preparation of a list of officially recommended disinfectants,

those most commonly used being coal-tar derivatives emulsifiable with water alone (agrisol, brunolinum plantarium, and izal). Three applications are made at intervals of 10 days and the treated surface is not usually covered. In severe attacks or during wet weather daily treatment may be necessary for about 12 days, followed by treatment every 5 to 7 days. Bordeaux paste is extensively used in some orchards for the disinfection of stem cankers, and the most promising protectant against wound infection from apple canker in tests at Long Ashton was a paste made with 5 gm. monohydrated copper sulphate, 10 gm. hydrated lime, and 9 c.c. linseed oil.

(*d*) Curative Measures against an Established Parasite

A great many, indeed most, of the protective measures against parasitic attack that have been mentioned above fulfil a dual purpose by destroying existing parasites on the surfaces of the plant or seed as well as preventing infection from parasites newly reaching these surfaces. Heat therapy has only the former effect, and many of the chemical seed disinfectants also act primarily in this manner. It is often almost impossible in practice to separate the curative from the protective aspect of the therapeutant; furthermore, so many variable factors are involved in determining the efficacy of a particular fungicide that it has proved very difficult to devise a standard measure of evaluation of protective or curative activity, the more so since many of the preparations used are proprietary and their exact composition not easy to ascertain. The attempt made to find a quantitative measure by determining the ' chemotherapeutical index ', that is to say, the figure obtained by dividing the *dosis curativa* or minimum lethal concentration of the fungicide for a given pathogen by the *dosis toxica* or concentration at which incipient injury to the host plant is observed, has proved a failure in field practice and has been largely abandoned. At present there is no really satisfactory alternative to the laborious system of field testing under as great a variety of conditions as possible, though there is much work in progress to develop laboratory methods. Until these have been improved, the attempt to distinguish curative from protective applications may present insuperable difficulties.

Fruit blemishes due to apple and citrus sooty blotch (*Gloeodes pomigena*) and apple fly speck (*Leptothyrium pomi*), and similar superficial fungi, may be removed after immersion of the fruit in bleaching powder and sodium bicarbonate or in calcium hypochlorite alone, rinsing in water, and wiping with a cloth. The washing of citrus and other fruits in antiseptic solutions to prevent wastage in transit from moulds whose spores may be present on the surface is a common practice. Borax and sodium bicarbonate have proved satisfactory against the species of *Penicillium* which are the cause of so much transit wastage. Though borax is not permitted by the food preservation laws in England and various other countries, it was largely used on citrus fruits for this purpose until gas treatment with nitrogen trichloride, the use of diphenyl-treated wrappers, and other methods supplanted it. Wastage due to *Penicillium* and *Botrytis cinerea* in exported Ohanez grapes has been experimentally controlled in Western Australia by mixing potassium metabisulphite with the granulated cork used for

packing. The liberation of sulphur dioxide from alum and sodium bisulphite tablets has had a similar effect in South Africa. Much work is in progress at the low-temperature research stations at Cambridge, Cape Town, and Trinidad on the control of fungal wastage of fruits in transit by the use of impregnated wrappers, oil dips, and the like, but it is mostly still experimental.

Surgical treatment for the cure of fruit cankers has already been mentioned; for various stem and root diseases of permanent woody crops it has also been undertaken on a large scale in rubber and coconut plantations and the like. In rubber the standard treatment against root diseases is based on a tree-to-tree examination of the collar and larger roots, extending outwards from cases of incipient attacks of the disease. If the mycelium is still superficial or penetration is slight the root is scraped clean and drenched with 2 per cent. copper sulphate. If infection is deeper, the root is amputated and the wound dressed. The root stump from which infection originated is traced and destroyed, all infected jungle timber removed from the diseased patch, and the limits of the infected area demarcated by isolation trenches. Somewhat similar methods are used against the honey agaric fungus (*Armillaria mellea*) on the roots of citrus and deciduous fruit trees in California, Australia, and other countries, infected trees being saved by surgical methods if not too far advanced, spread being checked by trenching, and all sources of infection removed. The attempted biological control of this fungus in Nyasaland has already been discussed. In Great Britain these root diseases have not hitherto been found under conditions necessitating the application of similar control measures.

MEASURES DIRECTED TO INCREASING THE RESISTANCE OF THE HOST

Some of these depend on inherent characters and are made use of in the production of crop varieties resistant to particular diseases. Others depend on external conditions and vary from place to place, from year to year, and with different agricultural practices.

Breeding Disease-resistant Plants

The breeding or selection of resistant varieties of the host is perhaps the most powerful weapon available in the fight against crop diseases. Resistant varieties against many of the major diseases are now grown in crops such as the cereals, potatoes, beans, peas, cabbage, turnip, beet, tomato, cucumber, celery, cotton, sugar-cane, and so forth. Unconscious selection of disease-resistant varieties of staple crops has certainly been operative against many of the commoner diseases over a period of centuries, but controlled rational efforts in this direction have only been in progress for little over a generation. Success has not been easily reached, and the hopes aroused in the first flush of enthusiasm at the discovery of the laws governing the inheritance of the characters of resistance and susceptibility to certain diseases have not been completely fulfilled. Not only is it difficult to couple resistance to a disease with the desired quality of the crop, but

far more serious complications are caused by the presence of numerous physiological forms of the parasite and the disquieting frequency with which new ones appear. Resistance to these may not be possessed by the varieties already available and may be separately inherited from that to the forms previously known. As an instance of this, the greatly increased prevalence of race '56' of *Puccinia graminis tritici* in the United States and Canada during the last few years has spoiled the prospects of some very satisfactory wheats (e.g. Ceres) that had been bred for rust resistance [6]. Furthermore, the increasing recognition of the complexity of the factors governing resistance to many diseases, and the fact that resistance obtained by hybridisation may be governed by entirely different factors according to the variety of the host that serves as a parent, have damped still more the earlier ardour of the advocates of this method of disease control.

Nevertheless, the use of resistant varieties is the only satisfactory way in which control of a great many diseases may be hoped for, and there are sufficient successes to the credit of the plant-breeders to justify the claim that it remains the best method against a host of diseases. Examples are separately given of the control of certain virus diseases in this way, and the list can be expanded many times when bacterial and fungal diseases are included. To mention only a few, the production of wart-immune potatoes of good quality has removed the menace to potato-growing caused by this disease in a large part of Great Britain. Breeding against club-root of swedes and turnips, a notoriously difficult disease to control, has now reached a stage of great promise [11]. Many of the best wheats now grown in England have been bred for resistance to yellow rust or have this as an important character in their constitution, while over a large part of North America varieties were developed by breeding and selection which withstood the ravages of black rust. With the change in the prevalence of particular physiologic races of this rust, other wheats since produced have come into favour and the process is still going on. Thus, work is at present in progress in Canada on a group of wheats bred in Kenya which have been found to be practically immune from all the physiologic races of *Puccinia graminis* now occurring in Canada. In commenting on the slight damage caused by this rust to wheat in Manitoba in 1939, it was officially stated that 77 per cent. of the wheat acreage was sown in that year with the black rust resistant varieties Thatcher and Renown, and another 18 per cent. with durum wheats, chiefly Mindum. Resistance to rust and bunt and to rust and mildew has been combined in several varieties of wheat, while the Marquillo wheats are resistant to certain insects, such as Hessian fly, in addition, and this resistance is being combined with that to the fungus diseases.

Stewart's bacterial wilt of corn (*Xanthomonas stewarti*) almost completely destroyed the early varieties of sweet maize in a considerable area of the United States, until it was largely overcome by growing new resistant hybrids. As mentioned in an earlier chapter, however, this organism appears to be highly plastic, and growth in resistant host varieties increases its virulence. This is believed to be due to the selection of virulent strains arising by saltation within the host. A similar explanation has been given to account for the fact that passage through President potatoes increases the virulence of a strain of *Phytophthora infestans*, against which this variety is normally resistant.

In breeding for the control of crop diseases, the object may sometimes be attained by breeding for disease escape or disease tolerance, instead of for true resistance. Early varieties may escape a disease, sometimes because meteorological conditions (temperature and so forth) may not be suitable for infection, sometimes because the parasite is not present early in the season in sufficient numbers to cause serious injury. The famous Marquis wheat escaped black rust in the Prairie Provinces of Canada because of its early maturity. The Indian cereal, ragi or kurakkan (*Eleusine coracana*), does not suffer from blast (*Piricularia* sp.) if sown at Coimbatore between October and April, but is heavily infected when sown from June to August; where soil temperature is an important factor in infection, as with the smuts and *Fusarium* wilts, a difference of a fortnight in sowing date may be very important. It may be equally important to aim at increased tolerance of a disease by breeding for particular root characters or for enhanced vigour during the period of exposure to infection. The indications from observation at East Malling that the kind of root-stock on which the scion is worked influences the extent of infection with apple scab and canker, and of silver leaf on plums, have already been mentioned. Comparatively little attention, however, has been paid to these aspects of plant breeding, which nevertheless deserve more consideration than they have received.

The ultimate object of all breeding work is to secure a sufficient range of variability in the crop in question to permit of selection for desirable characters. It may well happen that there is already available in the population of the crop at hand a wide enough range of characters to allow of selection into suitable clonal lines being undertaken immediately. It also happens at times, however, that nothing like the full range of chromosomal equipment possessed by a genus of plants has been accessible to the breeder. This is particularly noticeable in a crop like the potato, where until recently the bulk of the breeding work has been carried out with varieties of *Solanum tuberosum* that are tetraploid, with $2n=48$ chromosomes. Allied diploid, triploid, and hexaploid species of *Solanum* are known, some of which are quite immune from potato blight. The recent collections of these made by Russian, German, and British expeditions to Central and South America have greatly expanded breeding material, though they have not disclosed varieties resistant to *Phytophthora infestans* outside central Mexico. With this new material, potato-breeding in Great Britain, Canada, India, Russia, Germany, and elsewhere has entered on a new phase. The fact that not a single case of true resistance to *Endothia parasitica* has come to the notice of the Bureau of Plant Industry in the United States during twenty-five years of careful recording amongst the native Eastern States chestnut hosts of this fungus, not one of which has escaped attack, may be due to masking (as mentioned in the next paragraph) or to a real absence of factors for resistance amongst the local population of these trees [1]. Interspecific hybridisation may be needed to introduce the wished-for factor. This has been successfully accomplished in a good many cases, as in the transference of the single dominant factor for immunity from tomato *Fusarium* wilt and the factors against most strains of *Cladosporium fulvum* carried by *Lycopersicum pimpinellifolium* to cultivated tomatoes [3, 4].

There is good evidence that a population may have become fairly stable by

self-contained cultivation under more or less uniform conditions and yet contain hidden genes for characters of great importance, the existence of which is too seldom revealed to attract attention. It is in such cases that intervarietal or interspecific hybridisation becomes a valuable means of revealing the potentialities possessed by a heterozygous crop. It is hard to account in any other way for the development of sporadic cases of high resistance to witches' broom (*Marasmius perniciosus*), which began to appear amongst the Nacional and Venezuelan types of cacao in Ecuador, both of which appear to be equally susceptible to the disease, after the latter type had been introduced from Trinidad about 1890 and had become established. No similar cases of resistance have ever been encountered in this type either in Trinidad or on the eastern mainland of South America. The suggestion was made that the locally grown Nacional type carried one or more genes for resistance from its traditional home on the eastern side of the Andes, which had remained masked until crossing with another type revealed them. An expedition sent to the head-waters of the Amazon in 1938 yielded a number of types apparently resistant to the disease, which was found to be widespread in the area, and many of these, and of those that have appeared in Ecuador, are now under test in Trinidad and show considerable promise [12].

While the pathologist must turn for help to the plant-breeder for the solution of many of his problems, the latter not infrequently raises fresh problems for the pathologist. In breeding for improved quality it appears that the character of resistance to certain diseases may be lost. This may be a possible explanation of the recrudescence of the potato dry rot (*Fusarium caeruleum*) in Scotland, where dry rot was formerly a major disease but had diminished to almost negligible proportions in the varieties that became established in the half-century following the appearance of potato blight. An equally great varietal change has resulted from the onset of wart disease, and it may be that amongst some of the varieties now favoured, susceptibility to dry rot has again appeared. Similarly, there is some evidence that in the varieties of sugar-cane bred for resistance to mosaic disease and improved field performance in Louisiana and India susceptibility to red rot (*Colletotrichum falcatum*) has become enhanced, though the more generally accepted explanation is that strains of the fungus showing exceptional virulence have appeared. Many plant-breeders have found it necessary to discard promising varieties because of enhanced virulence in them of some parasite normally of secondary importance, such as ergot in wheat. As against these cases must be set those where the complete control of a disease has followed the elimination of certain susceptible varieties, as has happened with red stripe (*Xanthomonas rubrilineans*) of sugar-cane with the decline in the cultivation of Tip canes in Hawaii.

In the chapter on Resistance and Susceptibility details are given of the genetics of host-plant resistance to disease in a number of cases sufficient to illustrate the complexity of the problem (p. 151).

Enhancing Resistance by Measures other than Breeding

Many examples of this have been mentioned in earlier sections, especially that on the influence of nutritional conditions on plant diseases. It was

pointed out that even when the parasite is one which is favoured by a luxuriant growth of the host, it is sometimes possible to secure a more than compensating increase in growth and assimilatory vigour in the latter by judicious manuring or improved cultivation. But many hopes that have been based on this method of combating disease have proved illusory, and the tale of disappointment which has so often followed the many attempts that have been made to check the progress of some epidemic outbreak of plant disease by improvement in cultivation and the like is a warning against optimism on this score. There seems to be an inherent tendency, not always confined to the so-called ' practical man ', to attribute the failure of a crop from disease to some error in cultivation, but there is abundant evidence that any measure that does not take into account the activities of the parasite concerned is palliative only when judged by its final effect.

MEASURES DIRECTED TO IMPROVING ENVIRONMENTAL CONDITIONS

Some of these have already been discussed where they are specifically directed against the parasite (' field sanitation ') or in favour of the host (' enhanced resistance ' from manuring and the like). Others are of a more general character and affect the disease as a whole without any clear indication as to which partner is most concerned. Even with the most important of these — crop rotation — it is far from certain that the effect is wholly on the parasite, for there is much evidence that ' soil sickness ', as involving a particular crop, is not wholly due to the accumulation of pathogenic organisms in the ' sick ' soil. Hence the subject of crop rotation is included here in spite of the fact that its object often is to free the soil from particular pathogens. Leaving out these wider aspects of crop rotation, for they are outside the scope of this book and the cause of their influence on good husbandry is not always well understood, the practice gives powerful aid to the control of such diseases as the *Fusarium* wilts of flax and other crops, club root of crucifers, and foot and root rots of cereals of the *Ophiobolus graminis* type. The interposition of two or three non-susceptible crops has given effective control of the raspberry wilt due to *Verticillium dahliae*, and a three-year grass ley almost eliminates *Corticium solani* infection of clean potatoes in New Zealand. Unfortunately a good many grasses are susceptible to *Ophiobolus graminis*, but on heavily infected land timothy (*Phleum pratense*) and tall oat grass (*Arrhenatherum avenaceum*) can be used as grass leys with fair safety [5]. Against flax wilt a rotation of eight years is sometimes practised, though persistence up to eleven years has been reported, whereas club root may have largely disappeared after four. Potato wart disease, however, remains for ten to fifteen years in the soil and rotation is not a practicable defence against its recurrence. For rotation to be effective it is not necessary that the pathogen should have completely disappeared. Most crops will stand a reasonable degree of thinning without loss of yield — in several a 10 per cent. loss of early stand is scarcely reflected in final out-turn — and cleansing to below the level that will depress yield is fortunately often a simple matter. That standard systems of rotation may be partly based on this cleansing effect, even when the identity of the pathogens is not certainly known, is evident from

the results of departure from them under pressure of circumstances, as in war-time, when diseases ordinarily of little importance may become prominent. The dangers involved in growing a single crop over large continuous stretches of land, in the use of clonal lines, and in other practices which increase the chances of the spores of a parasite in finding a susceptible host to bombard, have already been mentioned. Windbreaks are sometimes of value, not only because of the protection against leaf injury they confer, as for instance in relation to citrus blast (*Pseudomonas citriputeale*), but also because they intercept or break up spore showers and thus reduce the intensity of bombardment.

In many diseases spores are produced or infection occurs only when free water, such as is provided by dew films, persists for some hours. Severe attacks by the strains resembling *Botrytis cinerea* that cause chocolate spot of broad beans (*Vicia faba*) occur during prolonged rainy spells with little wind or sun, and are promoted by a dense stand and by shelter from wind which might dry the film of moisture. Wide spacing, free ventilation, and good drainage are useful in reducing the risk from this disease. In the banana leaf spot caused by *Cercospora musae*, the damage, as in chocolate spot, depends primarily on the intensity of sporulation, which in turn, in much of the Caribbean area, is largely determined by the incidence of dew. Shade reduces dew formation and consequently spore production. Similarly, shaded plants of chilli pepper often escape damage from *Colletotrichum capsici*, as the die-back caused by this fungus at Pusa was found to be closely dependent on heavy and prolonged dew deposits after the rainy season, which were absent under shade. Standing water has also an influence on certain diseases communicated from wet soil to the lower parts of plants. In irrigated citrus groves in California the fruits are much more likely to become infected with brown rot (*Phytophthora* spp.) by splashing, if the water is held for a prolonged period around the base of the tree. Basin-irrigated oranges had 62 times as many fruit infected as furrow-irrigated trees in one reported experiment, and there were over 13 times as many rotted fruit where the water stood for from 24 to 72 hours as when it percolated through the soil quickly. In another group of diseases, soil moisture, linked perhaps with aeration of the roots, seems to play a part as yet little understood. Instances are the root rot of strawberries in Great Britain and North America, root rot and wilt of cotton in the Sudan, and the ' root disease complex ' of sugar-cane in Hawaii and elsewhere. Diseases of this type are characterised by the development of multiple lesions, often associated with a number of different fungi, which tend to appear where drainage is temporarily or locally impeded or where water-logging reduces aeration. The appearances suggest that the lesions are due to local necrosis, perhaps even to the action of a specific toxin on the root cells, and that the fungi that can be isolated from them are secondary invaders, not of high virulence. The improvement of drainage and root aeration are the only measures that can be taken against these diseases until more is known of their etiology. Drainage is also regarded by some as the first means of control of potato blackleg, due to *Bacterium phytophthorum* (Part II, p. 483), while there are many instances of its importance in tropical countries. Other illustrations of environmental factors favouring disease, some of which may be remediable, have been given in an earlier chapter (Chapter V).

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Chapter VIII

VIRUS DISEASES OF PLANTS

Introduction : Historical

Virus diseases of plants are dealt with here in a general way, and the more important types in Britain are described in the second part of this book.

There is no reason to suppose that virus diseases are of more recent origin than those caused by fungi or bacteria. The increased attention that they have attracted of late is probably rather the result of improved diagnosis and of the ever-increasing traffic in plants which has led to the wide dissemination of so many plant diseases from one country to another, than of the appearance of new disease-producing agents. Many virus diseases cause a progressive deterioration, with chlorosis and other obscure symptoms, and so have been more readily overlooked than the rusts or smuts or blights that injure crops. However, one disease now known to be caused by a virus, the ' breaking ' of tulips (Part II, p. 876) (Fig. 409), produces such beautiful effects in the flowers as to have been promptly noticed when it reached western Europe, possibly from Turkey, in the sixteenth century. It was described by the celebrated botanist de l'Ecluse (Carolus Clusius) in 1576 and can be seen depicted in the paintings of Dutch and Flemish artists in the late sixteenth and seventeenth centuries. Methods of inducing ' breaking ' in tulips were advocated, some of them highly fanciful ; in 1670 there was a suggestion that the variegation might be the result of a disease, and in the eighteenth century Van Osten claimed that a whole-coloured ' breeder ' could be broken by grafting a piece of a broken bulb on to it. This clue was not followed up, and there is little indication for another 250 years that broken tulips were thought to be diseased.

A similar state of mind must have existed among those horticulturists who knew more than a century ago that variegation in ornamental plants could sometimes be communicated by grafting. Even a variegation in the apple was transmitted by Vibert in France by shield-budding in 1863. In the elaborate scientific studies of the variegation which is characteristic of the *Abutilon* variety called *Thompsoni* by horticulturists (first observed in a single plant amongst *A. striatum* imported from the West Indies to England in 1868) transmission by grafting to normal green plants was fully established between 1904 and 1908. This type of ' infectious chlorosis ', as it was termed, was found to affect various other Malvaceae and unrelated plants (mountain ash, chestnut, *Euonymus*, jasmine, etc.), but as it was only transmissible by grafting and seemed to do little harm, it was not generally classed as a disease.

The first destructive outbreak of what are now known to have been virus diseases of a crop, of which records exist, appears to have been the ' leaf roll '



FIG. 164.—Leaf roll of potato, variety British Queen (photo by Foister)

of the potato (Figs. 164, 269), which was extremely prevalent in Great Britain for many years from about 1770 onwards. A similar condition was well known in Germany about 1785. Leaf roll, originally known as 'curl', was doubtless due to several viruses, often more than one in the same plant. The symptoms of generalised deterioration — pallor, deformity of the top, leaf and stem weakness, reduction in cropping vigour, and the like — led to the widespread belief that it was the result of 'running out' of the plant owing to long-continued vegetative propagation; degeneration and senile decay were held to be sufficient to account for the trouble. Even as early as 1802, however, a suggestion is found that greenflies were implicated; now amply confirmed, this suggestion received little support at the time, and the view that degeneration in the potato and other plants could best be prevented by outbreeding and the production of seedlings by fertilisation of the flowers held the field for more than a century. During this period it was noticed that degeneration progressed much more rapidly in some localities than in others, and the practice became established of importing potato seed tubers from Scotland for growing in England, with very beneficial results. It was not easy to account for this with the knowledge then available, but northern hardiness and the benefits of 'change of air' were vaguely thought to play some part.

During the nineteenth century several other important crop diseases now known to be due to virus agencies were recognised. Among these was the 'sereh' disease of sugar-cane seen in Java in 1882; this rapidly became a menace to the important and increasing sugar industry of the island and was the cause of the development of biological, chemical, and cultural research into cane problems which led to effective control of sereh and secured for the Dutch East Indies the leading place in sugar-cane research. In 1890 mosaic of sugar-cane was first mentioned under the name 'yellow stripe disease' in Java; unlike sereh, it subsequently spread to all the cane-growing countries of the world, and the extensive deterioration it caused makes its discovery a landmark in the history of plant virus diseases. Tomato mosaic (streak) was mentioned in England in 1887 and described five years later in the United States.

Meanwhile another disease, this time of tobacco, began to attract attention in northern Europe. It was described by Mayer of Holland in 1886 and termed 'mosaic' disease, a name based on the marbled effect of light and dark green patches on the leaves which found popular favour and was afterwards applied to many diseases causing leaf mottling. Mayer showed that tobacco mosaic could be mechanically transmitted from a diseased to a healthy plant, reproducing

the disease. Further studies of the same disease by Iwanowsky in 1892 demonstrated that infection of a healthy plant by the juice from a diseased one still occurred after the juice had been filtered through a porcelain filter that prevented the passage of bacteria, and also that the infective matter multiplied abundantly in its new host plant, resisted drying and alcohol, and thus differed from bacteria and the like; therefore he thought the infective matter must be something in solution or perhaps in minute bodies in the cell plasma. For the first time the existence of invisible disease-producing agencies smaller than anything yet known was postulated; their study was made possible and a basis for virus research established. A few years later the famous Dutch bacteriologist Beijerinck, knowing nothing apparently of Iwanowsky's work, made a detailed study of Mayer's disease and, reaching the same conclusions as Iwanowsky, propounded in 1898 his theory of the *contagium vivum fluidum*, an infectious living fluid or non-particulate agent of disease. It was in the same year that Loeffler and Frosch, working on foot-and-mouth disease, first demonstrated that animal diseases occurred of the type now known to be due to viruses.

Soon after Mayer's work Erwin F. Smith carried out studies of a destructive disease known as peach yellows in the United States (where it was first recorded in 1791) and in 1888 published the results of experiments proving that the disease could be transmitted by grafting or budding but not by any other of the methods that he tried. Beijerinck thought that peach yellows was another example of the *contagium vivum fluidum*, and it was later recognised as being allied to tobacco mosaic and similar diseases.

For a number of years after Beijerinck's work there was little progress in the study of virus diseases of plants though there were isolated investigations of interest. Thus, in 1901 the Japanese Takami succeeded in transmitting the 'dwarf' or 'stunt' disease of rice by the leaf hopper *Nephotettix apicalis*, the first insect transmission of a disease now known to be due to a virus on record. In 1902 Kamerling claimed to have transmitted sugar-cane mosaic in Java by injecting sap from a diseased into a healthy cane. Between 1906 and 1915 a group of workers in the United States associated beet 'leaf curl' with infestation by leaf hoppers, then showed that these insects caused the disease, and finally that they could only do so after feeding on a diseased plant and that the infectious agent was not merely carried mechanically by the insect. In 1913 Quanjer, who had already started his classical researches on the virus diseases of potatoes in Holland, reported that a necrosis and lignification of the phloem was a constant concomitant of potato leaf curl (roll) and was an infallible symptom of the disease, which he therefore termed 'phloem necrosis' of the potato. In 1914 Orton described and named potato mosaic which he had seen in Germany in 1911, and introduced the name 'leaf roll' instead of 'curl' into American and English usage, and also the term 'streak' for yet another type of these potato diseases. From 1914 to 1918 Allard greatly extended knowledge of tobacco mosaic. In 1916 Quanjer proved that potato leaf roll was an infectious communicable disease.

It was still possible, however, for one of us to write in 1918 in discussing tobacco mosaic: "It is, perhaps, the best known example of a group of diseases of obscure origin, in which not only has no parasite been detected, but there is

strong reason for believing that none of any type known to us can exist. Some hold that it is a physiological disorder, due to an innate alteration in the processes or functions of the plant, not brought about by the presence of an independently living parasitic organism ; others, that it must be parasitic but due to a form smaller than any yet known, or even to a ' living contagious fluid '— a suggestion which goes still further beyond the limit of any life known to us. It seems more reasonable to suppose, as other investigators have, that the virus is one of an enzymic nature, capable of producing extensive effects in minimum doses. . . . Similar diseases are known in tomatoes, chillies, and other plants. . . . They form a group which is perhaps the most obscure in the whole field of vegetable pathology."

Soon afterwards the successful insect transmission of spinach blight (Cucumber mosaic virus) and potato ' mosaic ' in the United States, of potato leaf roll in Holland, and of sugar-cane mosaic in the United States, together with the discovery in the United States that cucumber and bean mosaics were transmissible through the seed, marked the initiation of a period of intense activity in plant virus research. Investigation of potato virus diseases was actively prosecuted in Holland, the United States, Canada, Great Britain, Ireland, France, and many other countries. Records of diseases of the ' leaf curl ' and ' mosaic ' types became frequent, so that in 1921 it was stated that mosaic was known in 30 genera of 10 families of plants and mosiac-like diseases in 8 genera of 5 families. The view that these diseases were caused by ultramicroscopic organisms was gaining in credence. Studies of their behaviour in the insect host, especially in *Eutettix tenellus* (Fig. 172), the vector of curly top of sugar beet in the United States, were throwing light upon the relation between insect and virus. By this time, too, the view that ' degeneration ' was a concomitant of long-continued propagation from the same stock had been rudely shaken. The opinion was definitely expressed in England in 1921 that potato " degeneration is but a symptom of a disease, and this disease is probably mosaic ". In France in the same year it was stated that degeneration is only the consequence of disease or poor adaptation of a plant to its environment and that, in the potato at least, propagation by asexual means cannot be held to lead inevitably to degeneration of a variety ; while in the United States, again in 1921, the running out of a large series of potato varieties observed in Minnesota was believed to be due to the introduction of mosaic (using the term in a broad sense) with some of the tubers brought to the Experiment Station in 1883.

The Nature of the Virus

Throughout this period and for some time afterwards attempts were made to identify as the causal organisms of virus diseases of plants certain elements seen in the diseased tissues or obtained in cultures from them. In some cases bodies reminiscent of the ' elementary corpuscles ' described in certain animal and human virus diseases were thought to be the infective organism, and the name *Strongyloplasma ivanovskii* was given to bodies seen in tobacco mosaic. Amoeboid bodies found in maize and *Hippeastrum* mosaic and, later, in those of sugar-cane,

tobacco, and *Brassica* were thought possibly to be protozoa or other living organisms causative of the diseases, as some held the Negri bodies in animals affected with rabies or the Guarnieri corpuscles associated with smallpox to be. Flagellates and Trypanosome-like organisms were reported in the phloem of various plants suffering from virus diseases, but were later shown to be normal constituents of the cells. The new genus *Phytamoeba* was established for supposed amoeba-like organisms seen in Fiji disease of sugar-cane, while *Plasmodiophora tabaci* was reported in mosaic tobacco plants, and minute motile organisms were stated to occur in the cells and chloroplasts of mosaic tomato and other plants, but were later seen in healthy plants also and were presumably due to contamination of the material.

In 1922 Twort reported his constant failure to establish cultures of ultra-microscopic organisms occuring saprophytically. He supposed that more primitive forms of life than the bacteria or protozoa must exist and that the filter-passing lysins associated with many bacteria which he had found in 1915 (the bacteriophages of d'Herelle, 1917) may be in this class ; he inclined to the view that a precellular form of life, situated in a sense between the simple enzymes and the bacteria, may constitute the ultra-microscopic viruses. Another possibility which he discussed in the following year was that the viruses might be unimolecular stages of the host which for some reason preserved their independence when the fertilised cell first divided. Akin to this view was one expressed in the United States in 1923 that the agent might be a product of the cell such as a particle of chromatin or possibly some structure such as a gene which had escaped from the shackles of co-ordination in the cell. Again it was thought in 1926 to be conceivable that a virus might be an intermediate stage in the evolutionary path from the non-living to the living and might have acquired some of the properties of living matter, e.g. that of reproduction, but not others. Though successful cultivation *in vitro* of the causal agent of tobacco mosaic was reported in 1924, subsequent work failed to substantiate this claim. The inclusion bodies (' X '-bodies) found in the cells of many virus-infected plants were early considered to be nothing more than degeneration products of the cell, and later work indicates that they are the result of reaction of the cell to the presence of the virus and are an effect, not the cause, of the disease.

Up to 1935 most biologists regarded the viruses as living organisms, though few believed that any plant virus had been seen under the microscope or cultivated artificially in cell-free media ; there were still some, however, who maintained that an autocatalytic as opposed to an organic nature best explained their properties. In that year Stanley [14] announced in the United States that he had isolated from tobacco mosaic juice a crystalline protein having all the properties of the tobacco mosaic virus ; he considered the virus to be an autocatalytic protein, presumably requiring the presence of living cells for its multiplication. The following year he reported the preparation of a similar substance from the juice of tomatoes infected by tobacco mosaic, while Bawden and co-workers who had been engaged at Cambridge in the purification of certain viruses, obtained liquid crystalline proteins from three types of tobacco mosaic which were infective at high dilutions — a millionth of a gram per c.c. or even less — and could be transmitted from plant to plant in series, showing rapid multiplication in the

infected plants. These proteins agreed with Stanley's preparations in their high molecular weight, and X-ray studies indicated that they consisted of closely packed elongated rods. Further studies showed them to be nucleoproteins (containing phosphorus), separating as a jelly on high-speed centrifuging, and the view was expressed, in agreement with the American workers, that these heavy proteins, which were not found in healthy plants, might well be the actual viruses; this view was strengthened by studies of potato virus 'X' reported in 1938, which showed that the virus, when purified, appeared to be a solid mass of nucleoprotein showing under X-ray examination a perfect internal regularity in the particles, of the type found in some large protein molecules.

The rods found in the tobacco mosaic and some other viruses are not true crystals, having 2-dimensional but not 3-dimensional regularity, and are better termed paracrystals; purification reduces their filter-passing facility, a result apparently due to the aggregation of smaller particles present in the sap into longer rods; as shown by electron microscope $280 \pm 8{\cdot}6$ mμ is a most common length. Later studies of the virus of the 'bushy stunt' disease of tomatoes yielded nucleoproteins in a form more closely approaching that of true crystals, with particles approximately isodiametric in shape and not tending to aggregate on purification; the diameter of these particles is about 27·4 mμ* and their molecular weight has been variously calculated as from 8,800,000 to 12,800,00. In the United States, Stanley isolated and studied a heavy nucleoprotein from tobacco affected by the 'ring spot' virus and assigned to it a molecular weight of 3,400,000 and a spherical shape, with a particle diameter of 19 mμ; the tobacco necrosis virus has a particle size between 13 and 20 mμ, and the lucerne mosaic virus 16·5 mμ in diameter, these being about the smallest plant viruses so far reported.

This ring-spot virus is thrown down in isotropic pellets without crystalline structure on high-speed centrifuging and can reproduce the disease. Apparent recovery takes place sometimes, the new leaves coming out without visible symptoms; it was found that in these unmarked leaves only about one part of virus in 500,000 of fresh green leaf occurred, as against one part in 80,000 in leaves bearing many ring-spot lesions. 'Recovery' in this case is apparently merely due to a reduction in concentration of the virus in the host. The concentration of the virus, however, naturally varies according to the time allowed for its multiplication in the plant; ordinary tobacco mosaic protein has been estimated to increase over a million times in a four-day period, and reaches its maximum concentration five weeks after inoculation. The concentration reached may be one part of heavy tobacco mosaic protein in 500 parts of plant tissue.

Specific nucleoproteins have now been isolated from a number of plant virus diseases. Some are infective in dilutions of one-thousand-millionth of a gram per c.c.; in some, the molecular weight of the order of 20,000,000, and still heavier animal virus proteins, have been reported. In the other direction a heavy protein, having a molecular weight of 500,000, has been isolated from the bacteriophages, and these are now regarded as causing virus diseases of bacteria; they range in

* μ (micron) $= \frac{1}{1000}$ mm.; mμ (millimicron) $= \frac{1}{1{,}000{,}000}$ mm.

size from 10 to 75 mμ, are specific to particular bacteria, and are capable of multiplication on them. Each virus has its own particular protein irrespective of the host plant from which it is obtained : tobacco mosaic nucleoprotein is the same in any one of several quite unrelated hosts, while potato X virus inoculated into tobacco produces a protein differing from that of ordinary tobacco mosaic and agreeing with the X virus obtained from potatoes. Their properties of stability, resistance to heat and other agents, and so on, agree with those that had already been established for the virus in the plant. Even strain differences in a virus can be recognised in the purified product. In the highly purified products there is very little else than nucleic acid and protein ; no salts or carbohydrates or bound water, such as occur in all organisms hitherto known, have been detected.

Workers with animal viruses have had some difficulty in accepting all the implications drawn from the botanical work. It so happens that animal viruses exist that are large enough to form a descending series in size from some of the smaller bacteria, without the considerable gap that is apparent between the bacteria and the plant viruses ; indeed some animal viruses are larger than some of the organisms, like that causing bovine pleuropneumonia, that have been artificially cultivated in cell-free media. Furthermore, saprophytic viruses have been discovered by animal virus workers ; one from London sewage is cultivable on ordinary media. At the moment it is hard to say more than that some viruses seem to have the characters of minute organisms, others to be devoid of ' living ' properties as the term is ordinarily understood. Much turns on the definition of ' living matter ', but those who are most closely in touch with the recent work on plant viruses seem to be least inclined to regard them as organisms.

Diagnosis : Symptoms

No exact criterion exists for the diagnosis of a plant virus disease. None of the symptoms is exclusive, and there is no specific property by which the existence of a virus disease can be recognised. The negative characters in the pathogen, of invisibility by ordinary methods of microscopic examination using visible light and uncultivability apart from the living tissues, are those most made use of in diagnosis, and if, in addition, the juice is found to be infective after filtration through porcelain or other bacteria-proof filters and the disease can be transmitted by sucking insects or by grafting, it is fairly certain to be due to a virus. Not all virus diseases of plants, however, have been found to be caused by filter passers, and where the only known method of transmission is by grafting, as in ' paracrinkle ' (virus E) of the potato, ' big bud ' of the tomato, and ' spike disease ' of sandalwood, neither juice infection nor insect transmission can be used to confirm the diagnosis, though there is little doubt that most diseases of this class will be found in time, as peach yellows was found after many years, to be insect-borne. Transmission from plant to plant in series implies multiplication of the infective agent in the host and is a valuable help in diagnosis when no visible cultivable pathogen, such as a fungus or bacterium, can be detected.

The commonest symptom of virus diseases is probably the mottling in different shades of green or green and yellow (occasionally whitish) on which the term

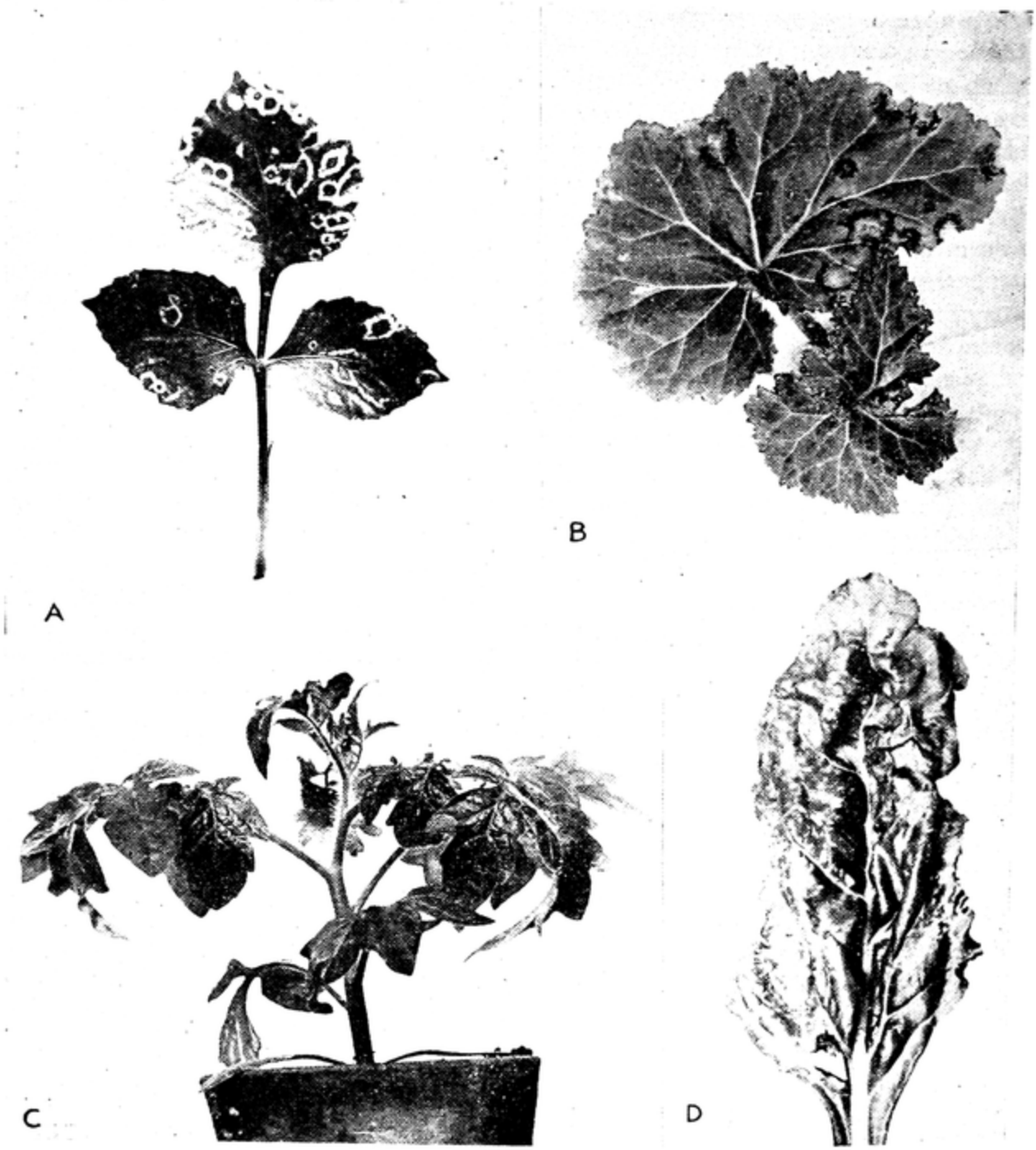


FIG. 165.—Symptoms of virus diseases. *A*, dahlia infected with virus of spotted wilt. *B*, begonia infected with tomato spotted wilt virus. *C*, tomato spotted wilt. *D*, sugar beet mosaic, infection with *Beta virus 2* (*A*, *C*, photo by Bewley; *B*, *D*, photo by Foister)

'mosaic' is based (Figs. 165, 166, 167). This symptom, however, is also found in various deficiency diseases and occasionally a genetical chlorophyll deficiency is expressed in a similar manner. Sometimes, both in virus diseases and these other disorders, the chlorosis or yellowing is generalised over the whole leaf surface. In many mosaic diseases the pallid or chlorotic areas produced on some of their host plants are arranged in concentric circles (the so-called 'ring spots') or in lines. A notable feature of some virus diseases of the potato, tomato, tobacco,

cauliflower, and other plants is the development of dark-green bands bounding the veins; this condition is known as 'vein banding' and may be evanescent. In 'bunchy top' of bananas the most reliable diagnostic symptom is the green streaking found alongside the veins in the leaves and sheaths, due to the phloem being replaced by a morbid tissue surrounded by ground cells rich in chlorophyll. A leaf symptom of value sometimes in diagnosis is a translucent appearance of the veins termed 'vein clearing' (Fig. 168); this may be a very early symptom as in lily mosaic and the tobacco and tomato diseases caused by potato virus 'Y', in which it is often followed a few days later by vein banding. Like the latter it is evanescent.



FIG. 166.—Tomato showing yellow mosaic. Left, normal leaf; right, infected (photo by Bewley)

More characteristic than mottling are various types of curling or crinkling

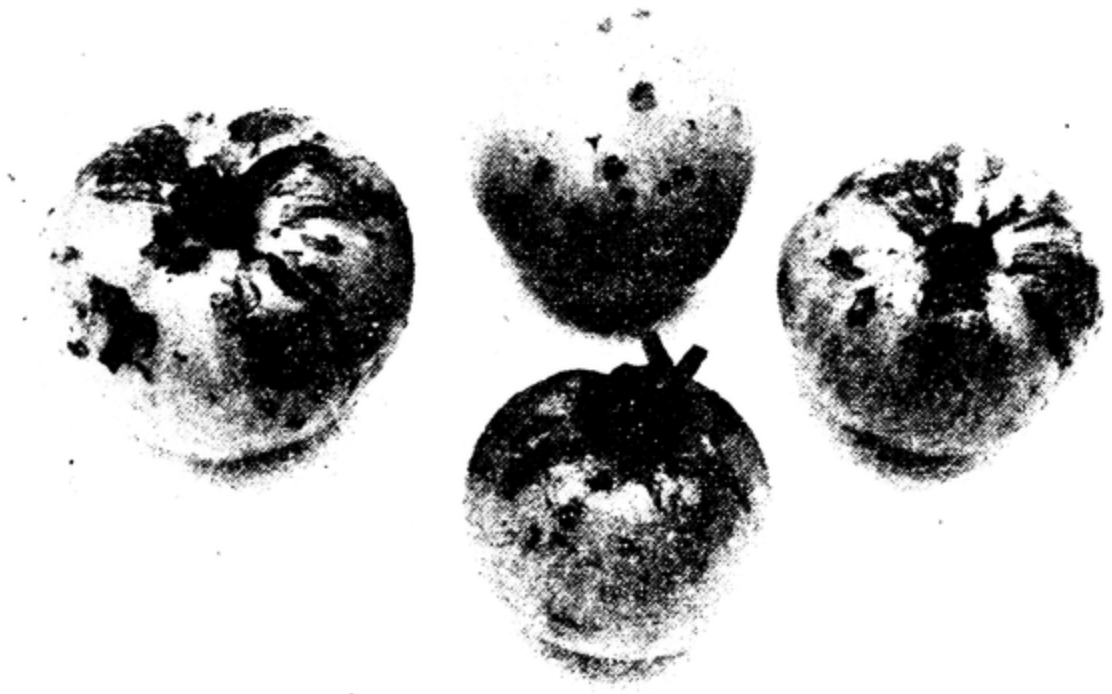


FIG. 167.—Green tomato fruits showing waxy, brown 'scald marks' due to infection with mixed virus streak (photo by Bewley)

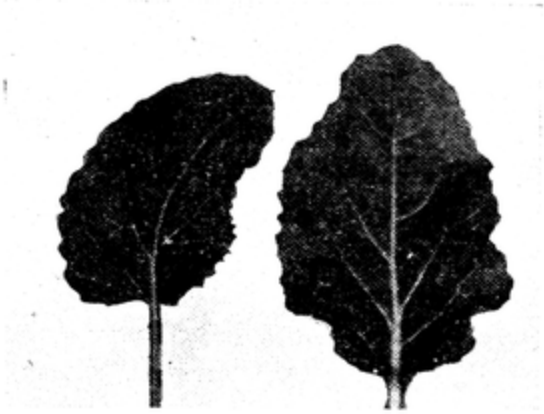


FIG. 168.—Symptoms of vein clearing in virus infected broccoli (photo by Caldwell & Prentice, *Ann. App. Biol.*)

of the leaves, though these again may be simulated by insect attacks or, more rarely, by those of parasitic fungi. The latter, however, are usually more localised than the effects of virus diseases, which tend to be generalised throughout the plant and are sometimes fully systemic.

Necrosis results from the attacks of a good many plant viruses (Figs. 175, 176); it may be restricted to spots on the leaf which may be of the ' ring spot ' type, or arranged in angular lines that have been described as resembling hieroglyphics, or may occupy small or large continuous areas; or the necrosis may occur in streaks on the leaves or stems, the leaf veins sometimes being the first to be affected. In its most severe forms necrosis affects the whole tip of a shoot, or the petioles of the leaves, or an entire branch system, causing the collapse of the parts above the area affected. Necrotic areas may also develop in storage organs, as in the nêt necrosis of potato tubers which results from the destruction of the phloem in plants affected by leaf roll. The tobacco necrosis virus can kill the cells of all organs except the root but never causes mottling or chlorosis; ordinary tobacco mosaic, on the other hand, only rarely, as at high temperatures, kills tobacco cells.

Distortions and malformations of organs and abnormal growths such as galls and leafy enations are evidences of the profound changes in the internal order of the plant brought about by some virus diseases. A remarkable example is the ' fern leaf ' type of foliage (Fig. 170) found sometimes in tomato plants infected by tobacco mosaic or by cucumber virus ' 1 '. In extreme cases the leaf lamina may be so reduced that the organ resembles a tendril. Elongation of the stems is checked in many virus diseases, to several of which the descriptive popular names of ' dwarf ', ' stunt ', and the like have been applied. ' Bunchy top ' and ' rosette ' are other descriptive names of virus diseases characterised by shortening of the internodes and crowding of the leaves at the tip of the shoot. Less often, increase in size results from virus infection, as in ' giant hill ' and witches' broom of potatoes, the overgrown tomato plants produced by tomato enation virus, and the abnormally large leaves found in elm mosaic. A character-



FIG. 169.—Tomato showing symptoms of streak disease (photo by Foister)

istic feature of potato witches' broom is the growth of many (up to 200) thin wiry shoots from the leaf axils and base of the plant; these may sometimes be thread-like and leafless. In 'spike' disease of sandalwood the new leaves formed after infection is established are narrow, pointed, stiff, erect, and crowded together by shortening of the internodes; they become progressively smaller and narrower, so that eventually the new shoots resemble fine spikes bearing four rows of bristles. Wiry, finely branched shoots with very narrow leaves arise from the trunk and branches of peach trees suffering from yellows.

Galls are caused by a few virus diseases, the best known of which are the 'big bud' of tomatoes and Fiji disease of sugar-cane; the latter has as its chief symptom the production of elongated swellings along the veins of the under side of the leaves. In the 'stripe' disease of narcissus and daffodils small galls or corrugations occur on the leaf surface, while small warts are found on the veins of the lower leaf surface in sugar-beet 'curly top'. Thickening and increased prominence of the veins are found in several virus diseases, especially of the 'curl' or 'crinkle' type. In leaf curl of tobacco and cotton [6] the vein thickenings are green and are often associated with the formation of cup-shaped or laminar leafy outgrowths known as 'enations'. Enations also result from infection of peas or broad beans by enation pea mosaic, and of tomato (enation tomato mosaic) and some species of *Nicotiana* by tobacco mosaic in the British Isles, North America, and elsewhere. A character-



FIG. 170.—Tomato plant showing 'fern leaf' symptoms caused by the presence of cucumber virus No. 1 (photo by Bewley)

istic spindle-shaped or cylindrical swelling of the shoots, due mainly to hyperplasy of the xylem, is a late symptom of the swollen shoot disease of cacao, epidemic in a part of the Gold Coast.

The floral abnormalities caused by virus diseases include the variegation of the petals so beautifully shown by many ' broken ' tulips (Fig. 409), and similar striping of the petals in mosaic narcissus, iris, stock, wallflower, sweet pea (Fig. 171), chrysanthemum, peach, and so forth. Phyllody (virescence of the flowers) occurs in aster yellows, spike disease of sandalwood, and big bud of tomatoes and beans. In ' false blossom ' of cranberry all the floral organs are abnormal and the flower may be replaced by whorls of leaves or a small branch. The well-known teratological prolongation of the axis of the affected flowers termed median floral proliferation is sometimes seen in ' aster yellows ' and ' big bud ' of the tomato. Complete sterility sometimes results from failure of the stamens to mature, while up to 50 per cent. of the pollen has been found to be abortive in tobacco mosaic; or it may be due to abnormalities of the gynoecium as in tomato big bud. Plants affected by the sugar-beet curly top virus may have twisted peduncles, dwarfed and desiccated flowers sometimes lacking the corolla, and malformed or aborted fruits and seeds. Flowering is largely suppressed by cucumber mosaic and aster yellows in some hosts; or young flowers and fruit may be lost by abscission or by drying up. In groundnut, ' rosette ' flowering may occur but no nuts develop after infection. Similarly whole fields of susceptible varieties of cotton have been seen in the Sudan where early total infection with leaf curl had not seriously impaired vegetative vigour, but it was difficult to find any mature bolls. Early blossoming is a feature of some potato and tobacco virus diseases and early fruit maturity of peach yellows.

The roots of plants are little affected by the presence of virus diseases. In sugar-beet curly top there is an increase in the number of thread roots (the ' hairy ' root condition), and in some hosts, such as tomato, the rootlets decay backwards from the tip. Several viruses have been successfully cultivated in the excised root tips of tomato for long periods without the slightest external symptom appearing.

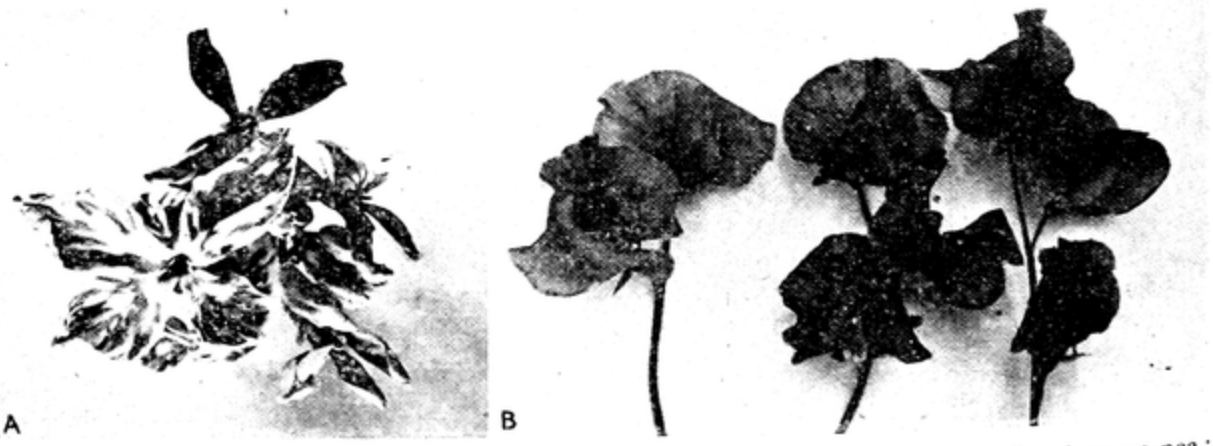


FIG. 171.—' Broken flowers.' *A*, of wallflower (photo by Foister & Noble). *B*, of sweet-pea; on left, not infected; on right, diseased; the flowers are a pale lavender colour, the ' break ' being of a reddish-purple colour (photo by Bewley)

The susceptibility of some crops, such as potato and tobacco, to infection by several different viruses was early recognised. The discovery in 1931 that simultaneous infection with more than one virus caused some of the well-known potato virus diseases, and the analysis and synthesis of these diseases, marked a notable advance in virus pathological research. At an earlier date it had been shown experimentally that inoculation of tomatoes with a combination of potato and tobacco mosaics caused a very severe ' streak disease ', while evidence was obtained that two tobacco diseases in Kentucky were each caused by a mixture of two viruses. It is now fully established that some of the most severe diseases of the potato and other plants are composite mosaic diseases ; potato crinkle (the ' mild mosaic ' of the United States) is due to a combination of potato virus A with X, potato net necrosis to A plus the virus of potato leaf roll, while tomato mixed streak is caused by tobacco mosaic virus plus potato virus X.

Another serious complication in the diagnosis of these diseases is the great differences in the symptoms they induce in different varieties of the host plant. This has nullified attempts to base a classification on symptoms. The most complete studies of varietal reaction to infection by particular viruses have been made on potatoes, and the literature on these diseases is loaded with detailed descriptions of their effects on the numerous varieties commonly cultivated. Apart altogether from the symptomless ' carriers ' of a virus and the phenomena of resistance to, and tolerance of, virus infection, varieties that are apparently fully susceptible may react in different ways to an individual virus. Tests in Holland of the reaction of a large number of varieties to infection by the ' Y ' virus served to distinguish five types of reaction : the potato varieties in group ' A ' suffered from mosaic with drooping of the leaf tips and margins ; in group ' B ' there was, in addition, a certain amount of rugosity with a few fine veinal necroses and yellowing or bronzing of the lower leaves ; group ' C ' added to these symptoms a streak disease causing leaf drooping and desiccation owing to necrosis extending back from the leaf veins to the petiole ; in group ' D ' the necrosis extended to the surface of the stem, while varieties in group ' E ' showed little or no mosaic but there was severe dark necrosis of the veins with some necrotic spotting of the leaf, the necrosis spreading extensively in the stem and being accompanied by leaf drop. A somewhat similar grouping has been made of the reaction of several varieties in the British Isles to the same virus. Tuber necrosis is a prominent symptom in some potatoes affected by ' aucuba mosaic ', while in other varieties there is none, though the leaf symptom of brilliant yellow spotting may be equally apparent in both groups.

As might be expected, still greater differences than those found within the varieties of a single species of host plant occur when the virus is tested on susceptible plants belonging to different species, genera, or families. These differences are greater than those observed when pathogenic fungi or bacteria are similarly tested, so much so as to raise the question whether they are not due to an entirely different order of phenomena. The common ' tobacco mosaic ', for instance, which is fully systemic in the cultivated *Nicotiana tabacum*, produces only local circumscribed lesions on inoculated leaves of *N. glutinosa* and does not spread to the rest of the plant ; similarly circumscribed lesions are produced by it in French beans, but

it is fully systemic in spinach, tomato, and several other hosts. 'Tobacco necrosis virus' occurs naturally in the roots of a wide range of hosts in glass-houses at Cambridge without causing any visible symptoms; natural infection of the leaves is unusual but has been seen in tobacco, while artificial inoculation causes only local infection, except in French bean, in which it may eventually become systemic. 'Tomato spotted wilt', which has probably the widest host range (Fig. 165 c) of any virus disease in England and affects monocotyledons as well as dicotyledons, produces every kind of symptom from local lesions without systemic infection to complete systemic necrosis which kills the plant: vein clearing, mosaic mottling, bronzing, ring spots, leaf curl, stunting, rosetting, necrotic spotting, vein and petiole necrosis, necrotic striping of the stem, distortion of leaves, flowers, and fruit, suppression of flowering, are all amongst the symptoms found in different hosts, though only a selection of them is usually found on any one host. The potato viruses seem seldom to pass outside the *Solanaceae*, but some of them show widely different symptoms on particular genera. 'Aster yellows' in the United States has a host range of over 160 species in many families, amongst which many distinctive changes in habit, overgrowth (excessive production of axillary shoots, aerial roots, elongation of the internodes, and so forth), dwarfing, deformation and pigmentation of leaves, floral abnormalities, necrosis, and decay have been observed; chlorosis or yellowing without mottling is, however, a general symptom. Artificial infection tests in the United States, reported in 1940, showed that cucumber mosaic virus could infect no less than 191 species in 40 families. The diseases caused by this virus or by distinct strains of it are so diverse in their symptoms as often to defy diagnosis without extensive testing or serological study; almost the whole range of symptom expression of virus infection may be found in some plant or other infected by it.

Carriers

The existence of 'carriers' of virus diseases, that is, of plants which contain a virus without any visible signs of infection, was early established. Thus, it was stated in 1923 that the juice from certain tomato plants that showed no symptoms two months after they had been inoculated with mosaic was capable in every case of causing visible infection of healthy susceptible plants. In the same year the existence of a carrier of hop mosaic was demonstrated. Similar results with potato roll and mosaic were reported in the following year. Later it was found that all the main cultivated varieties of potatoes in the United States were so universally infected with certain viruses that it was impossible without special precautions to obtain a virus-free tuber; for a time the name 'healthy potato virus' was applied to the infective agent in these cases, but eventually it was recognised that American potato varieties were all carriers of one or more latent viruses, the commonest of which were strains of the 'X' virus [2]; the vein banding viruses 'A' and 'Y' and a top necrosis virus 'B' are also sometimes concerned. In the British Isles, Up-to-Date potatoes always carry 'X', usually accompanied by 'B'; Golden Wonder and Irish Chieftain always carry 'A', and King Edward, 'E'; since 'E' is only transmitted by grafting, cannot spread naturally, and has been found in King Edward in all parts of the world, the stock must

have been infected at its very origin. The ' Y ' potato virus is reported to be carried masked by turnips and other brassicas as well as by red clover, garden peas, and bindweed; tomatoes show the early symptoms of infection (vein clearing, vein banding, and faint mottling) but become symptomless carriers as growth proceeds. In experiments at Cambridge leaf roll was carried by turnips, Brussels sprouts, stocks, and campanula. Carriers of the ' leaf roll ' and ' Y ' viruses, usually the two most destructive in the south of England, are rare in the potato varieties grown in Great Britain except in old-standing ' chronic ' infections, but tolerant varieties are known in Germany, and the Swedish variety Imperial is a perfect carrier of leaf roll. Carriers of yellow edge and crinkle of strawberries are fairly common, especially in varieties in which the parent species *Fragaria chiloensis* is predominant, while in raspberries the well-known Lloyd George variety is sometimes a symptomless carrier of mosaic.

In all these cases the presence of the virus in the carrier is revealed by infecting by grafting or otherwise a susceptible or ' indicator ' plant in which visible symptoms are produced. If potatoes carrying ' A ' or ' X ' virus are grafted with one or other of several potato varieties, e.g. Up-to-Date, a lethal top necrosis results and the plant is killed. The wild strawberry *Fragaria vesca* similiarly serves as an indicator plant for yellow edge and crinkle of strawberries.

The economic significance of carriers is twofold. In the first place, a carrier variety may suffer little from the presence of the disease, and under certain conditions the use of carrier varieties may minimise the losses in a crop. This is not of universal application, however, for cases have been reported in which yields have been substantially depressed by the unsuspected presence of a symptomless virus. Secondly, the carrier, while giving complete satisfaction in certain districts, may on transfer to a new locality become exposed to infection by another virus and the combination may be far more destructive than either alone; thus, when a strain of X producing no symptoms on the potato variety President becomes combined with ' A ', which alone causes only a slight mottle on this variety, the result is a severe disease of the crinkle type. The rapid degeneration from crinkle of Up-to-Date potatoes which are extensively exported from the British Isles into Southern Rhodesia is probably to be traced to the carried virus which is present in all the imported stocks meeting with another crinkle constituent found locally.

Transmission of Virus Diseases

In the transmission of these diseases it appears to be necessary for the virus to come into contact with living cell plasma. Unbroken cell membranes ordinarily oppose its passage, though there have been successful infections when a virus suspension of tobacco mosaic or of potato virus ' X ' is sprayed on the surface of the leaf; these two viruses are readily transmitted by contact, as for instance when leaves of healthy and diseased plants are blown together by the wind, and it is difficult to exclude the possibility of infection through broken hairs or other surface micro-lesions, though the experiments indicate that stomatal infection is involved.

Natural infection by direct transmission of the infective agent through the air,

as in most fungal and some bacterial diseases, has not been found in the viruses except in the very aberrant tobacco necrosis virus which has been recovered from the air and water in infected glass-houses and reaches the roots of the host plants through the soil when every other source of contamination but air or water has been excluded. This virus enters the roots through broken root hairs and other natural injuries and will not penetrate roots growing in contaminated water cultures unless these are wounded. Transmission through the soil also occurs in the wheat rosette virus in the United States, while the virus of ordinary tobacco mosaic survives in the debris in or on the soil from a diseased tobacco crop, and infection from this source is the most important cause of the disease in certain areas on land on which an infected crop was grown the previous year; it has even been stated that this virus can persist in soil from one season to the next after being leached out of the infected tissues [4].

In tobacco and tomato mosaic, natural infection frequently results from the handling of healthy after diseased plants, as in the operations of topping and priming of the crop, the hands of the workers becoming contaminated by the infective juice from the already diseased plants. Tobacco mosaic virus also survives for years in manufactured tobacco, and it has been proved that not only the fingers of smokers of infective tobacco but also the sputum from those who chew the tobacco, can, and often do, infect the crop. This virus is intensely infective, successful inoculation having been accomplished by cutting with sterile scissors the hairs on infected leaves without injury to the cells below and using the scissors thus contaminated to cut hairs on a healthy leaf; even at a dilution of one part in a million it is still infective. It is also extraordinarily resistant to drying and putrefaction, some virulence having been found still persisting in 1937 in tobacco cured in 1882, while bottled juice that had undergone putrefaction remained highly infective after fifteen months. Tests in 1940 of crushed mosaic tobacco leaves kept since 1925 in water in flasks gave only a very small number of successful infections, but similar material to which the preservatives benzene or xylene had been added caused 30 to 40 per cent. of the number of infections caused by a fresh extract. The inactivation of tobacco mosaic virus caused by the growth of aerobic micro-organsims is much greater than that caused by anaerobes.

Evidently it is difficult to avoid contamination of the crop with a virus such as tobacco mosaic, and both it and tomato streak have been found to be transmitted to healthy tomato plants on pruning knives, as has also sugar-cane mosaic on the knives used for cutting the setts. Fortunately there are not many viruses with such properties, and many lose their power to infect quite soon after removal from the living host; tomato spotted wilt survives in expressed sap only for about four hours and cannot stand drying. Natural spread by mechanical means can be of little practical importance in most virus diseases, for it is usually necessary to inoculate by needle-pricks or through surfaces abraded by carborundum and the like to ensure successful infection.

The transmission of plant viruses by insects is of far greater importance than the methods already mentioned and accounts for the natural spread of the great majority of these diseases; in a recent list 70 plant viruses are stated to be so

transmitted. Most of the insects concerned are leaf suckers of the order *Hemiptera*, a few are leaf abraders of the order *Thysanoptera*. There is a small number of cases of transmission by leaf-eating insects such as grasshoppers and *Diabrotica* beetles and one or two by mites (*Arachnida*). Amongst the *Hemiptera*, aphides, leaf hoppers, and white-flies are the chief vectors, but there are a few plant bugs (*Heteroptera*). In closely allied species, one may be a successful vector while another fails even though it may be able to pick up the virus. It is of interest to note that some viruses are rendered inactive if ingested or passed into the blood of non-vector insects and that even in the vector itself there is evidence that the virus may come into contact during feeding with some inactivating substance (e.g. a digestive enzyme), for it has been found that the insect can retain the virus longer when fasting than when allowed to feed.

Aphides are the most important vectors, both from the number of viruses they transmit and their prevalence. Some aphides can transmit several viruses, the peach aphis *Myzus persicae* more than twenty; conversely, some virus diseases can be transmitted by more than one species of aphis, yellow dwarf of onions by more than fifty. Leaf hoppers are responsible for the spread of several major diseases in overseas countries — curly top of sugar-beet, celery, aster, and peach yellows, rice dwarf, maize streak, Fiji disease of sugar-cane. White-flies transmit cotton and tobacco leaf curl and cassava mosaic. The chief disease in the British Isles transmitted by insects other than greenflies is the spotted wilt of tomatoes and many other plants, of which the local vector is the Thysanopterous *Thrips tabaci*. The plant bug *Piesma quadrata* is the vector of sugar-beet crinkle in Europe.

The leaf-sucking insects feed by a long and delicate stylet apparatus or beak which not only serves to suck up the leaf juices but also to inject saliva through a second channel along the passage forced through the tissues; this often reaches the phloem (Fig. 172). It is generally assumed that the infective agent sucked up with the juice into the insect is eventually passed into the new host with the saliva, though difficulties in accepting this view have been pointed out. A so-called 'incubation' or latent period in the insect between the time of ingestion of the virus and its suc-

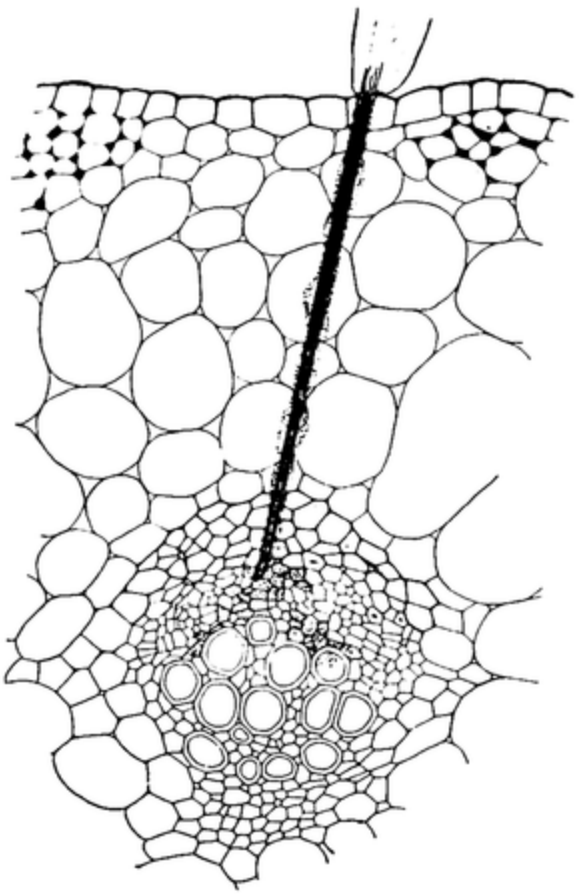


FIG. 172.—Stylet of *Eutettix tenellus* showing the relation to the tissues of the beet petiole, during feeding, in sugar-beet infection with curly top virus (× 180) (after Bennett, *J. Agric. Res.*)

cessful transmission to a new host is found in a group of virus diseases, especially those transmitted by leaf hoppers and thrips, but including a few that are aphis-borne. The latent period may be nine days or more in *Eutettix tenellus*, *Macrosteles divisus* (*Cicadula sexnotata*), and *Macropsis trimaculata*, the vectors, respectively, of sugar-beet curly top, aster yellows, and peach yellows. All the evidence available indicates that, at least in the group showing this latent period, the virus must pass from the intestine into the blood. Whether it multiplies in the insect is still in doubt. On the one hand there are experiments indicating that the aster yellows virus can multiply at least a hundredfold in the body of its vector, and it has also been thought that sugar-beet curly top virus increases in *Eutettix*, though, if there is an increase, it is not sufficient to maintain for long the amount originally picked up from the plant. On the other hand, the delayed ability to transmit these diseases may be explicable on a quantitative basis, the amount of virus that the insect can pass out into the plant increasing with time until a quantity sufficient to cause infection is reached; the length of time the vectors remain infective has been shown to depend directly on the period during which they had previously fed on the diseased plant.

In a second and much larger group of virus diseases, including most of those transmitted by aphides, the insect becomes infective immediately after feeding, and there is no evidence of multiplication of the virus in the vector. In such cases there is obviously a possibility that the insect serves as a merely mechanical instrument of transmission, similar to a needle. It has been found, however, that aphides that have had a preliminary period of fasting are more highly infective than those that are allowed to feed, and can transmit infection to a series of plants, while those that have fed freely usually fail to infect more than a single plant. It seems evident that the virus has to be taken into the insect even in this group. No evidence has been obtained so far that any plant virus undergoes a necessary part of its development in the insect, as the malarial parasite does in the mosquito.

The power to transmit a virus is not equally possessed by all individuals of the vector species. The most interesting cases are those in which ability and inability to transmit are inherited characters following a simple Mendelian rule, as in *Cicadulina mbila*, the vector of maize streak. Uninfective *C. mbila* may be made infective by releasing the gut contents into the blood by puncturing the intestinal wall after feeding on a diseased plant. A similar case is known in the mosquito vector, *Aëdes aegypti*, of the animal virus disease, equine encephalomyelitis. In a few cases the virus cannot be taken up by adult insects but only while they are still nymphs (the thrips vectors of spotted wilt), while the exact opposite has been reported in the transmission of beet crinkle by *Piesma quadrata*. The virus of rice dwarf passes from the mother to her offspring in *Nephotettix apicalis*, so that this leaf hopper can be a vector even when it has never fed on a diseased plant.

Ability to act as a vector is clearly not a simple matter. So also the relations between the virus and its host plant are complex. The production of local necrotic lesions instead of systemic invasion is well known amongst the hosts of many viruses; both types may sometimes be found in different varieties of a single host, such as the potato. These local necroses preventing systemic spread seem

to be examples of hypersensitivity similar to that to which many plants owe immunity from rusts. Sometimes all attempts to inoculate by needle pricks fail, whereas the stylet of the vector succeeds; this may be due to the slight injury caused to the cells by the latter as compared with a needle, for the needle has been successfully used to inoculate the vector of maize streak but not the plant. It is possible that the enzymes present in the vector's saliva or in the plant cells of the host may play a significant part in the process of infection as they do in infection by parasitic fungi, but there is as yet little evidence of this. With some viruses only young leaves can be successfully infected by the insect (e.g. cassava mosaic), in others there is a gradient of susceptibility between leaves at different heights on the plant, as in tobacco mosaic. It seems evident that the behaviour of viruses as infective agents is governed largely by physiological factors, and that, as with fungi, successful infection is a delicate process dependent on conditions affecting host, vector, and parasite.

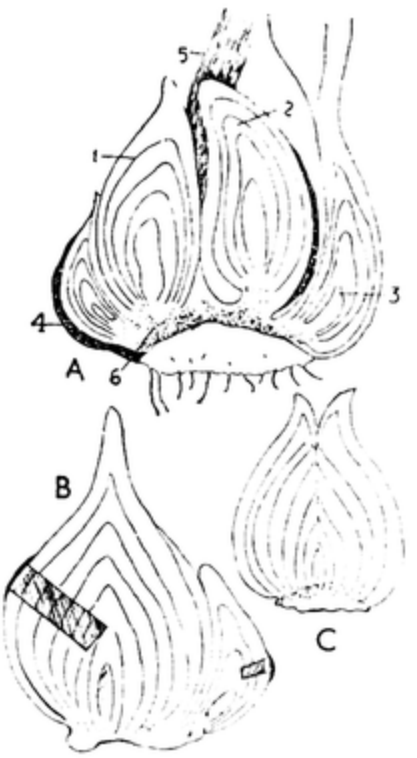


FIG. 173.—Transmission by grafting. 'Breaking' in tulips (see also Fig. 409). *A*, section through tulip plant, immediately after flowering: 1, 2, flowering bulbs for next year; 3, offset, which has produced a leaf; 4, dormant offset; 5, base of flowering shoot; 6, bulb base which will disintegrate when the bulbs ripen. *B*, a plugged bulb and lateral showing a plug of tissue from an infected bulb; the plugs are covered with paraffin wax and finally with canada balsam. *C*, two bulbs grafted together, kept together by tying with raffia (after Cayley, *Ann. App. Biol.*)

Transmission by grafting (Fig. 173) succeeds in all cases in which full organic union can be secured between a diseased and healthy susceptible host. It is the only method known in a few cases, the number of which is decreasing as their insect vectors are discovered.

Transmission through seed is known in bean, clover, cucumber, lettuce, and other mosaic diseases. It cannot occur where the virus is restricted to the phloem, as there is no vascular connection from mother plant to embryo. In all cases of seed transmission that have been adequately investigated, the virus has also been found in the pollen. External contamination of the seed may be responsible for infection of the cotyledons and other parts of the germinating seedling, and from the practical point of view may be as important as true seed transmission. Of much greater practical importance, however, is the spread of infection by the use of diseased tubers, bulbs, roots, suckers, grafts, and the like in plants that are propagated vegetatively.

The movement of the virus within the plant may be restricted to the phloem as in sugar-beet curly top, or may be independent of the vascular tissues. Movement in the phloem may be very fast, the curly top virus having been found 15 cm. down a sugar-beet seedling leaf 6 minutes after the vector was placed on the leaf, and maize streak virus 40 cm. down, 2 hours after feeding commenced. Tobacco mosaic virus, on the other hand, shows little movement for 2 to 4 days in super-

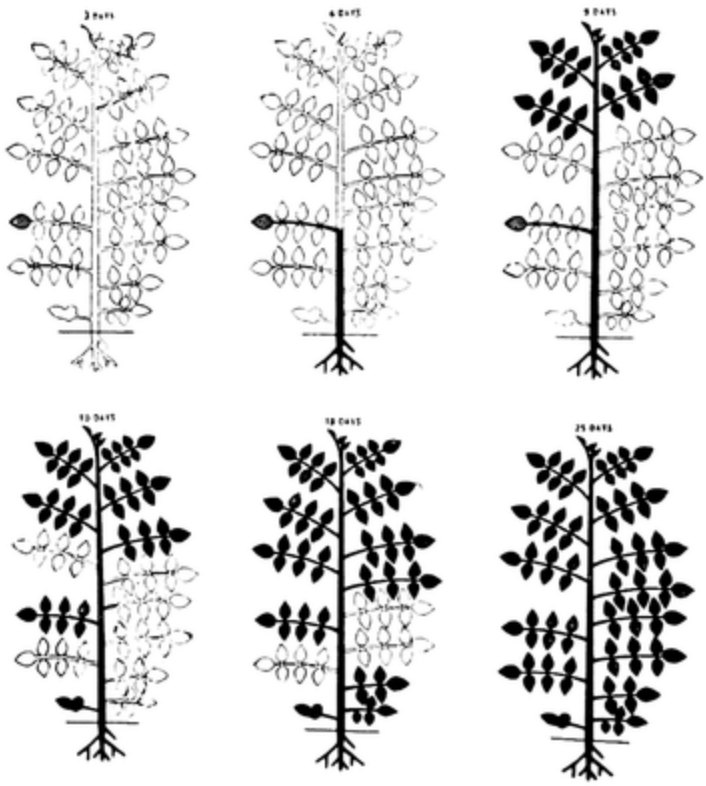


FIG. 174.—Movement of tobacco mosaic virus within the plant. Diagram to show the progress of the spread of mosaic (in black) through a medium young tomato plant; based on tests of Dwarf Champion tomato plants about 15 in. high, growing in 6-in. pots, in an unheated green-house; inoculated leaflet shaded (after Samuel, *Ann. App. Biol.*)

ficial inoculations but then passes rapidly from the inoculated leaf towards the root and top of the plant; a distance of 7 inches was traversed in 1 hour in some tests on tomatoes (Fig. 174). In the rapid movement, lateral organs such as leaves and fruit trusses may be missed for a time. It is believed that the virus on inoculation moves slowly from cell to cell through the plasmodesms until it reaches the phloem, in which it travels fast. In cowpeas inoculated with tobacco necrosis virus it took 12 days for movement to become rapid and 48 hours later the distance traversed was doubled. In passing across a leaf from one epidermis to the other, tobacco mosaic virus travelled only 7 or 8 μ an hour. The rapid rate of virus movement through the phloem is in harmony with what is known regarding the movement of solutes through this tissue.

The incubation period of plant virus diseases, that is, the time elapsing between inoculaton and the first appearance of symptoms, is very variable but is sometimes longer than that known in any fungal or bacterial disease. The shortest period that has been recorded appears to be 18 hours for the tobacco necrosis virus. At the other extreme is apple mosaic on certain hosts, since 20 to 27 months elapsed before symptoms appeared in inoculated roses 6 inches below the point of inoculation. The earliest symptoms of 'yellows' in peaches infected by means of *Macropsis trimaculata* appeared in one set of recorded experiments in from 14 to 268 (average 147) days, but when transmitted by budding the first symptoms may take 2 or 3 years to appear. In the 'phony peach' disease the incubation period is about 18 months, and in 'sandalwood spike' 7 months under natural conditions and $4\frac{1}{2}$ months in artificial inoculations.

Some viruses are localised in particular parts of the plant. The most striking example of this is the virus causing 'phony' peach disease in the United States which is found only in the roots and is not transmissible by sap or any known insect vector. It has been successfully transmitted only by root-grafting; although a healthy scion grafted on a diseased stock will show symptoms, it will not infect another tree if grafted or budded into it. Though some toxic principle is carried up from the roots, causing serious dwarfing and crop reduction, this cannot be the virus itself or it would be transmissible by above-ground grafts.

The tobacco necrosis virus is also present ordinarily only in the roots, though it sometimes becomes systemic and can be naturally or artificially inoculated into the leaves to produce local lesions. Several viruses occur mainly in the phloem, the sugar-beet curly top virus so much so that the other tissues seem to be actually toxic to it. A very few, of which the phony peach virus is the best example, are apparently restricted to the parenchyma, but the majority, including all the simple mosaics, are found both in phloem and parenchyma. The chief vector of maize streak, *Cidcaulina mbila*, can pick up the virus and remain infective for 9 weeks after feeding for only 15 seconds, much too short a time for the stylets to have reached the phloem where the insect normally feeds. Several viruses travel so slowly to distant parts of the plant that it may be a considerable time before all parts are reached. In sugar-cane mosaic healthy tillers can be found at the base of diseased plants, and in potato leaf roll the harvesting of the tubers intended for seed while still immature has been advocated as a means of reducing loss from this disease.

Influence of the Environment on Viruses and Virus Diseases

As in diseases caused by fungi or bacteria, temperature and other weather conditions may influence the incubation period, course, and effect of virus diseases. The viruses themselves are often more resistant to heat than are fungi, an extreme case being that of tobacco necrosis which has a thermal death point (10 minutes) at 90° to 92° C. but can withstand dry heat at 100° C. for 15 minutes. This virus is also very resistant to chemicals and retains virulence after 6 months in absolute alcohol, while the beet curly top virus resists 1 in 50 mercuric chloride, 1 in 25 carbolic acid, and 1 in 200 copper sulphate. Another resistant virus is that of ordinary tobacco mosaic which will stand 10 minutes at 93° C. though only 1 minute at 96°; it can also endure long exposure to mercuric chloride at germicidal concentrations. In the cured leaf this virus required 10 hours at 100° C. dry heat for complete inactivation. Potato virus 'Y' is inactivated by 10 minutes at 52° C. and by 75 per cent. alcohol, while tomato spotted wilt virus is even less resistant to heat. Ultra-violet light destroys viruses more slowly than bacteria or even bacterial spores.

In early experiments the incubation period of tobacco mosaic was shortest at 28° to 30° C., the development of symptoms being slowed down by lower temperatures proportionately to the growth of the plant; little or no symptoms appear at temperatures that check growth or if the plants are kept at high temperatures such as 36° or 37° for a fortnight. The incubation period for this disease was found in New Jersey to be 4 or 5 days in June and July as compared with 6 or 7 in December and January. Even severely infected mosaic tomato plants were noted as early as 1917 to have the symptoms completely masked at an average air temperature of 85° F. Similarly it was reported from Canada in 1921 that the symptoms and loss of potatoes affected by mosaic or leaf roll might be suppressed in the dry warm summer climate of the western areas, and this climatic effect has since been fully confirmed, e.g. in Kansas where mosaic, though completely masked, may seriously reduce the yield [1]. Light, soil temperature,

and soil moisture have little effect on the foliage symptoms of potato virus diseases, but an air temperature of 25° C. is sufficient to mask most of them ; yellow dwarf differs from the others tested in the United States in being very severe at 25° but masked at 15°. The yellow edge disease of strawberries (p. 786) is difficult to diagnose at certain seasons, the symptoms being most evident in the south of England in late autumn, though severe cases can be recognised from June onwards ; the temperature does not usually rise sufficiently for clear symptoms to appear before June, but even then the soil may be too dry to allow them to develop, since there seems to be a close correlation between symptom expression and soil moisture. In the control of this disease by roguing it is necessary to take into account the weather during the previous week or two and select periods likely to allow symptoms to be clearly seen.

The effect on virus diseases of the nutrition of the host has not been as extensively investigated as in other types of disease. The information available, however, suggests that a balanced nutrition is as important in reducing the losses they cause as in those due to fungi or bacteria. Susceptibility to yellow tobacco mosaic can be influenced by the level of supply of nitrogen, phosphorus, or potash, the phosphorus effect being related to the rate of growth of the host. With nitrogen or potash, however, some other factor seems to be concerned [12, 13]. The influence of nutrition on the concentration of the virus in the tissues is shown by the fact that high nitrogen feeding can increase the concentration of tobacco mosaic in the cells to about five times that in normally fed plants, while 8 days after inoculation the juice from plants receiving high doses of nitrogen may contain twelve times more virus than that from low nitrogen cultures.

The ecological relationships of the insect vectors are even more important in the incidence of virus diseases than the effects of climate on the diseases themselves. They have been most fully studied in the aphis vectors of potato virus diseases and the leaf hopper that transmits sugar-beet curly top. In North Wales cool, damp windy areas occur near the coast in which potatoes have been grown for long periods without degeneration. The initial infection of the crop each year has been traced to the migration to it in early summer of winged *Myzus persicae*, the principal vector of the diseases causing degeneration, chiefly leaf roll in the area in question. These aphides over-winter in the wingless form on winter crops of savoy cabbage, kale, and other brassicas which, as already mentioned, may be symptomless carriers of leaf roll. A certain number of the winged aphides migrating from brassicas to potato fields may contain the virus, and each serves as a focus of infection, while others settle on potatoes infected by way of the tubers from the previous crop and so pick up the virus. Migration of the winged form occurs almost exclusively when the temperature reaches 65° to 70° F., when the relative humidity is below 70 to 80 per cent., and the wind velocity is less than 3 or 4 miles an hour. Under these conditions the insects will readily find their way to potatoes from brassicas at least a quarter of a mile away and then move actively in the potato crop from plant to plant and field to field ; the plant-to-plant spread within a field, however, is mainly effected later by the crawling wingless form. In North Wales this period of active spread of leaf roll is usually between late June and the end of August. Where degeneration is rapid, the

infestation may reach 500 to 1,000 aphides per 100 potato leaves, whereas in the good seed areas not more than 20 may be found. Counts over a number of years have shown that infestation varies greatly from year to year, and there is evidence that the percentage of virus infection in the potatoes may be correlated with the intensity of infestation by *Myzus persicae* in preceding years. The time of the flight of the aphides is also important, for a small amount of infestation brought in early in the season might be spread considerably by wingless forms, whereas later there is less time for this to happen before harvest. Almost the same degrees of temperature, humidity, and wind govern the migration of winged forms of the strawberry aphis, *Capitophorus fragariae*, that transmits yellow edge and crinkle of strawberries in south-eastern England. This aphis is able to find and colonise even isolated strawberry plants at least 400 yards away. Its winged generation is active for short periods in May and June, when extensive spread of the viruses takes place.

Similarly with sugar-beet curly top in the western United States the virus, which has a host range (up to 1939) of 75 species of plants in 48 genera of 18 families, is brought into the crop by the spring generation of the leaf hopper vector which migrates from the weeds and bushes of the arid uncultivated plains and foothills as they dry up. Both the magnitude of the migration and the percentage of individuals that harbour the virus vary greatly from year to year. In one area it has been found that when November rain allowed early vegetation in the dry zone, the spring migrants had from 16 to 42 per cent. of infection, whereas in seasons of late rains (December or January) only 2 to 6 per cent. carried the virus. In southern Idaho the percentage of infected spring-brood *Eutettix* was found to vary from 4 to 67 in different years; sometimes the virus overwinters in living leaf hoppers, sometimes in susceptible desert vegetation (*Sophia*, *Salsola*, and the like), sometimes in the cultivated districts. In eleven years there were six of relatively late leaf-hopper movement (from the 4th June) and high yields, whereas in the five years when migration was on or before the 24th May the yields were low because of early infection of the crop; by harvest time practically all the leaf hoppers on beet contain the virus.

It is scarcely necessary to mention that the area of distribution of the vector may determine the distribution of a virus disease. This is well illustrated by peach yellows which coincides roughly in the United States with its vector, the plum leaf hopper *Macropsis trimaculata*.

Virus Strains: Variants

A character possessed by viruses which brings them closer to the organic than to the inorganic world is the occurrence within a single virus of strains differing from one another in physical characters, the symptoms they cause, and virulence. Strain differences have been most fully studied in the potato mosaic virus 'X' and that of ordinary tobacco mosaic but they are marked in cucumber, sugar-cane, and many other viruses.

Experiments at Cambridge reported in 1933 showed the inoculation into tobacco of potato virus 'X' did not always result in the same symptoms. When

the virus was taken from the green and the yellow areas and from necrotic rings respectively, on the leaf, three strains could be differentiated. About the same time the discovery was made in the United States that the yellow form of tobacco mosaic, previously thought to be due to a distinct virus (tobacco virus '6' of J. Johnson), sometimes arises spontaneously in plants inoculated with the ordinary form and that ordinary tobacco mosaic contains many strains. Three of these strains cause the well-known mild, aucuba, and distorting mosaics of the tomato in England.

The term ' mutant ' has been applied to strains appearing spontaneously or produced by heat, irradiation, and the like, but since there is doubt whether the viruses are organisms it is better to adopt the term ' variant ' used by some authors. The variants recognised by virus workers agree with mutants in being permanent departures from the parent type, though, like mutants, they can throw further variants or possibly revert to the parent type. Some of the variants of tobacco mosaic differ remarkably in the symptoms they cause (ring and necrotic spots, filiform leaves, and in their virulence, rate of movement in the plant, and other properties. The same is true for potato virus ' X '; its variants may appear spontaneously or be induced by passage through other hosts, sometimes being so weak as almost to become latent and without symptoms. The ' X ' variants studied in England tended towards reduced virulence, but some of those found in Germany caused much more severe symptoms when inoculated into tobacco than the parent form.

By exposure to X- and gamma-rays, variants of the purified tobacco mosaic virus have been produced with distinct chemical and biological properties. The former seem to be due to changes in the nucleic acid part of the virus molecule, not greatly affecting the molecular weight but causing different solubility and hydration relationships, and in some cases an increased tendency to polymerisation. The biological changes are marked not only by differences in symptoms in tobacco but by a delayed incubation period and sometimes an increase in the number of local lesions produced by a given quantity on leaves of *Datura stramonium* or *Nicotiana glutinosa*; this last may be due to an accretion of virulence (fewer molecules being required to produce a lesion) or to multiplication of the virus particles, or to disaggregation of the purified rod-shaped paracrystals into the smaller units of which they are composed. Unfortunately no way of proving that a single lesion is caused by a single particle of virus has been found, as can be done with single bacteria or fungal spores, and formal proof that all these types of variant are due to processes akin to mutation or saltation, and not merely to the separation of two or more unit particles, is lacking though the presumptive evidence for it is strong.

Resistance and Susceptibility

The cultivation of resistant varieties of crop plants has not proved as yet to be such a potent weapon in the fight against virus diseases, with certain notable exceptions, as it has in combating diseases due to fungi. Natural resistance against potato leaf roll and tobacco mosaic is rare, and without field evidence of

its occurrence in these old-standing diseases the search for resistance was long delayed.

In their efforts to control the destructive sugar-cane virus disease ' sereh ' in Java, the often-told story of which is one of the brightest chapters in phytopathological history, Dutch workers had a certain measure of early success in crosses between thick or ' noble ' varieties of *Saccharum officinarum* and the thin Indian variety Chunnee of the species *S. barberi*. Some of these proved tolerant also of mosaic, yielding almost as heavily when infected as when healthy, and their use, especially the P.O.J. variety ' 213 ' introduced into cultivation there in 1917, saved the cane-growing industry in Tucumán, Argentine, from imminent extinction. In high temperatures, however, these varieties tend to lose their tolerance. Later crosses of *S. officinarum* with Kassoer, a presumed hybrid between it and the mosaic-immune wild species *S. spontaneum*, yielded forms such as the P.O.J. ' 2,700 ' and ' 2,800 ' series which proved highly resistant to mosaic and in some cases to sereh. These rapidly spread throughout the tropics. In other countries also varieties resistant to mosaic were bred ; in the West Indies crosses between the resistant Java canes and the rich susceptible canes bred in Barbados have proved that resistance amounting to field immunity occurs when only one-eighth or one-sixteenth of the ' wild blood ' of *S. spontaneum* is present, and in Java itself the replacement of the older varieties by P.O.J. ' 2,878 ' reduced sereh and mosaic to practical insignificance before 1928. For a time other countries fought mosaic by growing the naturally resistant Uba and other *S. sinense* varieties, but by 1931 most of the world's supply of cane sugar was derived from varieties containing the Kassoer wild ' blood '. Several of the best of these varieties are also practically immune from a third virus disease, streak, in Natal, though curiously enough not in Egypt, while in Queensland their high susceptibility to Fiji disease and downy mildew (*Sclerospora sacchari*) has caused them to be barred where these diseases are prevalent.

The sugar-cane mosaic virus does not rapidly nor easily become systemic ; varieties may resist it or tolerate it and even recover so completely that it becomes impossible to transmit the disease to healthy canes. In Louisiana the varieties tested have been grouped into those that readily take and retain mosaic infection, those that take it with difficulty but retain it, and those that take it with difficulty and readily throw it off. A further complication is introduced by the occurrence of strains of the virus, some of which infect varieties that resist others. Thus, a virulent yellow strain, first seen in 1933, caused heavy losses in practically all the commercially important varieties of cane then cultivated in Louisiana, including those that had previously withstood the disease. Furthermore, some of the improved varieties tested in Louisiana have shown so great a susceptibility to fungal diseases that they have had to be discarded. These difficulties have led to the establishment in recent years of an elaborate system of testing varieties before release for cultivation, and illustrate in striking fashion how complicated crop breeding for disease resistance can be.

In the breeding of potatoes for virus resistance a notable advance has recently been made at the research station of the Scottish Society for Research in Plant Breeding at Corstorphine. It was found that certain varieties which react to

infection by the ubiquitous ' X ' virus by a lethal necrosis of the green shoots are ordinarily free from the disease in the field, presumably because infected plants are killed off. The same happens with the ' A ' virus. King Edward and Epicure crops are thus freed from both these viruses, other varieties reacting in the same way to one or the other or to the less important ' B ' and ' C ' viruses. The breeding programme was adjusted to combine lethal reaction to all these in one variety, and already Craig's Defiance, which is intolerant of ' A ', ' B ', ' C ', and ' X ', has been produced. The analogy between this and the use of the hypersensitive reaction in breeding against cereal rusts is clear. True resistance to the ' X ' virus has been obtained in the United States in a seedling ' S 41,956 ', while another seedling variety Katahdin (' S 42,667 ') shows field resistance to ' A ' and to some extent to ' Y ' but is susceptible to ' X '. Attempts are now in progress to combine resistance to ' X ' and ' A ', the two viruses important as constituents of mild mosaic in America.

Resistance to tobacco mosaic may be the result of hypersensitivity marked by the production of localised necrotic lesions as in the species *Nicotiana glutinosa*, or the virus may become systemic but causes few symptoms and little damage, as in certain Colombian varieties of *N. tabacum* of which Ambalema is the best known. Both these types have been used in breeding mosaic-resistant commercial tobaccos. The factor causing the local-lesion reaction has been transferred to *N. tabacum* type plants both in the United States and in Russia, but the most recent American report does not speak well of them [3]. Crosses of Ambalema with good commercial types have been under test in the United States since about 1936 and are giving excellent promise as regards immunity, growth type, and quality. In Rhodesia, while Ambalema showed no trace of mosaic on inoculation, the progeny of some of its crosses with good susceptible varieties were extensively infected, though they outgrew the disease to a considerable extent.

The strawberry yellow-edge disease which is responsible for much of the deterioration of the British crop that has been marked in recent years, causes little obvious signs of infection in certain varieties, largely of Continental origin; most of these, however, are carriers of the virus. Of the two parents of the cultivated strawberry, *Fragaria virginiana* and *F. chiloensis*, the former is highly susceptible and shows the symptoms readily and continuously, while the latter is highly tolerant and usually behaves as a symptomless carrier; the commercial hybrids probably react largely according to the degree to which these two parents are represented. The woodland strawberry *F. vesca* resembles *F. virginiana* in its reaction and serves as a useful indicator plant for revealing masked infection. Breeding against yellow edge and the crinkle-virus disease which usually accompanies it cannot with safety, on present knowledge, be directed to the production of carrier varieties, for it has been reported that they may suffer progressive deterioration (the crop being vegetatively propagated) and eventually ' run out ' like such fully susceptible varieties as Royal Sovereign. For the time being, therefore, control is being attempted by the development of virus-free clonal stocks of these fully susceptible varieties and their maintenance under nursery conditions by roguing and the control of the aphis vector, *Capitiphorus* (*Pentatrichopus*) *fragariae*.

In the United States the production of virus-resistant crops has reached the

stage of commercial development in curly top resistant sugar-beet as a result of prolonged work by the Federal authorities. In 1937 the resistant U.S. '12' variety gave 12·6 tons per acre in test plots in Idaho against 0·64 ton for susceptible varieties, and this variety showed high resistance also in California in three-years' tests reported in 1938. Resistance to the same virus and to common bean mosaic has been developed in selections of the small red Mexican variety of *Phaseolus vulgaris* in Idaho so as to give field immunity from these diseases. In New Zealand a number of varieties of garden and field peas have shown high resistance to the common pea mosaic virus and their improvement is in progress.

Though the examples cited indicate that breeding for virus resistance is as yet in its infancy and shows more promise than achievement except in sugar-cane, one of the most striking instances of control of a plant disease in the course of a very few years by the selection of resistant strains is afforded by the leaf-curl virus disease of cotton in the Sudan. Leaf curl was first seen in the Gezira in 1923, and within ten years was present in every field in the 200,000 acres of irrigated cotton in this area, often causing 100 per cent. infection and at times almost complete loss of crop. Individual plants of the Sakel variety of *Gossypium barbadense*, chiefly grown in the Gezira, showed marked resistance to leaf curl; two selections from these, X '1,530' and X '1,730', which had only 3 and 2 per cent. of leaf curl respectively, in a test in which the main crop showed 91·5 per cent., proved suitable for cultivation and most of the area was sown with them, especially the latter, within a few years. By 1938 leaf curl had been reduced to negligible proportions, though late attacks were still seen. Strains of the virus seem to exist, and there has been some recrudescence of severe infection possibly due to this cause in the last couple of years; there are now, however, no grounds for the fears expressed when the epidemic was at its height, that leaf curl would render uneconomic the cultivation of the valuable *G. barbadense* cottons in this important area.

The genetical bases of reaction to virus diseases have been determined in some plants. Separate dominant genes seem to be responsible for the lethal necrotic reaction to potato viruses 'A', 'B', 'C', and 'X' mentioned above; the resistance to 'X' of the U.S. Seedling '41,956' has been determined to be due to two necessary cumulative dominant factors. The resistance to tobacco mosaic of Colombian varieties of *Nicotiana tabacum*, including Ambalema, is due to two necessary recessive genes, and the local necrotic reaction of *N. glutinosa*, chillis, and *Browallia speciosa* to this virus depends on a dominant gene; modifying factors have been found to influence the results in most of the crosses studied. Resistance to lucerne mosaic in some of the varieties of *Phaseolus vulgaris* has been found to be due to duplicate dominant genes.

As mentioned in an earlier chapter, a method of inducing immunity in virus-susceptible plants was reported from Java in 1931; tobacco infected with the common green mosaic virus could not then become infected by the whitish-yellow strain, and vice versa. Independent investigations at Cambridge reported in 1933 showed that the same result followed the inoculation of tobacco with a green and a yellow strain of potato virus 'X'. Subsequent work has fully confirmed the existence of this kind of induced immunity and has also shown that plants which have recovered from virus attack may remain outwardly immune from reinfection

with the same virus. ' Recovered ' plants, however, may continue to harbour the virus in a latent or ' diluted ' state though it is still fully active when tested on susceptible plants ; the power to resist reinfection may sometimes be capable of transmission from plant to plant by grafting, and it has been suggested that this is an example of passive immunisation. There is some evidence that the degree of protection given by one virus strain against a second related one is quantitatively proportional to the amount of the first virus present in the tissues, and it is suggested that in some way there is an intense competition within the plant between related viruses since it is only with these that acquired immunity is found. Sometimes the presence of a virus does not prevent the invasion of the tissues by a second related one but only masks or modifies its symptoms, so that there appears to exist a form of dominance of one strain over another ; this may be complete or partial or may be temporary, being replaced after a time by mixed patterns of the two types of symptom. When the two strains are inoculated simultaneously, one may become dominant or the two may act in equilibrium, and a mixed pattern result ; complete dominance is sometimes shown at the tip of tobacco plants inoculated with a mixture of two strains of ordinary tobacco mosaic, while lower down the symptoms of both are apparent on the same leaf. Recent work does not support the analogies that were earlier drawn between the immunity acquired by ' vaccinating ' plants with weak virus strains and that conferred by vaccination against human and animal diseases ; there is no evidence of antibody formation in the plant, and the observed facts point rather to a pre-emption by one strain of the sites of multiplication of the second. When the two viruses are not related, one does not prevent the multiplication of the second.

Nevertheless, viruses produce serological reactions when inoculated into animals and can induce the formation of antibodies in the animal's serum. These antibodies, like those resulting from the presence of animal pathogens, neutralise the virus which caused their production and also give the precipitin reaction by causing a precipitate to form with sap from infected but not from healthy plants. The reaction is ordinarily specific and given only with related viruses, e.g. those which have the power of inducing immunity against one another ; a few exceptions, however, have been reported, as in potato viruses ' A ' and ' Y ' which are serologically related but do not confer immunity against each other, and differ in other ways. Mere failure to immunise is by itself an unsafe criterion of specific difference, since it is well known that strains exist of human and animal viruses (such as influenza and foot-and-mouth disease) which, while causing identical symptoms, afford no immunity against one another. It has been found possible to separate allied strains of a given virus serologically, as they can produce specific strain antibodies in addition to those common to all strains of the virus. The serological reaction sometimes affords a useful method for field identification of virus diseases.

Another property which plant viruses share with many animal pathogens is that of becoming attenuated in virulence by passage through appropriate hosts. The sugar-beet curly top virus has been attenuated by passage through the nettle-leaved goosefoot (*Chenopodium murale*) and remained stable even after several generations in older sugar-beet leaves, but regained virulence in the chickweed (*Stellaria media*) and sometimes in sugar-beet cotyledons or early

leaves. Attenuation of potato virus 'Y' has been observed in *Schizanthus retusus*, and also as a result of long cultivation in tobacco.

Morbid Anatomy and Histology of Virus Diseases

There is often no apparent change in the tissues and cells of virus-infected plants. A good many viruses, however, produce necroses in various organs, some produce distortion or abnormal development of organs, with or without hyperplasy or hypoplasy, while intracellular disturbances are not uncommonly marked by inhibition of plastid formation or the presence of abnormal cell inclusions.

In many of the true mosaic diseases there is some hypoplasy in the tissues of the lighter green or yellow areas of the leaves, the palisade cells becoming short and almost isodiametric or even, in early infections, remaining undifferentiated from the rest of the mesophyll, and all the cells and intercellular spaces being smaller than usual so that the thickness of the leaf is reduced. In cotton leaf curl large intercellular spaces are found in the rectangular palisade tissue.

The necrosis of the leaf blade produced by viruses which cause local lesions has been studied at Rothamsted in *Nicotiana glutinosa* inoculated by the strain of tobacco mosaic responsible for aucuba mosaic of the tomato in England. It begins by the formation of a dark-staining band between the cells of the epidermis and spongy parenchyma or occasionally between those of the palisade tissue, and extends through the leaf all around the inoculated spot. Nuclear, but not cell, divisions occur in the area enclosed by the band, the cells die and dry out, and the dark-staining material, which is very insoluble and chemically refractory, appears to prevent the virus from passing to cells outside the lesion.

Three types of internal necrosis of stems have been distinguished in certain virus diseases of the potato. In the 'acronecrosis' or killing of the top of the plant which may be caused (amongst others) by potato viruses 'A', 'B', 'C', or 'X', necroses develop especially towards the top of the stem, starting usually in the internal phloem and spreading to the adjacent parenchyma. A phellogen occasionally arises later in the parenchyma and tends to isolate the necrotic area by a ring of cork. The necrotic process begins by a thickening and separation of the cell walls of the primary phloem elements, the intercellular spaces thus formed becoming filled with a yellowish-brown gum-like substance. Similar changes occur in the phloem parenchyma and often cause collapse by pressure of the sieve-tubes and companion cells. The necrosis spreads to the xylem parenchyma and may even involve the vessels. Sometimes the outer phloem shows similar necrotic changes. The petioles are also involved, so that individual leaves are killed. In the tubers, necrotic patches may form like those in the stem, except that the cells of the storage parenchyma near the phloem are more extensively suberised and the necrotic areas always become eventually isolated by cork.

In the second type, the 'acropetal' necrosis caused by potato virus 'Y', the collenchyma in leaf veins, petioles, and stems is mainly affected, the vascular tissues remaining normal. Spread occurs vertically, causing streaking of the green parts, and also along the collenchyma around the stem. The cell walls become suberised and the cell contents disappear or are replaced by gummy matter.

The third type is characteristic of potato leaf roll, and is restricted to the phloem (Fig. 175). The walls of many of the primary internal and external phloem groups are thickened and lignified and small spaces appear between them, filled with a brown deposit. Eventually the phloem throughout the plant is affected, though the underground parts may be reached late; a visible 'net necrosis' due to browning of the phloem strands of the tubers occurs in some varieties. In severe cases pressure may destroy the elements of the primary phloem. The first cells to show lignification are usually those near the fibres, though occasionally it starts at the centre of a phloem group.

Among other virus diseases affecting chiefly the phloem, 'curly top' of sugar-beet in the United States, Fiji disease of sugar-cane, and 'bunchy top' of bananas in the East, show marked tissue abnormalities. In curly top, degeneration begins by hypertrophy, followed by death of some of the phloem parenchyma cells nearest the sieve-tubes (Fig. 176). Cells further out then divide, and a thick-walled hyperplastic mass of incipient sieve-tubes without sieve-plates, but sometimes with companion cells, results. New phloem elements are still formed by the cambium but they consist mostly of imperfect sieve-tubes with companion cells and parenchyma. This parenchyma, with the pericycle, enlarges and divides

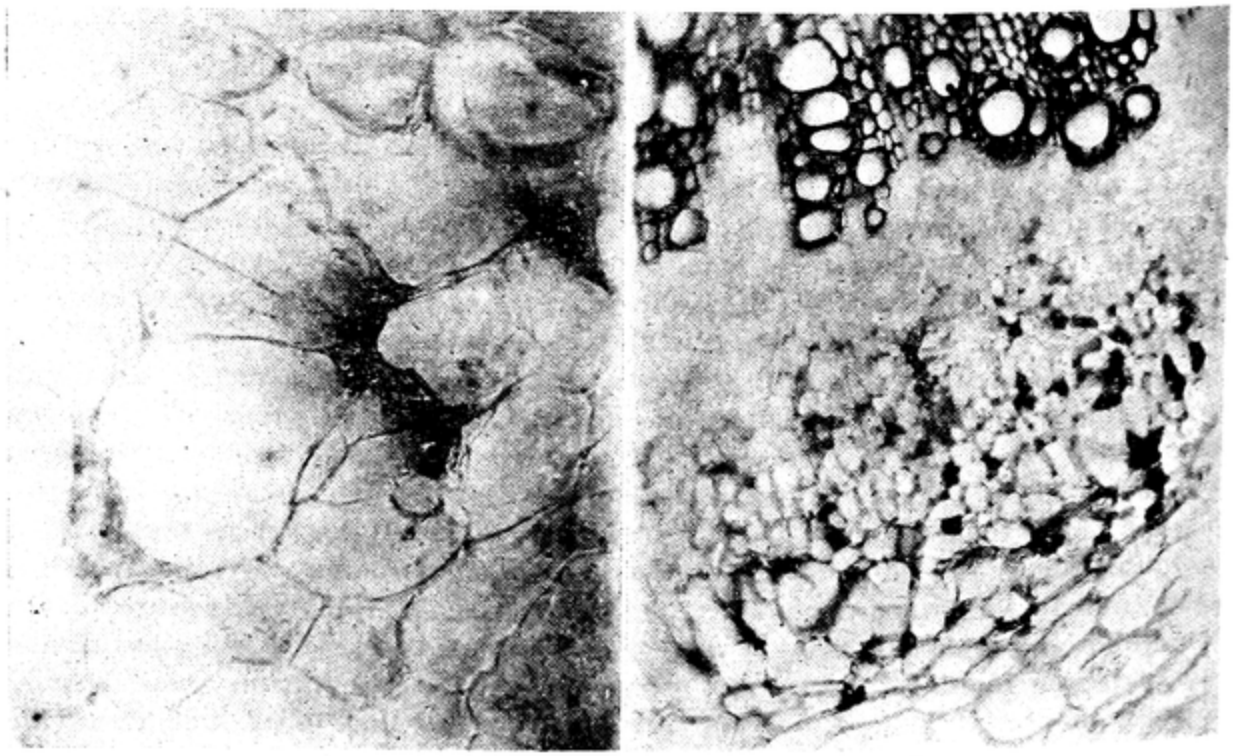


FIG. 175.—Phloem necrosis in potato leaf roll. Infection of potato variety King Edward with leaf-roll virus. *A*, in the centre is a necrotic phloem group; the cells are crushed and distorted so as to be individually indistinguishable; the whole group is shrunken and the cells immediately around it are enlarged and distorted so that they appear to radiate from it; at the lower right-hand corner of the figure is part of a normal phloem group. *B*, showing the irregular development of the secondary xylem and phloem, from the wavy band of cambium; most of the external primary phloem groups are necrotic and consequently heavily stained. (*A*, ×450; *B*, ×140. From hand sections of stems stained with phloroglucinol) (after Sheffield, *Ann. App. Biol.*)

into a callus-like tissue which may push up into small warts on the under surface of the veins, while the sieve-tubes and companion cells die and collapse. In the roots of infected beets the chromatin first increases, then breaks down with the cytoplasm. Other cells show a great increase in calcium oxalate and leucoplasts.

The changes in the phloem in bunchy top begin in the same way, but the hypertrophy and hyperplasia extend to the pericyclic fibres and ground parenchyma, which are largely transformed into a small-celled tissue rich in chlorophyll and visible as characteristic dark-green stripes bounding the veins.

In Fiji disease the galls always show on the under side of the leaf, but whether they originate in the phloem or are a product of more generalised meristematic activity is uncertain. The principal feature is still a hyperplastic phloem with callus-like proliferating parenchyma; this gives rise to sieve-tubes and tracheids.

The vein-thickening which precedes and accompanies the development of enations in leaf curl of cotton and tobacco results from an increase of the primary phloem and hyperplasia of the pericycle in which bundles with central xylem form.

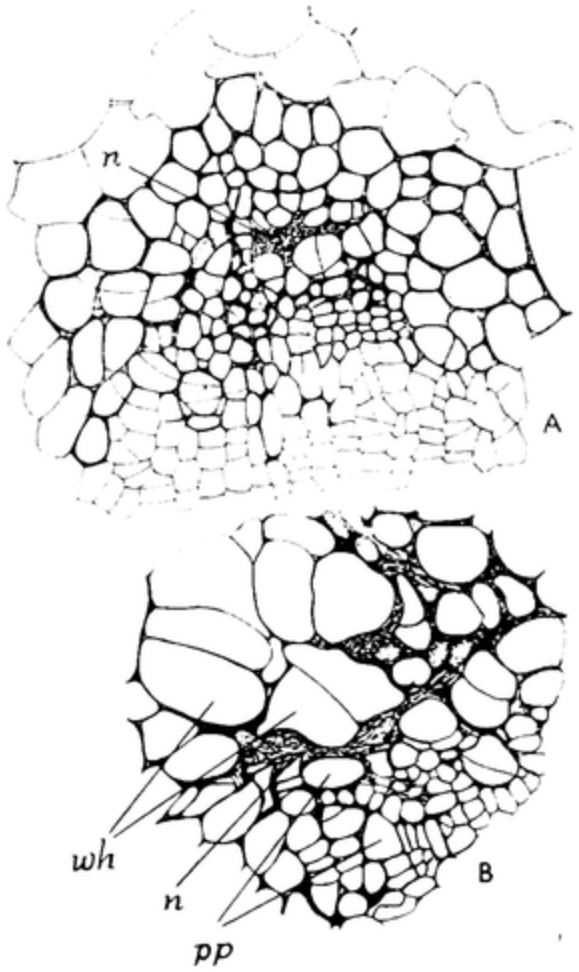


FIG. 176.—Phloem necrosis. *A*, curly top of sugar-beet. Ontogeny of the phloem in the leaves. Top, diseased phloem showing necrotic cells *n*, near the centre, and hyperplastic tissue surrounding the necrotic area; note sieve-tubes and companion cells below. *B*, diseased phloem in later stages of degeneration showing secondary necrosis and proliferation of parenchyma *wh*, near the collapsed walls; *n*, necrotic cells; *pp*, phloem parenchyma (×348) (after Bennett, *J. Agric. Res.*)

In the witches' broom of potatoes in North America, elongation of the shoots is due in part to unusually long internodes, while in potato 'spindle tuber' the tubers are lengthened by elongation of their cells, and both in tubers and shoots the ratio of length to breadth of the cells is increased.

The small galls sometimes found in stripe disease of narcissus and daffodils are due to hyperplasia of the epidermal and palisade cells or sometimes merely to their hypertrophy.

Enations develop usually, but not always, near the veins and surround a chlorotic area on the under surface of the leaf. They arise by a proliferation of the lower three or four cell layers, emerging as paired green leafy ridges or shallow cup-like protrusions. All the cells may undergo hyperplasy, until growth ceases. The fully formed enation in tobacco mosaic has two epidermal, a palisade, and four

spongy mesophyll layers, the palisade being on the inner side in paired or cup-shaped outgrowths and being often continued across the bottom of the cup, while the outer side of the enation resembles the under surface of the leaf. On tomato the enations are similar but in a type associated with ' fern leaf ' in Italy, warts composed of two or three layers of hypertrophied epidermal cells and ridges due to proliferation of the mesophyll covered by a hypertrophied epidermis have been reported. No marked deviations from the tobacco type seem to have been recorded in the enations of cotton and tobacco leaf curl and pea mosaic.

In the ' big bud ' disease of tomatoes the vascular system of all the above-ground parts is stimulated. Interfascicular vascular tissue develops precociously in the stems ; the flower stalk and the elongated segment between the calyx and the corolla become polystelic ; and the phloem in the stem and inflorescence is much increased, the sieve-tubes being filled with a yellowish deposit. In the fruits the bundles are woody from intensification of lignification ; annular and spiral vessels are replaced by pitted ones. The leaf mesophyll loses differentiation and contains only a few small spaces ; in the petals, however, a palisade layer may form. The profuse development of adventitious root initials may be accompanied by longitudinal stem fissures exposing the inner tissues. The stem and petiole thickening, which may cause a remarkably bloated appearance, is due mainly to abnormal development, by hyperplasia of pith cells close to the internal phloem, of a compact tissue of small cells with a few sieve-tubes and isolated tracheids. The phyllody of the floral axis is extreme, even the ovary being sometimes replaced by small normal leaves, through the centre of which the axis may be carried by a process of median floral proliferation and terminate in a small shoot or a group of phylloid flower buds. The short, dichotomously branched apical shoot when thus formed may bear minute, hyaline papillae, thought to represent ovules. Beans infected by the virus show similar floral abnormalities [10].

The pallor or yellowing of green tissues found in so many virus diseases is due to inhibition of plastid formation and, though this seems to be less common, destruction of existing chloroplasts. The chlorophyll content is reduced from 43 to 87 per cent. of the normal by different strains of tobacco mosaic virus, the yellow pigments carotene and xanthophyll being also proportionately reduced ; in the yellow strains chlorophyllase is increased. That there is no obligate relation between chloroplast and virus is evident from the fact that viruses have been propagated for long periods in isolated root-tip cultures.

The most notable effect of some viruses upon the cell contents is the formation of the so-called ' X-bodies ' or cell inclusions, which for a time were thought by many to be visible stages of the virus. The inclusion bodies are of two kinds, amorphous and crystalline ; they occur most often in the epidermis and leaf hairs but are also found in the ground parenchyma and the phloem. The amorphous X-bodies are often amoeboid, vacuolate, with mitochondria and oil drops, and give the usual protein reactions. They are formed from aggregations of small particles in the streaming cytoplasm of the cell. The crystalline bodies or ' striate material ' may occur in the same cells or independently. They are plate-shaped, fragile, colourless, and give protein reactions ; the slightest pressure causes them to break down into needle-like fibres. They may represent condensations of the

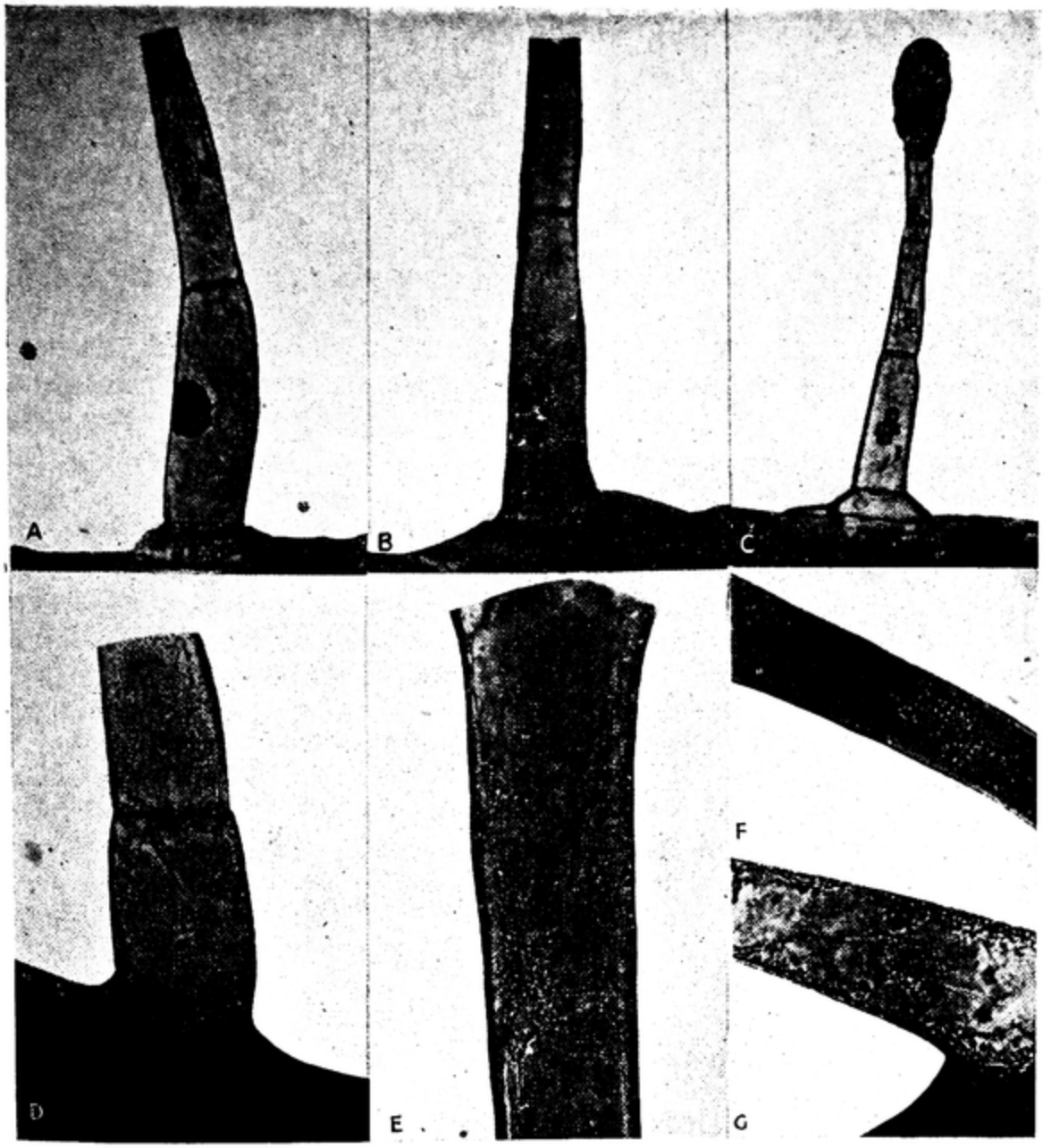


FIG. 177.—Intracellular inclusions in the tobacco (*Nicotiana tabacum*). *A*, in the upper cell most of the protein material is contained in one irregular large mass ; a fully formed body lies apposed to nucleus and wall in the lower cell. *B*, the cell contains a granular, vacuolate inclusion body, a spike-like crystal, and the nucleus. *C*, a small glandular hair ; inclusion bodies which were formed in the stalk cells have crystallised. *D*, *Solanum nodiflorum*. Basal cell from hair of normal healthy plant ; the nucleus is suspended in the centre of the cell by a few delicate cytoplasmic strands (× 300). *E*, *Nicotiana tabacum*. Hair cell a few days after infection with tobacco mosaic virus. The cytoplasm is much more conspicuous than in the normal cell and appears to have increased in bulk (× 350). *F*, *Nicotiana tabacum*. Inclusion body produced in cell of trichome as a result of infection with *Hyoscyamus III* virus (× 300). *G*, *Solanum nodiflorum*. Inclusion body produced in cell of trichome on infection with aucuba mosaic virus (× 300). All photomicrographs were taken from unstained living cells. *A*, *B*, *C*, show infection with aucuba mosaic virus of tomato (after Sheffield, *Ann. App. Biol.*)

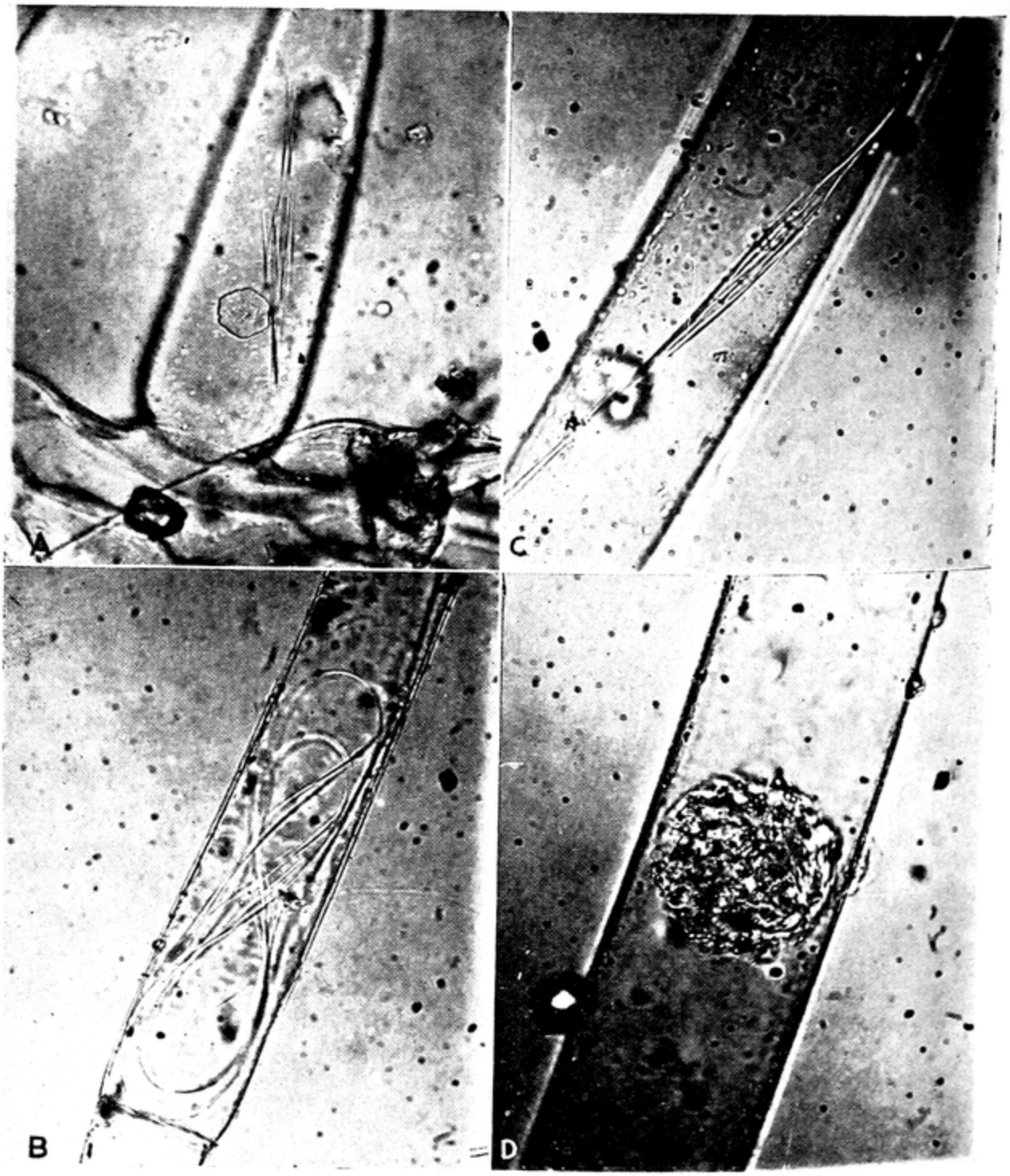


FIG. 178.—Variations in cytoplasmic inclusions. *A*, tobacco infected with tobacco mosaic virus; a crystalline inclusion and a mass of long fibres occupy the centre of the cell. *B*, tomato infected with aucuba mosaic virus; long fibres curve to form a figure of 8. *C*, the same; a spindle-shaped body appears to be an aggregation of long spindle-like fibres. *D*, tomato infected with aucuba mosaic virus; an inclusion body containing amorphous material and a few small crystals (after Kassanis & Sheffield, *Ann. App. Biol.*)

virus protein, while the X-bodies are more probably reactions of the cell cytoplasm. Small plate-like inclusions have also been found in the nuclei of solanaceous plants infected with the ' severe etch tobacco ' virus ; these are more stable than the cytoplasmic inclusions [11]. Isometric deeply staining crystals, 0·3 to 4 μ in diameter, occur in the nuclei as well as the cytoplasm of broad beans infected with certain pea and bean viruses and are said closely to resemble those of severe etch [8] (Figs. 177, 178).

Losses caused by Virus Diseases

Though the damage caused by potato ' curl ' was widely recognised a hundred and fifty years ago, many virus diseases are so insidious in their onset that the injury they do may not be appreciated or, more often, is attributed to other causes. There are, however, some virus diseases that are invariably fatal : ' peach yellows ' always kills the affected trees ; a ' spiked ' sandalwood tree dies some fifteen months to two years after the first symptoms are seen ; ' rosetted ' peaches nearly always die the following autumn or winter ; while the potato varieties that react by ' top necrosis ' to certain viruses are usually killed off before they produce tubers. Several viruses cause complete loss of crop without killing the plant, as in ' groundnut rosette ' and bunchy top of bananas. From these obvious extremes of damage, however, to the large group of viruses that cause only mild symptoms or even, in carrier varieties, none at all, every gradation can be found in the severity of the resulting injury.

The annual toll taken by virus diseases from the potato crop of England and Wales can be assessed with considerable accuracy at not less than £2,000,000 in normal times. In the United States the reduction in the yield of sugar beet from the 763,000 acres under the crop is reckoned to have averaged over a number of years, about 2,000,000 tons annually. ' Beet yellows ' is estimated to have caused losses in the sugar and fodder beet crops in Belgium of over 60,000,000 fr. in 1937. Louisiana is calculated to have lost sugar to the value of over 100,000,000 dollars from sugar-cane mosaic in the ten years from 1922, though at the beginning of that period there was still local doubt whether even 100 per cent. infection did much harm. In the Argentine, mosaic caused a progressive reduction in sugar-cane yields in Tucamán from the beginning of the century, reaching its climax in 1915 when the average yield for the previous five years was only 9 tons per acre. The introduction of Java mosaic-tolerant canes such as P.O.J. ' 36 ' and ' 213 ', giving yields of 20 to 30 tons per acre, saved the industry however, and by 1926, when they had completely replaced the older varieties, the province had the largest sugar yield (375,000 tons) in its history.

Many virus diseases of market garden crops and fruit also cause heavy financial loss. ' Lettuce yellows ', due to infection by the ' aster yellows ' virus, was recently estimated to have reduced the lettuce crop of New York State by an annual average of 5 or 6 per cent. from 1921 to 1938 ; bean mosaic caused about the same loss of crop in Iowa from 1924 to 1926 ; cucumber mosaic 7 to 15 per cent. in Iowa from 1924 to 1926 ; and 10 and 15 per cent., respectively, in Kansas and New York in 1928. The average loss from tomato mosaic in Utah in 1925 was 8 per cent.

Naturally much higher individual losses than these average figures occur and many have been determined in experimental tests. Sugar-cane mosaic has caused a loss of 80 per cent. in Hawaii and over 60 per cent. in India. In tests in Ireland leaf roll reduced the yield of British Queen potatoes from 9 to 3·7 tons per acre and of Ally from 11·4 to 2·7 tons. When tubers from leaf-roll plants were used as seed for the next crop in Wales, the losses for three consecutive years were 55·8, 45·6, and 52·6 per cent. respectively, showing that this disease can produce its maximum effect in the year after infection ; very similar figures were obtained in a three-years' test in England. In the United States lettuce yellows can cause 70 or 80 per cent. of a crop to be lost and the spotted wilt virus can destroy three-quarters of the yield ; soy beans can lose 30 to 75 per cent. of the beans and ordinary beans 50 per cent. from the mosaics of these crops. The rapid spread of bunchy top of bananas in New South Wales led to a falling off of the yield from 650,000 bunches from 4,750 acres in 1922 to 91,144 bunches from 1,002 acres in 1925, and many growers were ruined. Early infections of tobacco mosaic can reduce the yield in North Carolina by 30 per cent. and the value by up to 50 per cent., and infection of tomato mosaic in Maryland can reduce the crop by over 50 per cent. In glass-house experiments in England early infections of tomatoes with the streak virus and tobacco mosaic virus caused yield reductions exceeding 20 per cent. Early infections with groundnut-rosette virus cause total loss ; for no nuts develop after infection. In a test in Canada, raspberry mosaic reduced the fruit yield by 28 per cent. by weight.

Even such losses as these by no means exhaust the destructive possibilities of virus diseases. The record of potato varieties in Great Britain and of sugar-canes in Queensland, the West Indies, and elsewhere shows many instances of the disappearance from cultivation of very valuable types owing to susceptibility to some virus disease. ' Running out ' of the best varieties of strawberries and raspberries from the same cause is a present danger in Great Britain and North America. Even the tolerance of infection found in carriers may do no more than disguise this deterioration ; the 30 per cent. loss from potato virus ' X ' in certain carrier varieties in Australia was unsuspected until revealed by tests, for the crops were mostly uniformly infected, while carrier varieties of strawberries may at times suffer as great a deterioration from latent yellow-edge virus infection, as the highly sensitive Royal Sovereign crop.

Another possible additional source of loss from virus diseases is the increased susceptibility of virus-infected plants to bacterial and fungal diseases. While not as evident as the enhanced susceptibility to certain human diseases caused by the influenza virus, there is a rather widespread belief that potatoes are rendered more susceptible to blight (*Phytophthora infestans*) by becoming infected by viruses, and in Brazil a similar relation between tomato viruses and *Septoria lycopersici* has been claimed.

Control

A direct attack on virus diseases by the chemical methods so extensively used against parasitic fungi is out of the question. Viruses cannot be reached by these means and, at the most, control of their insect vectors may be undertaken when

the economic conditions permit. Practical control of the aphis vector of strawberry yellow edge and crinkle has been achieved in some cases in England by the use of vaporised nicotine. In commercial crops of the biennial drug plant, henbane (*Hyoscyamus niger*) a considerable reduction of the vector, *Myzus persicae*, was effected by weekly spraying with nicotine and soft soap during the first two months of vegetation in the first year. This gave a 30 per cent. increase in yield in the third crop, harvested during May of the second year.

Roguing for the elimination of virus-diseased plants has had a considerable vogue, especially in crops propagated vegetatively such as potato, sugar-cane, strawberries, bananas, and the like; it is still practised extensively where the crop is valuable and the amount of infection moderate. Its principal use nowadays, however, is in the maintenance of ' foundation ' or nursery stocks of disease-free plants which can be used for the planting of the main field crop. Where insect vectors can be kept down fairly readily and the plants are under observation closely enough for early infections to be recognised, as in glasshouse cultivations, roguing is of much value, as it is also, obviously, where the virus is seed-borne or the crop is propagated vegetatively and a healthy parentage is, therefore, essential.

Where the infection arises anew each year in an annual crop, success in reducing the source of infection by the destruction of ' volunteer ' plants or out-of-season plantings of the crop, which may serve to carry over the disease between successive main crops, has sometimes been considerable. Much benefit resulted from eradicating volunteer cotton in the Sudan to reduce early attacks of leaf curl. In Southern Rhodesia a notice under the Tobacco Pest Suppression Act, 1933, prescribed the 1st August as the date by which destruction of all growing tobacco plants is to be completed as a control measure against tobacco leaf curl; this close season appears to cause the vector (*Bemisia* sp.) to die out [5]. In California the enforcement of a celery-free period in districts where western celery mosaic causes severe damage has been effective in raising the yield, which had fallen from over 1,000 half-crates per acre in 1930 to 311 in 1934, to an average of 1,100 in 1939 and 1,000 in 1940 [9]. The growing of maize in the neighbourhood of sugar-cane fields has been discouraged for similar reasons as it is susceptible to sugar-cane mosaic and a favourite food plant of the insect vectors; this ban was enforced by legislative action in certain areas in Barbados in 1927.

Sometimes the eradication of weed hosts of the virus or of its insect vector has given good results. Destruction of the Russian thistle, *Salsola kali-tenuifolia*, on which the beet curly-top vector, *Eutettix tenellus*, and the virus are largely maintained before passing to the crop, has effectively reduced sugar-beet curly top in California. Excellent results in controlling the yellowish-green mosaic of cucumbers in the United States, where it has many hosts, have followed the eradication of certain weeds, especially perennials. In England, where, however, this mosaic is less common in glass-houses than the mild or green-mottle mosaic which seems to be confined to the *Cucurbitaceae*, the houses in which it occurs should be kept free from susceptible flower crops (primulas, asters, lobelias, and so forth) and the common bryony should not remain near cucumbers or marrows. With the great prevalence of the spotted wilt virus amongst ornamentals, keeping

the latter out of houses containing tomato seedlings is most important, while glass-house control in tomatoes and flower-crops alike must include elimination of the thrips vector from an infected house before another susceptible crop is grown ; thrips control out of doors, however, is difficult.

The complete eradication of these diseases by destruction of all infected plants is being effectively carried out by large-scale campaigns amongst perennial plants. It was early advocated (with legislative sanctions in some areas) for the control of peach yellows and rosette in the United States and later was recommended against sandalwood spike, together with the establishment of a sandalwood-free belt round the infected area in South India. Recently, powers have been taken in the Gold Coast to remove plants such as cacao from declared areas in order to check the spread of diseases, such as the swollen shoot virus disease of cacao, to uninfected areas. The object is to establish zones free from all cacao around the infected areas. The Psorosis Act of 1927 provides for the destruction, without compensation, of any citrus tree infected by psorosis within the Union of South Africa, together with quarantine measures against new introductions of the disease; as infection may remain latent in the tree for 20 to 30 years and seldom shows before the trees (which are ordinarily infected by budding with diseased wood) are 9 or 10 years old, it will be long before the effectiveness of the measure can be judged. In 1939-40, however, only 1,468 affected trees were found in a total of 513,123 inspected. When peach mosaic was first seen in Colorado in 1934, and California a year later, eradication was promptly undertaken ; in Colorado only 3,100 fresh cases were found in 1937, compared with 9,835 in 1936 and 30,467 in 1935, while in the four years from 1936 to 1939, 89,355 cases were found in California, 63,651 of which were removed. The attempt to eradicate the phony peach disease from the south-eastern States is more formidable; during 1936 over 2,000,000 abandoned and 3,500,000 ' escaped ' peach trees were removed in 11 States by a force of 2,000 men employed under the Emergency Relief measures, while altogether over 21,000,000 trees were inspected in 20 States and 146,072 of the 156,977 found infected were removed. In New Zealand an attempt is being made to eradicate ' onion yellow dwarf ' disease which was found to be confined to a small area in 1939.

The progress that has been made in the breeding of crop varieties resistant to, or tolerant of, virus diseases has already been discussed. In the opinion of many, this remains the most hopeful line of attack on them. Control by ' vaccination ' with a weak strain of the virus concerned, so as to induce immunity from more virulent strains, has been regarded as practicable by some. No satisfactory case of its use under field conditions seems to have been reported as yet and much further testing is necessary before the many risks associated with the use of such ' live ' vaccines can be discounted.

A considerable measure of success has sometimes followed the adjustment of agronomic practices so as to reduce the prospect of infection. The most effective has been the alteration of the date of planting the crop so that it is too far advanced for serious injury by the time the insect vectors become numerous ; this has worked well on occasion against the curly top of sugar-beet. In Tanganyika, experiments show that cassava (*Manihot*) planted in June is less injured by mosaic than if

planted at other times, for the crop then passes the main period of its growth when the probability of infection is at its lowest. Similarly in parts of Nigeria the native tobacco plantings escape serious injury from leaf curl because the vector (*Bemisia* sp.) has become scarce by the time the seedlings are transplanted. Late sown oats and other cereals (after the 20th May) in Siberia are not severely injured by the Zakooklivanie or 'pupation' virus disease, because the vector, *Delphacodes striatella*, which over-winters in the larval stage on grass weeds about the cereal fields, migrates in the winged form too early to be able to dwarf the plants. Infection by the groundnut rosette virus in Africa has been found to be considerably lessened by dense planting and also where grass mulching of the soil is practised; in the Gambia earlier sowings are less damaged than those that give young plants when the vector, *Aphis laburni*, is most active in July and August. Possibly, as with the vectors of leaf roll of potatoes, the activity of this aphis is reduced by high humidity.

The chief measure employed for the control of virus diseases of the potato and some other crops in the British Isles and elsewhere is the inspection and certification of planting stock. For this purpose an elaborate organisation has been built up by various Government Agricultural Departments. In areas in the British Isles where potato seed production is important, the inspection covers purity to type, as well as the amount of virus infection. In Scotland, for instance, over 50,000 acres grown for seed are inspected annually and certificates granted by the Department of Agriculture of different designations according to purity and the percentage of virus diseases found: 'Stock Seed' must be 99·5 per cent. true to type and practically free from virus symptoms except negligible mottle; 'T.S.(A)' is 99·5 per cent. pure and with not more than 1 per cent. total mild mosaic, severe mosaic, leaf roll, and 'wildings', and comprises varieties immune from wart disease, the corresponding non-immune grade being designated 'N.I.(A)'; 'T.S.(H)' and 'N.I.(H)' are 99·5 per cent. pure and with not more than 3 per cent. severe mosaic, leaf roll, and 'wildings'. These are all regarded in England as Class 1 Scotch seed; Class 1 Irish, and Class 1 Special English and Welsh stocks are also recognised, the latter from certain limited areas only. Recently certification has been extended to cover English crops which, though ordinarily grown for consumption, can, under certain circumstances, serve as sources of good seed; such crops are eligible only if grown from Class 1 seed and at least 50 yards from any crop not similarly grown. In Canada and Holland also there are important seed potato industries and a very elaborate certifying organistation exists; in 1935 the acreage certified in Canada was 83,537 [15]. Many other countries have adopted similar measures.

Methods for the same purpose in the United States vary to some extent from State to State but are kept in general consonance by periodical conferences at which Canada is represented. Considerable use is made of the tuber unit method of establishing clean stock for planting the main crop: a portion of each tuber bearing an eye is tested under glass before planting time and only if it gives a healthy shoot is the rest of the tuber used. The resulting seed plot is grown in isolation and carefully rogued to eliminate any virus diseases that may appear. Naturally, localities are selected for seed production where the risk of spread

of virus diseases is low. By this method in commercial practice cases have been recorded in New York State of the reduction of virus diseases from 2·6 per cent. in 1924 to 0·4 per cent. in 1932, and in Quebec of an increase in yield from 287·7 bushels per acre in 1928 to 417 in 1934. The outstanding importance of the ecological conditions controlling the insect vectors of these diseases is sufficiently demonstrated by the fact that there are areas in Ireland and elsewhere in which potato varieties have been maintained free from degeneration for many years. Early Rose potatoes have been grown on bog-land in central Ireland since 1867 without change of seed and without running out, and almost forgotten varieties such as Pink Eyes and Lumpers on the western seaboard for much longer periods. In these areas the insect vectors are rare and the cool moist climate is adverse to their dispersal.

The bunchy-top disease of bananas in New South Wales and Queensland has been brought under fairly effective control by a series of measures directed to the eradication of diseased plants and the prevention of spread. All plantations are registered and inspected, diseased plants are removed after treatment by prescribed methods to destroy the vectors (*Pentalonia nigronervosa*) present on them, diseased areas are quarantined so that no banana material may be moved from them, in the quarantined areas the land must be kept free from weeds for not less than 6 feet from each banana plant, all banana plants in other than registered plantations must be destroyed, and every care must be taken in the plantations to recognise the early symptoms of infection. Permission to sell planting material must be obtained, and is granted only to growers of disease-free stocks. In certain areas free from the disease no planting material from the infected areas is allowed ingress. These stringent measures, applied under legislative authority in 1927 and varied from time to time, have been effective in restoring the New South Wales banana-growing industry — probably infected from Fiji in 1913 and which had fallen between 1922 and 1925 to a seventh of its previous production, back to far more than the old figures by 1935 — and rank amongst the best examples of plant-disease control by administrative action based on sound scientific investigation [7]. Similar methods are applicable to other virus diseases in advanced countries, especially in so far as they relate to the inspection and certification of nurseries supplying planting material. In New York State, for instance, all raspberry nursery stock offered for sale must be regularly inspected, rogued when necessary, and certified reasonably free from virus diseases. A test of the same procedure in England has broken down owing to the prevalence of carrier varieties and others with a low expression of symptoms.

From this survey it will appear that the question whether the viruses are ' animal, vegetable, or mineral ' is largely of academic interest only. As agents of disease they behave sufficiently like the bacteria to allow of the application of the same considerations to the study of their behaviour and control. In particular, the problems of their origin, dissemination, and prevention, which at one time seemed so difficult as to lead many plant pathologists almost to despair of their solution, have yielded to intensive research sufficiently to give good grounds for the hope that the practical outcome will eventually equal that achieved in the fight against parasitic fungi and bacteria.

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Chapter IX

DEFICIENCY DISEASES OF PLANTS

INTRODUCTION

It has long been known that other elements besides those contained in the so-called complete ' fertilisers ' used by farmers are of importance for the growth, development, and fruiting of plants. Though nitrogen, phosphorus, and potassium are the main elements in which cultivated soils most frequently have inadequate supplies for the nutrition of plants, symptoms of distress curable by applications of iron have been familiar to growers, especially of the vine and of woody orchard trees, for generations. Calcium, too, exercises a marked effect on the vegetation and has been widely held to have an influence in nutrition, apart from its better known action in improving soil structure, facilitating the availability of other plant foods, and providing lime for farm livestock.

Of recent years knowledge of the crop disorders due to deficiencies in one or more of a whole range of other elements has rapidly accumulated. These elements have come to be known as ' minor ' or ' trace ' elements, not because they are necessarily present only in traces in the soil, but because the plant usually contains little of them and can get enough from most soils for its proper functioning. Boron, manganese, zinc, copper, magnesium, and sulphur are amongst the elements which have been so far implicated in the causation of the deficiency diseases of crops, and there is good reason to believe that this list is not complete. Not all the species of plants are equally prone to these troubles, which are sometimes also rather narrowly associated with certain soil types.

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IRON

The chlorosis of the vine and of fruit trees found in soils rich in lime or manganese and due to failure to obtain sufficient iron in suitable form in the green parts is discussed in the early literature on plant diseases. It has been especially noticed in the vine, pear, apple, quince, peach, apricot, plum, cherry, raspberry, walnut, orange, lemon, pineapple, rice, maize, lupins, cowpeas, conifers, and some vegetables. It always seems to be due not so much to an absolute deficiency of iron in the soil, for that scarcely ever occurs, as to some condition which reduces the availability of iron to the plant, or immobilises it within the plant. The chief ultimate causes of these conditions are the presence of excess of lime (' lime-induced chlorosis ') and of manganese (' manganese-induced chlorosis '), respectively.

Lime-induced Chlorosis

Lime-induced Chlorosis of the Grape Vine

Chlorosis of the grape vine is a familiar trouble in the vineyards on the marls and other calcium-rich soils of certain districts of France, Germany, and Switzerland. Its routine control by applications of ferrous salts has been successfully achieved by the intelligent viticulturists of these regions for many years. The methods used may be divided into three groups: summer spraying with weak solutions, winter treatment by swabbing with strong solutions applied after pruning, especially with a view to absorption through the pruning cuts, and soil applications around the base of the vines. The injection method used with orchard trees is little practised in vineyards owing to the habit of the vines.

Summer spraying or dusting is generally carried out as soon as the first signs of pallor are noticed; if delayed until yellowing of the foliage occurs, little benefit results. Ferrous sulphate at a concentration of 1 per cent. is ordinarily used, sometimes with the addition of citric acid at the rate of 150 gm. per hectolitre of the iron sulphate solution with the object of preventing the conversion of the ferrous to the insoluble ferric salt through the formation of an iron-citrate complex in which the iron is no longer present in the ferrous or ferric forms. A second application may be required later.

For winter applications pruning is done early (November) and the whole vine then swabbed with a solution of 25 to 40 per cent. ferrous sulphate. In this case also citric acid is sometimes recommended at the rate of one-fourth to one-fifth by weight of the ferrous salt; the solution can be made up in the summer and evaporated for use after re-dissolving when required. The winter treatment is often followed by summer spraying during the next season. Both summer spraying and post-pruning swabbing can cause injury to the vines. Spraying is often followed by slight burns on the foliage, but the margins round these turn deeper green and the green gradually spreads over the whole leaf. Swabbing may burn the exposed wood and bark at the pruning cuts.

The addition of ferrous iron to the soil is less certain in its action than the method mentioned above, but is employed to some extent. Crystalline or powdered iron sulphate is placed in a trench some 10 cm. from the base of the vine, about 300 gm. of the salt being used per vine. The disease is also combated by the use of the so-called calcium-tolerant varieties or species of vines. The European *Vitis vinifera* group is in general less susceptible to lime-induced chlorosis than some of the American species which became so extensively used as stocks in Europe owing to their resistance to *Phylloxera*. There are various records of hybrid and other stocks that can be successfully cultivated on chlorosis-inducing soils. Thus at Lausanne in Switzerland good results were obtained by hybrids between Mourvèdre (*V. vinifera*) and Rupestris (*V. rupestris*) in heavy compact soil containing 48 to 60 per cent. available lime. In the United States chlorosis of the susceptible Concord (*V. labrusca*) grape has been successfully overcome by grafting on *V. vinifera* stocks such as Muscat, Tokay, etc [28].

Lime-induced Chlorosis of Fruit Trees

In England lime-induced chlorosis is in the main an orchard trouble, correlated with soils derived from certain limestone formations, rich in carbonates. Treatment of apple chlorosis with ferrous salts has been adversely criticised as being uncertain in its results (though sometimes giving temporary improvement) and injurious to the trees. Considerable control, however, has been secured by the use of permanent cover crops (grasses, clovers, weeds) in susceptible orchards. The beneficial result is believed to be due to the production from the cover crop of organic materials capable of forming, with iron, complexes akin to that given by citric acid. Although this chlorosis has often followed liming, it is difficult to relate its occurrence to either soil reaction, the water soluble iron content of the soil, or the iron content of parts of the tree.

Both in France and the United States lime-induced chlorosis of fruit trees is regarded by nearly all workers as an iron-deficiency trouble and is combated by methods similar to those used in viticulture. In some parts of southern France peach chlorosis can be very destructive ; it has been successfully controlled by sprays or swabs of 15 to 40 per cent. ferrous sulphate applied during dormancy. The treated trees recover their normal green colour, and instead of no fruit or small and unmarketable fruit, a good crop has been obtained the following summer. The effect, however, does not usually last for a second season. Cures have also been obtained by the injection of iron salts into holes bored in the trunk of the tree. The double tartrate of iron and potassium, the double sulphate of iron and ammonium, and ferric potassium oxalate have given good results when injected at the rate of one-third to one gramme of iron per tree, according to age. For summer spraying of chlorotic peaches, dilute ferrous sulphate or the double sulphate of iron and ammonium applied in May have been recommended in France. Similar results have been obtained on pears. Injection gives the most lasting results, but painting the pruning wounds and summer spraying can be used concurrently.

In orchards in the United States lime-induced chlorosis is well known : it is reported as perhaps the most serious single disease of plants in Utah and it is also severe in California, where pears are particularly injured by it. All the methods already mentioned for ameliorating the chlorotic condition have been recommended not only for tree fruits but also for shade trees. The claim has been made that definite improvement has followed the injection of ferrous salts into forest and shade trees growing in soils liable to iron-deficiency chlorosis in 95 per cent. of the trees tested. In California the injection of any soluble iron salt solution at the rate of one ounce per gallon of water is recommended for orchard trees, especially the pear. The solution is run in during dormancy, from a can into holes $\frac{1}{4}$ to $\frac{3}{8}$ inch in diameter, one or more according to the size of the tree, and extending two-thirds to three-quarters of the way through the trunk. Or the salts may be used dry, ferrous and ferric citrate being specially recommended for this purpose. Spraying with a solution of ferrous sulphate of one ounce per gallon late in the day, so that absorption may occur during the night, is another recommended practice ; and soil applications of crushed ferrous sulphate, either

in trenches 1 to 2 feet deep and 1 to several feet from the tree base, or in evenly spaced holes about the same depth and 2 inches broad in one or more rings around the tree, are followed by good results if made in late winter or early spring. Chlorotic apple trees in calcium-rich soils in Montana were found to benefit from trunk injections of a 0·25 per cent. solution of ferrous sulphate or 2 to 3 gm. of dry ferrous sulphate or ferric nitrate, watered after insertion. Even the old-fashioned orchard practice of driving 15 to 20 iron nails, 1 to 1½ inches long, into the apple trunks gave an improvement, while spraying and soil applications of ferrous sulphate were ineffectual.

Lime-induced chlorosis is prevalent in orchard and tree plantations in the south and south-east of the U.S.S.R. Apple, plum, and raspberry are amongst the fruit most affected. The trouble may be counteracted by soil applications of iron salts and sulphuric acid, the avoidance of alkaline fertilisers, and the use of tolerant varieties. The corn bindweed (*Convolvulus arvensis*) is stated to be a useful indicator plant for revealing chlorosis-inducing soils. Chlorosis of conifers in calcareous soils is sometimes a serious trouble, especially in forest nurseries in the United States, and, like the chlorosis observed in beech plantations on the chalk in England, is presumably due to iron deficiency [7].

In citrus plantations this type of chlorosis has proved troublesome in some parts of the United States and there are a few records of its control by the injection of iron salts (e.g. the citrates) into the trunk, or by applications of ferrous sulphate to the soil. In Arizona, where these methods have been used, the disease appears in 8- to 12-year-old trees in alkaline calcareous soils, and reaches its maximum when the trees are 18 to 20 years old. It is probable, however, that many of the earlier records refer to other deficiency diseases, especially that now treated by applications of zinc and known in California and elsewhere as mottle leaf.

There are several records of the existence of marked root-stock effects in orchard trees growing in chlorosis-inducing soils. Thus in Californian experiments with pears both the quince and *Pyrus serotina* proved to be more susceptible to lime-induced chlorosis than the pear (*P. communis*) stocks. Amongst commercial varieties the Bartlett, Commice, and Nelis were more susceptible than Clairgeau or Hardy. The acute type of chlorosis appears to be restricted to certain plants, but in these it causes not only yellowing of the leaves but defoliation and also death of a part of the root system.

In the U.S.S.R. chlorotic apple and pear trees have been reported to show increased susceptibility to the leaf spot and black rot caused by *Sphaeropsis malorum* (*Physalospora obtusa*), and good results in controlling the disease have followed the use of an iron sulphate spray (6 or 8 per cent.) in early spring or late autumn.

Lime-induced Chlorosis of Lupins

Another lime-induced chlorosis worthy of mention is that of the yellow lupin (*Lupinus luteus*), which has been extensively reported from Central Europe on calcareous or limed soils. This plant is generally held to be a calcifuge, but the chlorosis appears to be due rather to a disparity between calcium and available iron than a strict calcifuge habit; manuring yellow lupins with magnesium car-

bonate produces a similar chlorosis to that induced by lime, and this can be equally cured by applications of iron. The perennial lupin (*L. perennis*) is not a calcifuge and the blue (*L. angustifolius*) is less so than the yellow. A similar relationship seems to hold with manganese as with calcium in these plants, and chlorosis curable by applications of iron has been induced in *L. luteus* by excess applications of manganese salts. Since similar results have also been obtained with ammonium salts used as a top dressing on the seedlings, it is suggested that in such cases ammonia accumulates in the sap and renders the iron inactive. The chlorosis is accompanied by withering of the foliage and either stunting or abnormal elongation of the roots. The first few leaves formed do not show symptoms, possibly because they obtain enough iron from the seed ; these leaves always contain more iron than those formed later. As the plants reach maturity the typical symptoms disappear.

Manganese-induced Chlorosis

Turning to manganese-induced chlorosis, the pineapple disease known as ' yellows ' is one of the best-known examples. It occurs chiefly in the extensive Hawaiian plantations in highly manganiferous, acid soils, deficient in calcium carbonate. Affected plants turn yellowish-white, growth ceases, and the leaves die back until the whole plant is dead. The disease can be cured by spraying with ferrous sulphate, in the same way as the lime-induced chlorosis of the same plant in Porto Rica can be treated. In spite of the difference in soil conditions under which the two diseases occur, their proximate cause appears to be similar, namely, a lack of available iron in the plant ; manganese and calcium carbonate can each exert an additive chlorotic effect in the presence of the other.

Several of the cereals are susceptible to manganese-induced chlorosis, as are also sugar-cane and tobacco, but many other crop plants are practically unaffected by an excess of manganese.

Causes of Iron Deficiency Symptoms in Plants

In both manganiferous and calcareous soils, plants susceptible to chlorosis appear to be unable to obtain the iron that they require in a suitable form. Various theories have been advanced to account for this, but the real cause seems to be obscure. Sometimes there may be an absolute deficiency of iron, but cases occur commonly where there is no real shortage of the element. Since in many of these cases analysis does not reveal a lack of total iron in the chlorotic plants, but rather its immobility or restriction to certain parts, the physiological conditions within the plant are evidently of importance, and this appears to be particularly the case in the manganese-induced form. It has also been suggested that manganese interferes with the rôle of iron in the formation of chlorophyll.

BORON

Boron deficiency has received more notice during recent years than that of any of the other trace elements, partly because it has accounted for several major

plant diseases that had long puzzled plant pathologists, but also in part, no doubt, because of the excellent propaganda employed by commercial interests.

Though earlier French work had shown that boron could act as a catalytic fertiliser and that it was an essential element in the development of maize, the first description of symptoms of nutritional diseases due to lack of boron was issued from Rothamsted in 1923. It was not until 1931, however, that it was established by work in Holland that a major plant disease, the well-known heart and dry rot of sugar and fodder beet, which seems to have been seen in France over seventy-five years ago, was due to boron deficiency. Since then several other important plant diseases have been traced to a similar cause, and there is no reason to suppose that the list is as yet complete. Unlike most other deficiencies, that of boron affects the storage organs of the plant, roots, tubers, fruits, and the like, usually more than the green parts, though these seldom escape altogether and in some plants show marked symptoms (9, 10, 11, 12, 13).

The heart and dry rot of beets was long known to be associated with alkaline soil conditions, and to be promoted by liming, especially in dry alkaline soils. At the same time the almost constant occurrence of *Phoma betae* in the diseased parts led to many attempts to implicate this fungus in the causation of heart rot. It is now known that these are merely accessory factors and that typical cases of the diseases may occur in their absence if boron is not available to the plant. Soil applications of boron have been exceedingly effective against the disease, whether applied alone as borax or other compounds, or used to reinforce the ordinary fertiliser mixture for the crop. In Germany it is stated that 30,000 tons of sugar-beet fertiliser in the form of borated superphosphate were used in 1938. From 10 to 20 lb. borax per acre may be required. Heart rot is further described in Part II.

In a form of the disease occurring on vegetable beets and known as ' girdle ' in the United States, dark sunken spots may partially encircle the beets below soil level; though borax treatment gives good results against this disease it is not certain that its true cause has been established. Another beet disease successfully treated by borax is the internal black spot of canning beets in the United States, a disease characterised by the presence between the rings in the thin-walled tissue layers of the bulb of irregular hard or corky black spots, usually accompanied by distortion and reddening of the foliage.

Following at short intervals in 1935 and 1936 it was reported in New Zealand, Canada, the United States, and Finland that the serious disease of apples known as internal cork, corky core, and drought spot was due to boron deficiency. The disease had been known for a good many years in these and other countries, and its association with soil conditions, especially soil moisture and unbalanced nutrition, had been stressed.

The fruits on affected trees are characterised by dead, dry brown spots scattered through the flesh or confined to the core. In the form termed drought spot, the spots or brown streaks following the vascular bundles start just under the skin, which in some varieties shows a corresponding roughness. Affected trees may show a tendency to increased fruit drop, and in extreme boron deficiency die-back of the terminal branches may be observed. Some varieties are more liable to the drought spot type of disease than to internal cork, but others have both,

and late in the season the latter type is predominant. The condition is aggravated by drought and is worst on light or shallow soils. There is an abnormal meristematic activity in the internal spots, resulting in the development of a cork cambium around the lesion and the formation of linear rows of cells; individual cells may also divide. Starch is retained in the necrotic areas.

Soil applications, spraying, and injections have all been used in controlling this disease by boron compounds. In soil applications in Canada, 30 lb. boric acid per acre applied in the autumn around the affected trees, two or three feet from the base, has given good results. In view of the heavy loss that may occur, only one-tenth of the boron applied being sometimes available after a year, smaller annual applications may prove to be the most satisfactory. Heavy applications have also been found to impair the keeping qualities of Jonathan apples in New Zealand. The beneficial effects of soil applications have been found to last for three or four years in Canada and the United States. Since treatment with boron compounds has been adopted, the disease has ceased to cause concern in British Columbia, where it was formerly a serious trouble.

Experiments in the United States indicate that the obscure apple disease known there and in Australia as 'measles' or 'internal bark necrosis' may be another manifestation of boron deficiency. The disease, which has been known since 1908, is characterised by an eruption of small pimples in localised areas of the bark. These become isolated by cork and exfoliated, cankers then developing in severe cases. Isolated necrotic nodules occur deep in the bark. In cultures deprived of boron both the eruptive and buried necrotic lesions that resulted were macroscopically and microscopically indistinguishable from those of natural cases of measles. Their development was prevented by application of boron. The disease has been reported to be prevalent on compact clay soils, in which it is recommended not to plant the highly susceptible Delicious variety. There are marked differences in varietal susceptibility to it.

Pears and cherries sometimes suffer from drought spot similar to that of the apple, and a condition in pears marked by cracking of the calyx-end of the fruit and die-back has been readily cured by soil dressings of 1 lb. borax per tree, in Australia. In apricots a brown spotting of the flesh, especially near the stem end, and a dry spongy condition near the stone, was controlled by borax in New Zealand. In Canada apricots under boron-deficient conditions suffer from drought spot and die-back. Plums are liable to drought spot and gum spot from the same cause, the fruit showing dead pulpy areas and the flesh a tendency to stick to the stone; in this case also the trees may suffer from die-back. To complete the catalogue of the tree-fruit diseases hitherto attributed to boron deficiency, additions to which are still being made, the report that boron was essential for the healthy growth of citrus in Southern Rhodesia, and that the disease known there as 'hard fruit' could be controlled by soil applications of borax, should be mentioned. Trees showing this condition have small leaves, the branches tend to die back, and fruit drop is accentuated. Such fruits as are left are dry, hard, deformed, their development may be arrested, and gum pockets or corky patches are found in the fruit pulp. A great increase in marketable fruits was obtained by applications of 50 to 500 gm. borax per tree.

Several lists of vegetable and other crops affected by boron deficiency have been published, together with, in some cases, the recommended doses of borax which have been found safe and effective [5, 11, 20]. These doses vary with soil and crop conditions but are generally between 10 and 20 lbs. per acre [8]. Heavy applications may cause damage, especially to a succeeding crop that may be sensitive to the toxic action of boron, such as beans. In the eastern United States and Canada boron deficiency is sometimes responsible for a serious cauliflower disease, termed browning, internal browning, or brown rot. Small brown concentric, water-soaked areas develop in the stem and in the branches of the curd (head) and rusty-brown patches may show on the surface of the curd. The affected heads have a bitter taste. In cultures under conditions of extreme boron deficiency the leaves around the head are deformed, the development of the curd is arrested, and browning spreads from the top into the flesh and stalk. Spinach is similarly liable to suffer from boron deficiency, the plants turning yellow and the leaves becoming small and deformed when deprived of boron. In celery a disease known in Florida since 1924 under the name ' cracked stem ', which has since become prevalent in the United States and Canada where it may destroy half the crop in patches, was reported in 1937 to be due to boron deficiency and to be preventable by soil applications of borax or by spraying the plants with a solution of borax. It is characterised by a transverse cracking of the epidermis above the vascular bundles, the broken tissues curling back and turning brown. At an earlier stage a brownish mottling of the leaf may be observed. The stalks are brittle and the roots brown, with loss of many lateral roots.

Sunflowers have been used as indicators of boron deficiency in the western United States. Their leaves show marked abnormalities, and apical growth is arrested, when they are deprived of boron, while their susceptibility to the powdery mildew, *Erysiphe cichoracearum* is greatly increased. In this area, the soils of which are notably deficient in boron below 3 feet, in certain geological formations, humus depletion accentuates the tendency to boron deficiency which may be lessened by the use of compost.

Boron was found to be necessary for potatoes by experimental work some years ago. Since then there have been several reports of potato diseases due to lack of this element. In Scotland a non-parasitic leaf roll found in certain soils and in certain varieties, especially in dry seasons, was prevented by soil applications of borax and an increased yield of 35 per cent. obtained. Indications were also given that the tuber diseases known as ' internal rust spot ' and ' spraing ' might be due to the same cause. In Holland a tuber disease in boron-deficient soil was greatly reduced by fertiliser applications containing boron. Affected tubers show a total or partial browning of the vascular ring, usually most pronounced at the stolon-end of the tuber. This browning sometimes involves the neighbouring cortex and occasionally the pith. The diseased tissues rapidly darken on exposure to air.

It was reported in 1930 that the tobacco disease in Sumatra known as ' top disease ' could be largely controlled by a boric acid solution applied to the plants. Subsequent experiments in Holland in 1934 showed that tobacco is very sensitive to boron deficiency, which caused weakness of the root system, death

of the growing point and axillary buds, chlorosis, thickening and wrinkling of the leaves, often with a downward bending of the main veins, and discoloration of the vascular system. The similarity of this condition to the Sumatra tobacco disease was pointed out. In the United States and Germany also, tobacco disorders of a similar origin and effect have been described, the affected plants showing chlorosis of the bud leaves with collapse of the tissues and contorted or unilateral growth of the top of the plant, followed by die-back of the terminal bud and of any later-formed lateral buds. Top disease is usually most severe on alkaline soils in Sumatra and under such conditions the best control has been given by a solution of 6 mg. boric acid in half a litre of water per plant, poured into the hole during transplanting or applied around the seedling after setting out. In Germany borax is reported to be regularly applied for tobacco at the rate of 20 kg. per hectare.

Various types of yellowing, reddening, and bronzing of the foliage are sometimes the principal symptoms of boron deficiency in herbaceous plants. The lucerne disease known as ' yellow top ' in the United States and Canada has been traced to this cause. The yellowing occurs between the veins in diffused patches, or in streaks if the leaves are affected when mature. The leaf margin may die and curl up. In severe cases the growing point is stunted and bears small yellow leaves. Many of the leaves turn bronze or reddish, but usually the main veins remain green. The internodes are shortened and the plant stunted. Good control has been obtained with soil applications of boron compound ; with borax applied in the previous August an increased yield of 16 per cent. was obtained at the second cutting. A somewhat similar condition has been observed in clovers in the United States, and a lettuce crop showing symptoms resembling those due to manganese deficiency was cured in calcareous soil in North Carolina by a soil application of only 4 lb. borax per acre. In this soil 5 lb. borax per acre applied in March effectively controlled ' yellow top ' in lucerne. It is of interest to note that experiments in Holland showed that lucerne and peas were particularly sensitive to lack of boron, but cereals suffered little. Carrots grown in culture solutions without boron at Rothamsted had reddened or yellowed, down-curling leaves, followed by death of the growing point ; there was a small tap root, and the laterals were short and locally thickened from failure of the rootlets to emerge. All the elements of the vascular tissue in the root were affected, beginning with hypertrophy and collapse of the parenchyma cells, enlargement and irregularity of the cambial and phloem elements, and blocking of the cavity in many xylem vessels. Hypertrophy and breakdown also occurred in the ground cells of the cortex but, unlike what happens in the turnip, the central tissues rarely showed lesions. These internal changes generally start near the growing apex of the plant.

Causes of Boron Deficiency Symptoms in Plants

No entirely satisfactory chemical explanation of the frequency of heart rot and other boron deficiency disorders in alkaline soils seems to have been given. In some soils there is absolute deficiency of boron, in others boron may be present in appreciable quantity but in forms of very low availability to plants. In general,

high exchangeable calcium, which is associated with high *p*H values or alkalinity, reduces the availability of soil boron. A microbiological explanation has been sought, especially by Russian workers. It has been supposed that the available boron is used up by the soil microflora, the activity of which is increased by liming, or that liming increases the reduction processes in the soil by causing a rapid increase in the development of anaerobic bacteria, or even that liming induces parasitism in soil bacteria which are not harmful and may be beneficial when boron is in adequate supply. It is of interest in this connection to note that work at Rothamsted has shown that in the absence of boron the normally symbiotic nodule bacteria of the *Leguminosae* may become parasitic owing to a disturbance in the transport of carbohydrates to the nodules (see also p. 755).

It has proved possible to correlate the occurrence of 'raan' in south-west Scotland with the geological origin of the soils. In general, the boundaries between high and low incidence of the disease correspond to the division between boulder clays of southern and northern origin, the latter being rich in tourmaline, a mineral containing a considerable amount of boric oxide, while the southern boulder clays have little. This is a good instance of the type of absolute boron deficiency. 'Raan' of swedes and turnips is further described in Part II (p. 571).

MANGANESE

Manganese deficiency is widespread and is particularly important because it injures such extensively cultivated crops as cereals, potatoes, and sugar-beet.

Though earlier work had shown the value of manganese as a fertiliser, the first suggestion that a definite crop disease, the 'grey speck' of oats, might be due to its lack was made in Germany in 1914, and it was not until between 1922 and 1928 that it was established by work in the United States that manganese is essential for the growth of various plants. In Australia a little later, manganese was found to be necessary for all the 15 species of plants tested, which ceased growth, often became chlorotic, and then suffered a necrosis of the growing points, in its absence.

Grey speck disease of oats was long known in Central and Northern Europe, especially on limed acid soils, rich in humus, such as reclaimed moorland. The use of manganese for its control in these areas gradually became established from 1923 onwards. The 'Veenkoloniale' disease also affected other crops, rye, barley, wheat, potatoes, beets of all kinds, turnips, etc., though only a few of them showed spotting, the others having yellowed or chlorotic foliage; this condition was also successfully controlled by manganese. Subsequent observations in Denmark have extended the list to include spinach, tomatoes, and various pasture grasses, and in Sweden flax has been added. In the United States, especially, it was found that chlorosis due to lack of available manganese was not confined to crops grown on reclaimed heaths, but occurred on other characteristic soil types; chlorosis of spinach, lettuce, beet, onions, etc., in certain parts of Rhode Island, of market garden and other crops in acid sandy loams in the coastal plain of North Carolina, and of sugar-cane in Hawaii (where 'Pahala blight' of this crop was controlled by

manganous sulphate), were recognised as manganese deficiency diseases. In 1928 a very complete study of grey speck as a manganese-deficiency disease was published in Australia, where cereal crops on alkaline volcanic or reclaimed swamp soils were chiefly affected and it was established that the availability of the soil manganese was dependent on complex soil factors. In the British Isles oats and many other crops are affected in soils agreeing in their high content of organic matter and lime, but of varying reaction above *p*H 6·5. All the affected crops may recover as they grow older, provided they are not too severely injured by the early attack [27]. Grey speck is further described in Part II (p. 420).

Amongst other crops affected by lack of manganese in England are globe beetroot and spinach, which are extremely sensitive to this condition, potatoes, onions, marrows, cos-lettuce, parsnips, and beans. In the United States the symptoms in spinach resemble chlorosis ; in England they are described as being similar to ' speckled yellows ', except that the spots are greyish-yellow rather than yellow. Onions grown on unproductive peat soils in New York State have yellowish leaves which die off at the tips. Many of the plants die before harvest, but an application of 100 lb. manganese sulphate per acre has been found to check the disease. In potatoes the younger leaves show dark-brown to black, round spots along the sides of the veins, especially in the lower half of the leaflets. Later in growth, the whole leaflet becomes light in colour. The spots are sometimes confused with those associated with potash deficiency, but the latter occur principally on the older leaves in irregular areas, especially at the edges. In a very severe type of manganese deficiency known in beans in Florida in humus-rich soils to which calcium has been added, the leaves first turn pale and mottled from retention of the green colour for a longer period, near the veins, then golden yellow. Growth is retarded and the affected leaves progressively become smaller. Stipple spotting develops before the yellowing is complete, the small necrotic brown spots appearing parallel to the veins. Later the under surface becomes cupped between the veins and the interveinal tissue then breaks down. Eventually the buds die, secondary growth lower down also dies, and the plants wither. Almost fantastic increases in the yield of beans (more than 25-fold) were obtained in this area by manganese sulphate applied at the rate of 50 lb. per acre, and still better results were given when to this application an equal quantity of sulphur was added. Many other plants have been reported to suffer more or less severely from manganese deficiency in Florida ; in highly calcareous soil containing a small admixture of peat and only a trace of manganese, tomatoes show a pronounced yellowing of the leaf areas farthest from the veins, pin-point necrotic spots appear and expand into dead patches, growth becomes increasingly spindly, little blossom develops, and there is no fruit. Histologically, manganese-deficient tomatoes have thinner stems and leaves, with less xylem, and smaller palisade cells than normal. There is a good deal of plugging of the vessels, and crystals are abundant [16].

For several crops, better results have followed the application of a small amount of manganous sulphate as a spray than the use of far larger quantities in the soil.

The obscure pea disease known in England as ' marsh spot ', in Holland as ' rotten heart ' (' kwaade-hart '), in France as ' moucheté ', and in Finland as

'internal necrosis' was first suggested in 1936 to have some relation to manganese deficiency. Marsh spot is further described in Part II (p. 625).

That fruit trees may show marked symptoms of injury from manganese deficiency was established by culture work on citrus in California from 1936 onward. Oranges and lemons in New Zealand were reported in 1938 to suffer from a disease resembling the 'mottle leaf' disease due to zinc deficiency (see below, p. 309), but treatment with zinc compounds failed to effect an improvement while very promising results have been obtained with manganese sulphate [2]. Fuller accounts of the symptoms and control of manganese deficiency in citrus groves in Florida and California followed. A few cases of chlorosis in apples attributed to manganese deficiency were reported from Canada in 1937. Both apples and raspberries are affected in England, and evidence is accumulating that deciduous fruit trees in South Africa may be severely injured by a shortage of manganese. In the Western Cape Province of South Africa manganese deficiency appears to be about as prevalent as that of iron or zinc, the symptoms being of the 'mottle leaf' type and affecting chiefly the stone fruit.

Citrus trees suffering from lack of manganese show a network of fine green veins on a light-green background in the younger leaves, where the symptoms are difficult to distinguish from those due to zinc deficiency. As the leaves expand fully, the pattern of dark green near the veins and light green to bronze, fading to dull whiteness, between them, is less sharply defined than that of true mottle leaf; nor are the leaves as narrow and the internodes as short as in that disorder. In severe cases, growth and foliage are reduced and the fruits are light in colour. Spraying in spring and summer with manganese sulphate solution (50 to 100 gm. per gallon) has given good control but causes some injury to the leaves; this may be reduced by the addition of hydrated lime or sodium carbonate to the spray. Soil applications in winter of 65 per cent. manganese sulphate at the rate of 5 lb. per tree have also been recommended, but all treatments are still somewhat experimental.

Peaches suffering from manganese shortage in South Africa have similar leaf symptoms to those already described, but the interveinal areas may turn yellow. Even in trees having mild leaf symptoms there may be a substantial diminution in the yield of fruit, but this can be fully restored by winter spraying with manganese hydroxide or injections of manganese into the wood, the cost of the latter treatment being only 2d. per tree in an orchard which bore no commercial crop before treatment, and one worth from £2 to £3 per tree afterwards. A similar case of loss of crop following chlorosis has been observed in cherries in England, where branch injections of both iron and manganese were effective in securing a healthy new growth, the response to manganese being the better of the two.

Tung trees (*Aleurites fordii* and *A. montana*) are adversely affected from lack of manganese in Florida, not only on calcareous or over-limed soils but on other soils in which exchangeable manganese is scanty; and a wide range of ornamental shrubs in acid, sandy, as well as in over-limed or calcareous soils in the same State show similar symptoms, and respond well to manganese treatment. In most of these plants the leaves are mottled from retention of the green colour longer near

the veins than farther away, and there are necrotic spots in the yellowed or chlorotic areas. In some, the spots may be larger, and deep reddish or purple anthocyanin-like blotches occur.

Causes of Manganese Deficiency Symptoms in Plants

It would seem that disease due to manganese deficiency, like that due to boron deficiency, sometimes results from an actual scarcity of the element in the soil within the root range of the plant, and sometimes from the manganese being present in a form which the plant cannot utilise. In this last type of deficiency, which is the common form in England, there is a certain similarity in the soil conditions under which it occurs and those under which deficiencies of iron and zinc occur, and there have been several reports of deficiencies of two of these elements occurring simultaneously. Grey speck has been found to be accentuated by large quantities of lime, humus or colloids, by the application of nitrates or alkaline phosphatic fertilisers, and by drought. Acidification of the soil with sulphur is effective in the prevention of grey speck and allied diseases where the soil is not actually deficient in manganese, and is recommended in England in preference to manganese applications, in many such cases. Presumably local acidification, water-logging, and reducing materials increase the availability, by reducing manganese dioxide. The effects of over-liming appear to depend in part on the fact that the oxidation of manganous salts proceeds more rapidly and completely in alkaline solutions. Where the original reserve of total manganese is relatively high a considerable time may elapse before injury from liming is visible, but if reserves are low the symptoms soon develop.

The liming of acid fen soils and certain other types of soil in England carries with it the risk of inducing manganese deficiency. So, also, the ploughing-up of grass-land on calcareous formations where the turned-over sod provides a humus-rich layer in association with excess of calcium has been followed by severe outbreaks of manganese-deficiency diseases.

It has been suggested that the effects of an absolute manganese deficiency are aggravated by certain bacteria or moulds in the roots. The occurrence in affected soils of bacteria capable of precipitating soluble manganese compounds into insoluble oxides is also believed by some to play an important part in the etiology of the disease [19].

ZINC

It may be that little would yet be known of the injurious effects of a shortage of zinc upon the health of certain plants were it not for fortuitous observations reported in 1932, first on the treatment of ' rosette ' or ' little leaf ' of apple and other fruit trees in California by soil applications of ferrous sulphate, and a little later on the effect on ' pecan rosette ' in Louisiana of injecting ferric salts into the wood or spraying the green parts with iron sulphate. These showed that the beneficial results obtained were due to small amounts of zinc contained in the iron salts.

The clue was immediately followed up and control of the diseases secured by the use of zinc compounds. Tests were at once made of the effect of zinc on the well-known ' mottle leaf ' disease of citrus trees in California, since some of the symptoms of this obscure disorder resemble those of ' little leaf ' in deciduous fruits, and success in the treatment of mottle leaf in oranges and lemons by soil applications of zinc sulphate was reported in 1933.

Mottle leaf or ' frenching ' of citrus has been known in the United States since a very early date. Apple rosette was described as a specific and prevalent disorder in the inland regions of Washington State in 1923. Pecan rosette was first recognised about 1900 and intensive studies on it were carried out for many years with disappointing results. Little leaf or ' Californian yellows ' of the stone fruits, peach, apricot, and plum, was also well known in California for many years. In a study of ' walnut yellows ' or ' little leaf ' published in 1928 it was stated that this disease, the little-leaf disease of stone fruits, pecan rosette, and mottle leaf of citrus formed a group of physiological tree diseases in California probably all due to the same cause and correlated in some way with the base relationship in the soil. Little-leaf or mottle diseases of apple, grape, avocado-pear, fig, and cherry in California and Washington were later included.

Mottle leaf in citrus trees has, as its main symptoms, a wrinkling and pale yellowish-green mottling of the leaf, contrasting sharply with green bands along the main veins, followed by desiccation and leaf drop. On severely affected trees the leaves are small, pointed, and narrow, and the fruit is small, hard, and dry. Bud development and tissue differentiation at the growing point are delayed. In the pale leaf areas the cytoplasm of the cells is reduced and the few undeveloped chloroplasts tend to collect at one pole of the cell. Photosynthesis is feeble in the palisade cells, which are often divided transversely, but starch formed elsewhere tends to accumulate in the spongy parenchyma which seems to be incapable of utilising it. Leaves on the south side of the tree are usually more mottled than those on the north, and culture experiments showed that the mottling varied directly with the light intensity. After treatment with zinc, normal chloroplasts are produced, and there is a striking improvement in growth; the xylem cylinder of twigs 14 months old on the treated trees in one set of experiments averaged 518·5 μ in diameter against 381·3 μ on untreated twigs of the same age.

The best and safest method of applying zinc compounds to citrus trees appears to be by spraying in concentrations equivalent to about 1·15 lb. zinc to 100 gallons water. Zinc sulphate or zinc oxide may be used, and it is advisable to add hydrated lime or soda ash to the former or to combine the latter with lime-sulphur. Dusts have not given complete control and soil applications have caused injury to the trees and, in California at least, have proved expensive, since most of the soils have so high a fixing power for zinc that heavy applications are necessary. In the Southern Hemisphere striking improvement in mottled orange trees resulted from the application in mid-September of a spray consisting of 10 lb. zinc sulphate (23 to 25 per cent. zinc), 5 lb. hydrated lime, with a little spreader, in 100 gallons water. A similar treatment at a corresponding period (March) in California gave an increase in yield of oranges of about 6·5 per cent. in the same season and of 24 per cent. the following year, while in grape-fruit the same treatment at the

beginning of blossoming is reported to have increased the total yield in bad cases by several hundred per cent.

Mottle leaf of citrus, also sometimes termed 'foliocellosis', is known in almost all citrus-growing countries. In Western Australia observations suggest that the incidence of citrus anthracnose (*Colletotrichum gloeosporioides*) is correlated with that of mottle leaf.

Apple rosette is marked in advanced cases by the dense cluster of small, narrow yellowish leaves at the tips of the twigs, the rest of which is bare. The new growth is spindling, with narrow crotch angles; the internodes at the tips are much shortened and there may be a production of short lateral shoots lower down. Die-back of these and of the rosetted twigs may follow in a few months. In the earlier stages the chief symptom is a yellowish-green mottling of the foliage. Pears may be similarly affected, and indeed no clear distinction can be drawn between the symptoms formerly classified under 'little leaf disease' in stone fruits (peach, apricot, plum, and cherry) and 'rosette' of pome fruits. In the Western Cape Province of South Africa, apple, pear, apricot, and plum all show similar symptoms, the disease being far more destructive than the equally prevalent manganese-deficiency disorder in the same area [15].

Cytologically and histologically these diseases are characterised by inhibition of development or by destruction of the chloroplasts, sometimes with a lack of differentiation of the tissues in the affected parts of the leaf, especially those most intensely illuminated. Affected cells may have very scanty contents. In the apical meristem, cell multiplication is inhibited, and there is premature vacuolisation and polarisation, with an accumulation of phenolic materials in the vacuoles. In the apricot and peach, gums were present in senescent leaves while tannin was increased in the dwarfed leaves. It is suggested that the oxidation processes in the cells are impeded by zinc deficiency.

Treatment of rosette and little leaf is on the same lines as that of mottle leaf. Old, affected stone fruits may require spraying with higher concentrations (50 lb. zinc sulphate per 100 gallons if severely affected, 25 to 30 lb. if less so), applied just before the buds swell. Young trees can be given the same treatment during dormancy or the foliage may be sprayed 4 to 6 weeks after it appears with 18 to 25 lb. zinc sulphate, plus one-third as much hydrated lime, in 100 gallons water. In Florida good results have followed zinc sulphate-lime sprays on peaches, using a 5–2½–100 formula [14]. Stronger solutions, up to 10–15–100 may be required to correct severe cases in avocados [22]. Treatment by injection into the wood round the base of the trunk or by driving zinc tacks into the wood, as is sometimes done with iron nails in iron-deficiency chlorosis, has also given good results on some fruit trees, lasting throughout several seasons. Dust and soil applications are less satisfactory. In Queensland good results against little leaf in apples in the first season were only obtained from dormant spraying, with 50 lb. zinc sulphate per 100 gallons water, none of the other treatments giving a positive result quickly enough to affect the growth in the same season. In South Africa also the best results on pears were given by dormant applications. Good controls of little leaf in grape vines in California was obtained by swabbing with a solution of 2 lb. zinc sulphate in 1 gallon water immediately after pruning, as is done with iron

sulphate against the vine chlorosis due to iron deficiency; this treatment was less effective in fruit trees.

Little leaf or rosette has been reported in deciduous fruit trees from Australia, New Zealand, South Africa, Southern Rhodesia, the Argentine, and Hungary, in addition to the United States.

Pecan rosette was one of the first of this group of disorders to be described. As in apple rosette, its chief symptom is the development of small, crinkled, brittle, yellowish-mottled leaves with short petioles, clustered at the ends of the branches. In slight cases, or early stages, yellow mottling with prominent veins on the young leaves at the tip of the shoots may be the only symptom. In advanced cases there is considerable reduction in growth, the leaves are deformed, and the affected branches die back during the late part of the growing season, the same fate overtaking the secondary growth which is put out from dormant buds lower down. The nuts on affected branches are small and misshapen. At first only a part of the tree may suffer. Different varieties show rather different symptoms, some having more leaf necrosis and die-back, others more chlorosis and malformation of leaves and fruit.

The histological changes in affected pecan leaves vary, but may include hypoplasy and reduction in differentiation of the tissues and smaller intercellular spaces. In the centre of the yellowish areas, chloroplasts are almost absent and the translocation of starch from affected leaves seems to be inhibited.

Walnut ' yellows ' in general resembles pecan rosette, but there is a downward curving of the petioles of the leaves and the leaflets are similarly curved.

Both these nut-tree diseases, reported from the United States, are now satisfactorily kept under control by treatment with zinc compounds, though pecan rosette has in the past led to the abandonment of hundreds of acres. Pecan rosette is also known in Australia.

The disorder of tung trees (*Aleurites fordii* and *A. montana*) known as ' bronzing ' in Florida was regarded as of minor importance in the early days of the cultivation of these trees, but became increasingly prevalent and severe as time elapsed. The first visible symptom is a bronzing of a few or many leaves, coupled with a deformation of the terminal ones on the twigs. The leaves become progressively smaller and more malformed and may be crowded into rosettes. The colour deepens to dark bronze and parts of the leaf blades die, leaving a ragged appearance. Control by zinc sulphate has been effective, whether applied to the soil or used as a spray. Besides Florida and Louisiana, ' bronzing ' of tung trees has also been reported from South Africa. A few cases of disease symptoms ascribed to zinc deficiency have been reported in forest and shade trees, both conifers and broad-leaved trees.

In herbaceous plants records of zinc deficiency, so far, are scanty, but pot-culture work and some field studies indicate that more are to be expected. Thus, though the effects of zinc deficiency have not been observed in potatoes in the field in Holland, characteristic symptoms have been produced in cultures. In Queensland, a pineapple disease known as ' crookneck ' responded favourably to treatment with both copper and zinc, but the former when used alone only delayed the symptoms, whereas when zinc was added to the copper they were

prevented. In South Australia, many light soils that are deficient in copper show a less important deficiency in zinc, the crops affected including oats (3). Symptoms of zinc deficiency are stated to be visible in beans, cowpeas, millet, and other plants in Florida, and the serious disease of maize known as ' white bud ' in the same State has been cured by applying 10 to 20 lb. zinc sulphate (89 per cent.) per acre. White bud has been experimentally induced in maize by growing the plants in soil from a peach orchard severely affected with little leaf and was prevented when zinc sulphate was added to the soil. It is marked by a white to very pale-yellow coloration of the unfolding buds of seedlings and a chlorotic streaking and spotting of the older leaves.

Causes of Zinc Deficiency Symptoms in Plants

Soils usually contain very little zinc but what little there is appears to be essential for the proper development of plants, though plants seem to vary widely in their requirements for this element. As in so many of the other essential trace elements the amount available to the plant is more important than the total soil content. Soluble zinc is readily leached from open sandy soils but becomes partly fixed in clay soils or those with a high humus or marl content. Phosphates increase the fixation of zinc.

There is some evidence that soil micro-organisms may be responsible for rendering zinc unavailable to plants ; bacteria have even been isolated from affected soils which, when added to a complete culture solution containing sufficient zinc to give a healthy growth (0·023 p.p.m.), caused white-bud symptoms to appear in maize. Increasing the zinc to 0·046 p.p.m. or injecting zinc sulphate into the stem prevented the symptoms.

COPPER

Among the group of crop disorders which became increasingly prevalent in Germany, Scandinavia, and Holland owing to the active measures taken during modern times to reclaim peat moor, swamp, and polder soils, that which eventually became widely termed ' reclamation disease ' is one of the most important. Knowledge of these diseases is largely due to the fine work of soil scientists and biologists in Holland. In that country, though the diseases themselves had been recognised earlier, it was first clearly established between 1923 and 1925 that there was a sharp distinction between the three main physiological diseases of crops cultivated on alkaline or acid-reclaimed or poor soils, namely, the Veenkoloniale disease (grey speck), controllable by manganese, the Hooghalen or ' soil acidity ' disease, now known to be associated with deficiency of magnesium, and the black-peat disease, or Ontginningsziekte (Urbarmachungskrankheit, or ' reclamation disease ' of Germany). A similar conclusion as to the distinctive character of the three diseases was reached in Germany in 1925.

In Germany reclamation disease has been long known in oats under the name Weiss-seuche ; it is found in the north mostly on podsolised acid dry peats over-

lying sand, with ling (*Calluna vulgaris*) as the dominant plant. Low-lying swampy bogs or marshes, however, are not immune, nor is there any close correlation with the peat-forming plant association. In Holland the 'black peat disease', as it was at first termed, was described in 1923, when it was stated to be confined to peat soils reclaimed from moors and heaths; these peats were mostly black and amorphous, sometimes, with up to 30 per cent. humus, and, as in Germany, both dry sandy moors and marshy bogs or swamps were eventually found to be affected. In Denmark the disease was at first restricted to reclaimed *Calluna* heaths in Jutland. Both in northern Sweden and in Poland extensive reclamation schemes undertaken about 1924 showed early signs of similar trouble, not only in the cereals but also in the meadow and pasture grasses that were established on the reclaimed peat; these included timothy (*Phleum pratense*) in both areas, and *Poa trivialis* and *Agrostis stolonifera* in Sweden; Norway, France, and Switzerland have also reported the disease, and it is hardly likely that it is absent from Great Britain.

By 1937 it was estimated that the soils liable to reclamation disease covered 50,000 hectares in north-western and eastern Germany, the provinces affected being mainly Schleswig-Holstein, Hanover, and Oldenberg but lesser areas being found in the Lower Rhineland, Westphalia, Mecklenburg, and elsewhere. As soils in north Germany, known to have been under cultivation for 80 years, were found clearly affected and similar trouble was reported on land cultivated for 300 years, while in Jutland the cases were seen in land reclaimed 20 to 50 years earlier, the term 'reclamation disease' does not imply that the trouble is only to be expected on newly reclaimed land.

Wheat, barley, and oats are all susceptible, but reclamation disease is seen most frequently on oats as the cereal most commonly grown on these peat soils. The moderately susceptible crops include swedes, turnips and other cruciferous crops, carrots, field peas, broad beans (*Vicia faba*), certain grasses, lucerne, and red clover (*Trifolium pratense*). Potatoes, mangolds, some pasture grasses, and white clover (*T. repens*) are resistant, while rye is the least liable of the cereals, winter rye usually showing no symptoms until the spring, while spring-sown rye may escape altogether.

In cereals the chief symptoms of reclamation disease is a whitish or yellow discoloration of the tips of the leaves. In oats the tips and edges of the leaf tend to be whitish and papery, and affected fields show a pale shimmer from which the old German name of Weiss-seuche was derived. Unlike grey speck, the symptoms of which are most evident in the juvenile stage of the crop, reclamation disease reaches its height as the plants mature. No symptoms may be visible before 4 to 6 weeks or the early leaves may show a greyish-white to reddish-brown colour and dry up prematurely. In barley the discoloration of the leaf tips is more yellow, hence the names 'Gulspidssygen' (Denmark) and 'Gulspetssjuka' (Sweden), both meaning 'yellow tip disease'. Wheat and rye resemble oats, the leaves of wheat showing pronounced elongated white strips. In all the cereals turgor is reduced and there is an increase in the straw-to-grain ratio. In oats the glumes are loose, whitish or brown, under-developed and unravelled. In severe cases panicles are not formed: if formed they carry few and light grains. A notable feature of affected oats is the excessive tillering due to the production of

fresh green shoots from lateral buds; these may continue to form even in the stubble, but they come to nothing. In mild cases affected cereals may have few leaf symptoms but the ears tend to be 'deaf' and the grain-to-straw ratio is reduced.

Red clover, which is severely damaged, shows pallor of the leaves sometimes accompanied by a dry, brown spotting, spreading inwards from the margins, with abnormally large basal leaves and loose, scanty, flimsy heads, developed later than usual. Swedes and turnips are markedly stunted, with large yellowish-white patches spreading inwards from the leaf margins. In peas the symptoms are somewhat similar, the basal leaves first showing a yellowish discoloration, after which those at the tip wilt and shrivel. Broad beans have less marked symptoms, the onset of which is more gradual than in peas. In timothy grass the leaves dry up before flowering occurs; permanent pasture grasses, however, seem to be slower than annual crops to show the ill-effects of reclamation disease, presumably because their more gradual development fits them better to use small amounts of necessary nutrient elements, though a tendency to the gradual dying-out of the more desirable pasture grasses in certain reclaimed soils has been attributed to this disease.

In the low-lying raw peat soils of the Florida Everglades, as well as in marsh soils in Russia, failure of various crops from causes that seem to be identical with those inducing reclamation disease have been reported. In the Florida soils it proved impossible to grow a wide variety of crops until the copper treatment was introduced.

Another large area in which cereal and other crops suffer from a disease due apparently to the same cause as reclamation disease in Europe occurs in South and Western Australia. It was described in 1938 as affecting wheat, barley, and oats but not rye, on alkaline, calcareous, blown-sandy soils, in South Australia. The leaves of the affected plants droop, there is much withering, curling, and dying off of the tips, and if heading occurs at all the ears fail to mature. Improved pasture grasses that were under test on these soils were also much injured, and trials with lettuce, turnips, and broad beans gave a poor stand and much reduced yields. In Western Australia a very severe form of the disease was reported in 1940, on marly peat soils, acid swamp soils, and sandy brown soils. In these areas it is not uncommon to find cereal cultivation quite impossible without treatment, and tests showed that the yield of potatoes and various truck crops was also reduced. In certain of the marly soils manganese is also deficient, as is seen especially with potatoes [24].

At first, the control measures recommended against reclamation disease included the application of clay which, at the rate of 100 cubic metres per hectare, gave good results and was strongly advocated in Sweden, and heavy dressings of urban refuse or compost, with or without sewage, which were effective in Holland. In 1925, however, it was reported in Holland that great benefit followed the application to affected peat soils of copper sulphate, and tests of this treatment in all the affected areas rapidly substantiated this claim. Tests over a number of years in northern Europe indicate that reclamation disease in oats can be prevented by soil applications of 50 to 100 kg. copper sulphate per hectare, the heavier amount being advocated in Holland if the soil is rich (over 8 per cent.) in humus. In

Germany the standard rate of application seems to be 100 kg. per hectare, and in 1937 it was calculated that adequate treatment of the area liable to reclamation disease would require an annual consumption of 1,275 metric tons of copper sulphate. In Denmark the application of 50 kg. per hectare is recommended, increased yields of barley and oats averaging about 500 kg. per hectare having been obtained from this treatment in a series of 249 experiments; it was reported in 1934 that the disease was waning in Jutland as a result of treatment with copper sulphate. In Sweden the oat yield was trebled with an application of 100 kg. copper sulphate in 1936, and the fertiliser recommended for use on reclaimed peat is 200 kg. superphosphate, 150 kg. potash, 200 kg. sulphate of ammonia, and 100 kg. copper sulphate, which is stated to have proved notably successful in securing good crops. Lesser amounts of copper sulphate (20 kg. per hect.) have proved adequate to check the loss of the meadow grasses on reclaimed peat bogs in Poland, while in Western Australia, where the soil conditions under which the disease occurs may be very different from those in Europe, 15 lb. copper sulphate per acre has proved sufficient to give a crop of 25 bushels of wheat per acre where none was harvested without treatment; larger applications or the addition of manganese, zinc, or magnesium to the copper did not appreciably improve the results. In further experiments in Western Australia quite good results have been obtained with only 5 lb. copper sulphate per acre when used with 1 cwt. superphosphate. Yields of potatoes and truck crops were also improved by this treatment. The beneficial effects of copper sulphate applications have been observed in Denmark and Germany to last for three or four years.

More recently an effort has been made in Germany to replace valuable copper sulphate by relatively worthless low-grade, copper-containing ores and slags. Large quantities of these are required, up to 900 or 1,000 kg. per hectare, as the copper content of some that were tested was only 0·41 per cent. Other elements present included zinc (1·9 per cent.), manganese (0·33 per cent.), and magnesium (0·50 per cent.), all of which might be useful, but the presence of 0·14 per cent. lead suggests that their employment may not be free from danger.

Marked varietal resistance to reclamation diseases has been observed in oats and barley. Black oats as a class are more resistant than the white, and several varieties are known which can be cultivated satisfactorily on soils liable to the disease; these include some that have been bred specially for moorland cultivation in Sweden, Germany, and Holland, and that also resist grey speck, as well as the American Black Mesdag. *Avena strigosa* is very highly resistant. There is some indication that resistant varieties are capable of extracting more copper from the soil than susceptible ones but none that the copper requirements of the two categories of plants differ. Some of the resistant varieties have given better yields without treatment than can be obtained from good susceptible varieties given heavy applications of copper sulphate. In barley the 4-rowed types include some that are resistant to reclamation disease.

Histologically copper-deficient oats in Western Australia were found to have a thin-walled epidermis with poorly developed cuticle, thin-walled unlignified cells with few fibres in the cortex, and thin-walled bundle-sheath and pith cells. The leaf mesophyll was less differentiated than normal, with small and compact

cells. Hairs were reduced in number on the leaves and roots. In the yellow areas the chloroplasts were disintegrated and the cells contained brown tannin-like bodies.

A chlorosis of deciduous fruit trees curable by soil applications of copper sulphate at the rate of $\frac{1}{4}$ to 2 lb. per tree was reported from South Africa in 1932. Peach, apricot, and plum trees showed a marked yellowing of the leaves, the interveinal areas of which were very pale green to bright yellow. Terminal growth ceased and rosettes formed at the tips of the twigs, which then died back, while lateral buds sprouted lower down. Little or no fruit was borne. In apples, chlorosis was often absent but the other symptoms were marked, while pears showed scorching of the apical leaves and die-back. The soils affected were deep, acid, sandy or sandy-loam, with a varying (poor to rich) humus content. The chlorosis was reported in 1934 to be very severe in peaches but was remedied by doses of copper sulphate sufficient to give 20 p.p.m. copper in the soil.

In Western Australia a similar apple disease known as ' wither tip ' or ' summer die-back ' has been known for many years, chiefly in orchards on leached soils or those ordinarily carrying *Eucalyptus diversicola* forest. Soil applications of 2 lb. copper sulphate per tree, as well as trunk and branch injections and spraying the leaves with copper compounds effected a marked improvement; zinc and other elements tested were ineffective.

In California, Bartlett pears are very prone to a disease termed exanthema, though in its general symptoms it resembles the die-back disease of South Africa and Australia more closely than the better-known exanthema of citrus. The terminal leaves and shoot tips die back, necrosis (marked by brown-orange striations) starting at the tip and margins of the leaf and extending inwards. By the end of the summer three-quarters of the current year's growth may be killed, and eventually the trees have a dense bushy appearance due to proliferation of dormant buds. Little or no fruit is borne. Affected trees respond rapidly to soil applications, injections, or sprays with soluble copper compounds, other heavy elements being ineffective. Analyses showed that the affected trees were deficient in copper. Apples are also affected in California, the symptoms being similar to those on pears. In plums, exanthema has been reported from California and Australia, the disease in this fruit more closely resembling exanthema in citrus in that, besides the die-back and resulting growth habit, gummosis is a marked feature. Olives are also affected in California, and avocados in Florida.

Exanthema in citrus was described from Florida as far back as 1896 and has since been recorded from almost all citrus-growing countries. As early as 1917 the beneficial effect of copper salts on the incidence of the disease in Florida was reported, while in Western Australia it was stated in 1925 that favourable results followed soil applications of copper sulphate, the spraying of the leaves with Bordeaux mixture and the introduction of copper sulphate in solution of 1 oz. to the gallon through the roots, though considerable injury was caused to the trees by the latter treatment. In New South Wales, where the disease has been known since 1909 and was regarded as the most important citrus disease of the coastal regions, both Bordeaux mixture spraying, and soil applications of copper sulphate given in 1927-8 practically cured the disease. Investigations reported from Cali-

fornia in 1935 showed that the leaves of affected citrus trees contained less copper than normal and that the disease occurred in soils deficient in copper.

The changes induced by copper deficiency are more remarkable in citrus than in any of the other plants in which it has been studied. The dominant symptom is a widespread gummosis: gum pockets form in the young shoots and cause blister-like swellings from which an amber-coloured gum exudes, gum-smears show on the stems and brown gum-soaked areas develop in the fruit, which often remains pale yellow. With the gummosis are associated deep-seated leaf changes, many affected plants producing 'giant' leaves here and there and the foliage assuming a deep-green colour, though some leaves show considerable chlorosis or 'frenching'. Affected trees are stunted, the shoot internodes are shortened, a bushy growth of lateral dormant buds takes place, and there may be considerable die-back and fruit drop. Severely affected trees may produce no marketable fruit year after year.

Causes of Copper Deficiency Symptoms in Plants

As in the other deficiency diseases already discussed, there is evidence that, while there may sometimes be an actual deficiency of copper in the affected heath moor soils, the element is often present but unavailable, especially in marshy soils. There is no doubt that the copper content of affected cereals is lower than normal, and in Holland livestock on affected pastures or fed with hay from them suffer from 'licking disease' which is alleviated by providing copper sulphate 'licks'. Fixation of copper is known to be high in heath soils and appears to depend on some humus constituent, though fixation by bacteria has also been suggested [21]. From the black peats ('gliede') of Holland an organic substance termed 'gliedine' has been isolated which, when added to soil in which healthy oats and peas were grown, produced typical symptoms of reclamation disease. It has been suggested, however, that gliedine acts not by poisoning but by fixing copper. A method of measuring approximately the availability of copper in soils has been based on the colour changes produced in the spore heads of *Aspergillus niger* by varying amounts of copper; if an adequate quantity is present, the heads are black, but they change to brown and yellow as the amount of available copper gradually diminishes. That reclamation disease is due to actual copper deficiency in the crop is supported by analyses, by the results of water cultures which showed that the symptoms could be produced when copper was withheld, by the beneficial effect of applying soluble copper compounds directly to the foliage, and by the fact that no crop injury results from adding 100 kg. copper sulphate per hectare to humus-rich moor soils with a high power of fixing copper, whereas even 50 kg. may cause injury on sandy moors low in humus.

Copper deficiency disorders appear in northern Europe to be more closely restricted to certain soil types than those due to deficiencies of manganese or magnesium. Reports generally agree in associating reclamation disease with colloidal or amorphous types of peat, especially those that tend to be saturated in winter and dry out in the summer. The beneficial effect of clay, which has not been called into question, may depend on some alteration in the physical properties

of the soil, and the suggestion has even been made that copper sulphate acts not by correcting a copper deficiency but as a coagulant of the peat colloids, so that the capillary system of the soil is opened and the movement of the soil moisture is facilitated. Green manuring and improved tillage are advocated in Germany and may have similar effects. Another hypothesis advanced to explain the action of copper is that it regulates the oxidation-reduction potential of the soil by acting as a catalyst to activate oxidation. In the absence of an oxidation activator the reduction processes in the types of soil affected by reclamation disease may lead to the accumulation of toxic substances (e.g. ferrous iron). Soil reaction is not an important factor, but there are many reports that liming promotes reclamation disease.

While the action of copper on the soil and in the plants suffering from reclamation disease is not yet clear, the necessity for available copper seems to have been established by the characteristic symptoms produced in oats, beet, and other plants by growing them in water or sand cultures without copper and their cure when copper was added. It has been suggested that copper is necessary for the formation of chlorophyll in sugar beet.

MAGNESIUM

Of the three main diseases intensively studied in Germany and Holland from about 1922 onward, as a consequence of the attention being given to the cultivation of poor or reclaimed soils, that termed ' Hooghalen ' disease in Holland and soil acidity disease in Germany was the last to be associated with deficiency of a particular element in the soil. Manganese and copper were established as the elements, deficiency in which led to the development of grey speck and reclamation diseases respectively, before there was any suspicion that the so-called soil acidity disease was also a deficiency disorder, the element involved being magnesium.

Soil acidity disease began to attract attention in Germany in 1920 and was described in 1923. Coincidentally it was reported as a new disease in Holland. In 1931 magnesium deficiency was recognised in Germany to be responsible for a diseased condition of cereals, and in 1932 the similarity of the symptoms of soil acidity disease in that country and of those of the tobacco and maize disease termed ' sand drown ' in the United States was pointed out. Work in the United States had previously established that ' sand-drown ' was a magnesium-deficiency disorder.

The symptoms of the soil acidity disease in its most typical form on cereals are characteristic. The foliage is of a distinctive yellowish-green or livid colour, with longitudinal pale green to white mottling of the leaves between the veins, the resulting striations resembling the stripes on the skin of a tiger. The leaf tips are often reddened and in-rolled ; in rye the mottling may be yellowish to brownish, and the whole blade may fail to unfurl, so that newly formed leaves are held tightly enclosed. Reddening may also occur at the base of the haulm and in the centre of the nodes. The roots are elongated and have few root hairs.

The disease is most evident in Holland in light acid soils. Most of the soils in which magnesium deficiency symptoms have been encountered are easily leached, and it is probable that an actual deficiency of magnesium rather than its unavailability to the plant is involved, though the antagonism of other ions in the soil may play a part. Complete control of the Hooghalen disease by soil applications of 80 kg. magnesium sulphate per hectare has been reported.

Grain crops have been reported in New Brunswick to suffer from a similar disease, and control has been effected in certain cases by soil applications of magnesium sulphate. In the United States maize was one of the first crops observed to suffer from sand drown, the symptoms being more like those reported in cereals suffering from soil acidity disease in Europe than those found in tobacco in the American disease to which the name ' sand drown ' was first applied. Applications of magnesium compounds equivalent to 20 to 40 lb. per acre, according to the soil conditions and degree of leaching, gave effective control. In Texas the rice disease termed ' white tip ' also appears to show a resemblance to soil acidity disease. White chlorotic areas appear at the leaf tips and spread backwards over about half the blade, the tip meanwhile drying up. When the flag leaf is affected the panicle is held tightly enclosed and has difficulty in emerging; the ear is distorted and sterile or with only few grains. Soil and water cultures of rice indicate that similar symptoms result from withholding magnesium, though it was necessary to add calcium as well as magnesium (27 p.p.m. of each) in order to restore normal growth.

A chlorosis of tobacco and other crops on light sandy soils liable to heavy leaching in the South Atlantic Coastal Plain of the eastern United States was described under the name ' sand drown ' in 1922. In tobacco the chlorosis usually begins at the tip and margins of the older leaves and then spreads inwards and downwards and also to the younger leaves. The interveinal spaces are blanched but the main veins and adjoining area remain green. When ' cured ', the leaves are dull, faded, thin, light, and lacking in elasticity.

Sand drown, which has also been reported in Canadian and Nyasaland tobacco crops, is amenable to control by magnesium compounds—the sulphate or chloride as well as dolomitic (magnesium) limestone. The amount of magnesium required is usually about 20 lb. per acre. Organic manures which contain appreciable quantities of magnesium have also given good results. The symptoms have been produced in pot cultures deficient in magnesium, but in water and sand cultures certain differences have been observed which have led to some doubt whether magnesium deficiency alone accounts for the disease, though adequate magnesium is certainly essential. The quantity of magnesium that must be present in the leaf for normal functioning appears to be about 0·25 per cent. of the dry weight.

Soya beans were found to suffer from a disease resembling sand drown in tobacco and equally amenable to control by magnesium applications, in the same area in which the tobacco disease was observed. In this area, and further north, potatoes in acid leached soils have been extensively affected by a chlorosis of the lower leaves which in severe cases turn brown and become thickened and hardened. A similar disease has been observed in Canada. In eight years' experiments in Maine effective control of this disorder in potatoes was given by magnesium

applications sufficient to supply 25 lb. magnesium oxide per acre. In 1934 yields were increased by over 30 bushels per acre by this treatment. In Rhode Island an increase of 100 bushels per acre with 100 lb. magnesium sulphate was reported in 1934, and it was stated that the sulphate, hydrate, or magnesium limestone were all equally effective if, at least, the equivalent of 25 lb. magnesium oxide were applied. In Canada an equivalent of 20 lb. magnesium oxide has been reported to give good results.

Sand drown has also been found to affect cotton in South Carolina, the chlorosis in this crop being followed by the development of a red colour in the leaves.

A disease of young cabbages and cauliflowers characterised by yellowish or yellowish-green blotches between the veins was reported on very acid magnesium-deficient soils in New South Wales in 1937. A similar cauliflower disease was seen in the United States that year, while at Long Ashton cauliflowers, brussels sprouts, and cabbages were affected in 1940 [27]. In the young seedlings the early formed leaves are thick, brittle, and in-curled. In older plants the yellow blotches coalesce to form bands between the still green veins, and the affected leaves (usually only the lower ones) are stiff, thickened, distorted, and eventually bronzed. These leaves are shed and the head (in cauliflower) is much reduced in size. The magnesium content of the leaves was found in Australia to be lower in diseased than in healthy plants, and control has been effected in America by heavy applications (not less than 300 lb. per acre) of magnesium oxide.

Magnesium deficiency is considered to be responsible for one of the ' yellowing ' diseases to which sugar-beet is liable in northern Europe. Beet grown in soil known to be deficient in magnesium in Belgium shows an interveinal chlorosis of the outer and middle leaves. These symptoms have been produced in sand and water cultures deficient in magnesium, the plants dying in about two months if the deficiency is severe. Heavy dressings with potash are stated to promote the disease.

Magnesium deficiency diseases also occur in fruit and forest trees. Deficiency in magnesium, as in zinc and copper, is responsible for a characteristic disease of citrus trees in the United States, where the magnesium deficiency disease is termed ' bronzing '. It begins as a yellow chlorosis of the early spring flush of leaves, the youngest leaves escaping discoloration. This changes to a bronze colour, which is more pronounced as the magnesium content of the leaves is lessened and the calcium-to-magnesium ratio increased. The addition of magnesium to the complete fertiliser used for citrus has decreased the disease. In culture work in California it was found that magnesium deficiency in citrus caused a yellow striping along the dark-green midrib, followed by a general bronzing of the leaf.

Magnesium deficiency in fruit trees in acid sandy loam soils in England was reported in apples and black currants in 1939 and soon afterwards in plums and gooseberries. In apples the pale-green interveinal areas suddenly turn brown and dry out. In some cases the foliage assumes a purplish colour. Black currants show well-marked patterns, usually in red, on the upper leaf surface. In plums the chief leaf symptoms are interveinal chlorosis and marginal scorching, followed by defoliation. The leaves are very thin and tend to form the absciss layer pre-

maturely; this is probably why the affected apples and gooseberries were found to be very liable to spray injury. Older leaves are always the first to show symptoms. These appear in pot cultures if there is less than 0·40 per cent. magnesium oxide in the dry matter of the leaves, and it may require two or three seasons before the beneficial effects of magnesium dressing are evident. Clear indications were obtained that magnesium requirements are increased when liberal supplies of potassic fertilisers are given.

In New Zealand, magnesium deficiency has been found to cause a serious disorder of apples in acid leached soils. Varieties differ considerably in the resultant leaf symptoms: in some the interveinal spots found usually towards the centre of the leaves, progressively from the base towards the tip of the current season's leader, rapidly turn from light or greyish green to purplish or dark brown, in others the chlorosis is more like that described elsewhere. Premature defoliation follows, so that only a tuft of green foliage is left at the tip of the shoot. This was most marked where the trees had been liberally fertilised with potash. Both the size and colour of the fruit are diminished. A correlation was established between the intensity of expression of the symptoms and a low magnesium content of the leaves. Cures were effected by the injection of a 0·25 per cent. solution of magnesium sulphate into the wood.

In Canada also severe cases of injury to apples from magnesium deficiency have been seen in recent years, especially after heavy leaching rains. In some varieties the leaves are marked by wide yellow bands alternating with the narrow green ones along the veins. In others, blotches form at the leaf margin and the interveinal strips die early; leaf rolling and defoliation follow. In others again the tendency is for the whole leaf to become blanched. The soils affected are dark brown and very acid, with little calcium or magnesium, but while the leaves had a low content of magnesium, their calcium was about normal.

Studies carried out in South Africa by the East Malling injection technique mentioned below, indicate that deciduous fruit trees in the Western Cape Province suffer not infrequently from magnesium deficiency.

In forest trees the only disorder hitherto attributed to magnesium deficiency is a 'yellow tip' disease of conifers (pine and spruce) in Germany, reported in 1937. In pines the needle tips turn reddish brown, in spruce pale or livid green. Control has been effected by the application of 180 lb. per acre of sulphate of potash-magnesia.

Causes of Magnesium Deficiency Symptoms in Plants

Low available magnesium in the soil appears to be seldom found except in sandy soils liable to leaching and usually strongly acid. The outbreak in brassicas at Long Ashton followed a July rainfall of nearly 8 inches. In the area along the Atlantic seaboard of the United States in which sand drown is most prevalent, the affected soils have less than 0·2 per cent. of magnesia. The element is a constituent of the chlorophyll molecule, but the degree to which chlorosis is induced by its lack differs considerably from one species of plant to another. Limiting values of 0·25 and 0·4 per cent. of the dry matter of the leaf have been

given for different plants. Tests of 17 crops in Massachusetts reported in 1935 indicated that buckwheat and spinach were the most sensitive, followed by turnips, mangolds, maize, and tobacco. Clover and potatoes were stated to be less affected, but water and sand cultures in Holland showed that magnesium deficiency caused very severe symptoms in the latter crop, the older leaves varying from almost pure white to pale yellow according to the scarcity of the element. Sand cultures of a wide range of greenhouse ornamentals showed that, in general, magnesium deficiency causes reduced rate of growth, chlorosis of the older leaves, yellowing followed by abrupt necrosis of the interveinal areas, with puckering and shedding of the leaves in some species, a scarcity of roots, and poor coloration of the flowers.

There have been a few reports of increased susceptibility to fungal diseases (e.g. *Septoria nodorum* in wheat) as a concomitant of magnesium deficiency.

SULPHUR

Very few cases of symptoms of disease attributable to sulphur deficiency have been reported and only one of these, that affecting tea in Nyasaland, has been studied in any detail.

Tea growing in dark-brown friable soil of lateritic origin in Nyasaland was reported in 1927 to be suffering from a disease, ' tea yellows ', which was at first attributed to *Botryodiplodia theobromae*. The rainfall in the affected areas varies from about 60 to 100 inches annually, and heavy falls, amounting to as much as 8 inches in twenty-four hours, sometimes occur. Analyses indicate that the affected soils are not inferior to good tea soils in other countries.

Fields suffering from the disease are recognisable by their yellowish appearance, due in the early stages to the development of a pale-green to yellow interveinal mottling of the leaves, separated by a network of green veins. The progress of the disease is slow : the yellowing becomes more generalised, though the green veins remain prominent for a considerable time ; the leaves are reduced in size, narrowed, with upturned edges, stiff and brittle ; their tips and margins may become necrotic and turn dark brown. The internodes are shortened, the shoots become thin and weak, and severe defoliation sets in until often only the youngest leaves are left as tufts at the end of the twigs. At the same time lateral buds lower down proliferate into stunted shoots with very small yellow leaves. A progressive die-back occurs and death of the bush may follow in a few years after the first symptoms are seen, or the defoliated bushes may survive with the production of weak basal shoots for a considerable period. The lateral feeding root system of young plants is often poorly developed, especially in the upper layers of the soil, but the older bushes show little disorganisation of the roots except that in severe cases they are usually depleted of starch.

Histologically, the small yellow leaves of affected shoots show an arrest of development ; their palisade cells are poorly differentiated and the mesophyll consists of crowded, rather uniform cells, with thin walls and much reduced intercellular spaces. In the chlorotic early stages, the chloroplasts are reduced in

size and number, and they degenerate completely in the later stages.

After it was shown that yellows was not due to fungal infection, tests with fertilisers were carried out. These indicated that no benefit resulted from the ordinary nutrients, nitrogen, phosphorus, and potash, but that when sulphates were present recovery was initiated. Further investigation established that sulphur applied either alone or as the sulphates of ammonium, potassium, sodium, or magnesium always effected a marked improvement, badly diseased plots giving as much as 85 per cent. improvement after treatment. Applications of sulphur-containing fertilisers to virgin soil in this area resulted in the establishment of uniformly healthy bushes, whereas without treatment an average of 36 per cent. yellowed plants was found, in particular fields examined in 1931, after the first year's growth.

Water cultures confirmed these results. Exactly similar symptoms to those observed in the field appeared when sulphur was withheld. Leaf analysis indicated that the chlorotic leaves, especially the younger ones, contained less sulphur than the healthy. Nevertheless the soils concerned do not appear to be seriously deficient in sulphur, as those examined contained on the average 0·031 per cent., which is within the range normally found in the soil. The action of sulphur applications in the control of the disease, therefore, is as yet unexplained.

Since the use of sulphur-containing fertilisers has become general in the areas affected, the 'yellows' disease of tea (which has also been doubtfully reported from Tanganyika) has ceased to be a serious trouble in Nyasaland.

Tests with tobacco, which is extensively cultivated in Nyasaland, showed that it is also subject to the yellows disease when grown in the affected tea areas, and that applications of sulphur prevented the disease. In the United States an unusual form of yellowish, apical chlorosis of the upper few leaves of topped tobacco plants, observed in 1934, is tentatively attributed to the same cause [26]. The disease occurs in hard, cracked, hilly soils subject to erosion. Affected leaves show a greyish-yellow discoloration of the tips, sometimes extending well down along the margins; in these cases the sulphur content of the tissues was reduced, the magnesium, phosphorus, and calcium being also low. On 'curing', the colour turns chamois yellow, or honey yellow, instead of the normal brown. Another possible case of sulphur deficiency has been reported in rice in Burma, where yellow stunted plants were restored to normal health when treated with iron or copper sulphate or sulphuric acid, while the chlorides of iron, manganese, or sodium and ammonium nitrate had no effect. A combined deficiency of nitrogen and sulphur has been reported in sugar-beets grown for seed in Oregon, where the addition of sulphur alone had no effect on the yield but sulphur plus nitrogen cured the condition [25]. Culture work on the symptoms of mineral deficiencies in tobacco has shown that lack of sulphur induces a chlorosis in which the veins are paler than the interveinal areas; this unusual symptom has also been reported in sand cultures, lacking in sulphur, of a wide range of ornamental green-house plants tested in the United States. In soya beans, sand cultures without sulphur gave stunted plants with yellowish-green leaves and small leaflets, the upper foliage being the first to be affected. The stems were thin, hard, and bare from shedding of the lower foliage. Root development was poor, but the root system

was less stunted than the green top. Sunflower, kale, rape, and mustard were similar, but in tomato the lower leaves were the first to turn yellow. In coffee, the effect of sulphur deficiency has been studied in water cultures in Java : the plants without sulphur had yellowish convex leaves and small thin roots.

Several other crop diseases have been reported in which the application of sulphur or compounds containing the sulphate radical have effected an improvement. In some of these the influence of sulphur is doubtless to render available other minerals, as in the lime-induced iron deficiency chlorosis and in manganese deficiency diseases mentioned above.

POTASSIUM

It would lead too far to discuss the physiological disorders of crops inadequately provided with the major elements of plant nutrition, nitrogen, phosphorus, and potash, or the effects of a shortage of lime. The specific results of potash deficiency in certain cases, however, should be briefly referred to, since such characteristic diseases as scorch in fruit trees, ' rust ' in cotton, ' rougeau ' of the vine, and the like, have been traced to this cause.

Intensive work on the causes and control of leaf scorch of fruit trees has been in progress at Long Ashton Research Station since 1913. Excluding the type due to the action of salt spray from sea winds, the bulk of the cases to be found in the south of England are associated with certain soils (' scorching soils ') which occur in scattered patches of various sizes. These soils are, for the most part, light sandy or stony, but are sometimes light to heavy loams. In the scorched patches the foliage appears brownish from scorching of the leaf margins, most marked in dry summer weather. The affected plants are stunted, their root systems poorly developed, and the fruit reduced in size.

In northern Europe a similar type of scorching has been a serious trouble in red and black currant, and gooseberry plantations, during the last twenty years or so, especially in Scandinavia, Holland, and Germany. Deciduous fruit trees are affected by leaf scorch in Australia and apples in Tasmania and Canada.

While marginal scorch remains the predominant symptom in fruit trees, those affected in Australia often show an extension of the discoloration until only a small part of the centre of the leaf remains green. In Tasmania also, affected apples are marked by leaf spots in the interveinal spaces as well as by marginal scorch. Pot and field experiments in Holland showed that scorched red currants similarly develop dark purple scattered spots in addition to the marginal lesions. Peaches are reported in the United States to have, in place of leaf scorch, a reddening of the leaf margin, which is inrolled towards the midrib, the leaf surface being much crinkled and tinted reddish ; plums also have this upward inrolling but the margins are pale or brown, and in severe cumulative attacks growth is checked, and fruiting in young trees retarded [4]. In California, marginal leaf scorch in citrus trees is accompanied by gum exudations from the leaves and stem.

In an extensive series of pot experiments at Long Ashton reported in 1928,

leaf scorch of gooseberry, black currant, and raspberry only occurred in the cultures deprived of potash. Since then evidence has accumulated that the primary cause of marginal scorch of fruit trees is lack of potash. Successful control has been effected in England by soil applications of potash, and in gooseberries by spraying the leaves with 1 per cent. solution of sulphate of potash. The soils at 23 affected centres in England were all found to be low in potash in the surface layers as judged by accepted agricultural standards. A reduction in the potassium content of the ash, and of the dry matter of scorched leaves has also been found. In Germany, good control of scorch in currants and gooseberries was secured in 1930–31, by soil applications of 150 gm. sulphate of potash per tree, and even half this amount effected a considerable improvement. In another series of German experiments, satisfactory control of scorch in red currants in poor soil was given by potash dressings (other than the chloride, which was injurious) sufficient to supply the equivalent of 65 gm. potassium oxide per plant. Though it has been stated in that country that scorch will appear in red currants when the potassium in the leaf falls below 1 per cent. of the dry matter, it is actually impossible to fix such definite limits, since the limiting values for scorch vary with the other constituents in the leaf. Scorch in red currants has been controlled in Holland and Denmark by dressings of 1200 and 500 kg. of sulphate of potash per hectare respectively. Fairly heavy potash dressings are stated to be necessary to prevent scorch in deciduous fruits in Australia, while in Tasmania, as in England, scorch is not found, as a rule, where adequate potash fertilisers are used.

A close connection exists between the development of marginal leaf scorch and the water relationships of the soil and plant. When these are satisfactory, a low, available potassium content of the soil does not necessarily lead to scorch. Water-logging is a frequent precursor of scorch in England and Canada. In England scorch has also been induced by gradually drying out the soil and then wetting the foliage. It has been found that the marginal areas of affected leaves are first turned yellow by the destruction of chlorophyll during drying out of the cells, and browning only sets in when the dried tissues again reabsorb moisture. The proximate cause of scorch seems to be a sudden discontinuity in the transpiration stream, which is local, and does not arise in the conducting system of the shoot.

Another important factor in promoting scorch is a high ratio of nitrogen to potassium in the plant. The recognition of the importance of this factor has led to an increase in the practice of 'grassing down' orchards: grass takes up much of the available nitrogen in the soil and reduces its intake by the tree, but the risk of over-reduction leading to a shortage of nitrogen must be guarded against, either by judicious use of nitrogen fertilisers or by such measures as cutting the grass and restoring it to the soil by mulching around the trees.

Varietal and root-stock effects on the intensity of marginal leaf scorch are evident in apples. Unworked stocks, as well as commercial varieties, differ considerably in their proneness to the disease, and while some varieties develop scorch on all stocks, others do so only on some, the dwarfing stocks being particularly liable to promote scorching.

The effects produced by 'potassium hunger' have been described and illus-

trated in a great variety of other plants. In some of these, as in tea, marginal leaf scorch like that of bush fruit is caused and the leaves are shed. In others the symptoms are so characteristic that the diseases received definite names before their true nature was recognised, as for instance the well known ' rust ' in cotton in the United States, and ' brunissure ', ' rougeau ', and allied disorders of the vine in France. Tomatoes grown under glass in England suffer from disorders termed ' blotchy ripening ' and ' green back ', one of the chief causes of which is lack of potash ; potash deficiency in tomatoes impedes the translocation of starch, accelerates blossoming, and lengthens ripening. In the blotchy fruit it is suggested that the sugars are used to build up the cell membranes instead of taking part in the normal processes of ripening, so that the trouble may be regarded as primarily due to a derangement of carbohydrate metabolism [18]. Symptoms similar to those of the destructive root rot (' mentek ') of rice in Java have been given by withholding potash from the nutrient solution in which the plants were grown. The remarkable effect of potash in reducing cotton wilt (*Fusarium vasinfectum*) in certain areas in the United States [29] has been attributed in considerable measure to the prevention of potash hunger, and it has also been claimed in England that potash deficiency, in certain cases at least, predisposes broad beans to chocolate spot (*Botrytis cinerea*), which can be largely obviated by a dressing of 1·5 cwt. per acre of muriate of potash [23]. Potash deficiency increases the tendency to flooding of the intercellular spaces of tobacco leaves under certain atmospheric conditions and is said thereby to increase their liability to wildfire (*Pseudomonas tabaci*) [1].

CALCIUM

References to nutritional deficiencies of calcium in plants are not very numerous, this aspect of the agricultural importance of the mineral being far overshadowed by its rôle as a soil improver and its importance for farm livestock.

Culture work in the United States and Holland has established that symptoms of disease are induced in a great variety of plants by withholding calcium from the medium in which the plants are grown. These symptoms have been described in tobacco, potato, citrus, peach, coffee, peas, beans, soya beans, and some fruit trees. In several plants much the same type of injury is said to be caused as results from lack of boron. In tobacco the first symptoms are seen in the bud or top leaves, which become diseased as they unfold and show a hooking-down of the tip of the leaf and deformation followed by death of the margins ; in severe cases the terminal bud dies. In potatoes the first symptom is the development of a pale-green band along the margins of the young leaves, which become wrinkled and do not unfold properly. In severe cases the failure of the young leaves to unfold becomes more general and the whole top dies. The tubers show the disease, which has been termed ' rusty spot ' or ' medullary necrosis ' and which is known as a field disease in the Dutch East Indies, Holland, and probably South Africa.

Medullary necrosis was first described in 1926, under the name of rusty spot, as causing serious injury to potatoes in Java and Sumatra. There are no external

symptoms to mark the disease, but the tubers contain scattered rusty spots in the parenchyma internal to the vascular ring; a more diffuse rusty discoloration develops from the stolon end and there may be similar lateral patches extending along inside the vascular ring. The necrotic cells have thickened walls and the spots become isolated by a cork cambium. The disease also occurs in Holland on poor, sandy, acid soils. Varieties differ somewhat in their reaction to it and in some (e.g. King Edward) it may cause hollows in the tuber. It can be readily distinguishable from the virus symptom known as 'net necrosis' and is also considered to be distinct from, though having a certain resemblance to, 'spraing'. Dutch workers think that the tuber disease known as 'internal brown fleck' in South Africa may be the same as medullary necrosis; brown fleck is confined to acid sandy soils, on which it seems to affect all varieties.

Heavy liming has been found to reduce considerably the incidence of these internal potato tuber discolorations in the Dutch East Indies, Holland, and South Africa: percentage reductions of affected tubers from 96·8 and 94·8 to 26 and 10·4, respectively, were obtained by liming in two experiments in the Dutch East Indies, the spotting being so reduced in the remainder that they were marketable. In these tests, and in South Africa, superphosphates also effected an improvement, but sulphate of ammonia increased the injury.

There have been several reports of calcium-deficiency diseases of apple trees. Tests by the injection technique revealed the existence of this nutritional trouble in Devon some years ago, and more recently it was found widespread in deciduous fruit trees in South Africa. Apples in Queensland suffered from a disorder marked by the development of blotches on the leaves of Jonathans and of a wine-coloured tint on those of Gravensteins. In experiments on the control of this condition it was found that it practically disappeared after an application of quicklime, at the rate of two tons per acre, was given, and it is regarded as a calcium-deficiency disease.

A physiological disease of cauliflowers in very acid soils in Long Island, in the United States, where it is known as 'whiptail', and also in New South Wales, where it may spoil 20 to 50 per cent. or even sometimes all the plants, is also attributed to the deficiency of lime. The plants are stunted and show various malformations such as a strap-like narrowing and ruffling of the leaves, and loose, unmarketable heads with leaves coming through the curd. Satisfactory control has been geven by liming, the Australian cases sometimes requiring 1½ to 2 tons per acre. A similar disease has been observed in cauliflowers and savoy cabbages in south-western England, apparently under quite different soil conditions.

Causes of Calcium Deficiency Symptoms in Plants

Calcium salts have been shown to promote the absorption of other nutrients by the roots of plants; the presence of calcium ions can make others physiologically available, and when calcium is deficient in the soil, malnutrition may result from other nutrient ions being unavailable or even from their being leached from the roots. In the development of plant tissues calcium appears to be important mainly from the part it plays in the formation of the cell wall, particularly the middle

lamella. Little seems to be known, however, of the causes that result in the symptoms of calcium deficiency hitherto observed in the diseases mentioned above.

GENERAL CONSIDERATIONS ON THE DEFICIENCY DISEASES OF PLANTS

In many of the deficiency diseases mentioned above, the effect is a syndrome or group of symptoms sufficiently characteristic to have established in popular usage a name as specific in its application as those of many of the diseases caused by fungi. Heart rot of beet, brown heart or ' raan ' of turnips, grey speck of oats, marginal leaf scorch and exanthema of fruit trees, marsh spot of peas, are all terms the meaning of which to the grower is as precise as is that of the terms smut, scab, or canker applied to parasitic diseases. Many others, however, are marked by less definite symptoms — chlorosis, yellowing, defoliation, and the like — and their cause is less easy to determine. The plant pathologist is liable to be asked for advice on all such diseases and he must know what to look for and when to seek the aid of his chemical colleagues. It must also be borne in mind that such symptoms as chlorosis or yellowing are, by themselves, of little diagnostic value, for they can be the result of a fungal or virus disease or be merely due to some environmental condition such as low soil temperature, or even have a genetic origin as in strawberry or maize, or result from mutation, as in barley.

In a considerable number of these deficiency disorders it is doubtful whether an accurate diagnosis can be based on symptoms alone. A chemical determination of the mineral content of the leaf or other organ of the plant is sometimes of value, but several instances have been given above of the difficulty of relying upon this method, due to the fact that the limiting value for a particular element may be dependent on the form in which it occurs in the cell, or the presence of other elements : thus, analysis may indicate an adequate supply when in reality a shortage exists. This is still truer when applied to the soil, for it is abundantly evident that questions of availability of the ' minor ' elements in the soil are at least as important as with the ' major ' elements, phosphorus and potash.

Yet there are few classes of plant diseases in which accurate diagnosis is more necessary than those referred to in this chapter. Hence it is that such work as that developed at East Malling Research Station, where the deficiency is determined by direct test of a range of elements, holds such promise of useful application.

Methods have been perfected at East Malling, and applied there and in South Africa, by which the injection of any part of a plant from the main stem to a portion of the lamina of a leaf can be carried out with precision, and the leaf results can be used to give a rapid indication of the existence of a lack of some element of importance to the proper functioning of the plant. The methods have been particularly successful, sometimes after preliminary indications have been given by leaf analyses, on apples affected by lime-induced chlorosis and by nutritional deficiency of calcium, and on cherries suffering from a combination of iron and manganese deficiency, in England ; while in South Africa, where deficiency

troubles are prevalent in various fruits, they were effective in the detection of a wide range of mineral deficiencies. The adequacy of the supply of the various minerals in the leaf is revealed by the response given by the injection of one or more of them into immature leaves (several can be tested simultaneously by using different leaves): in a short time (up to 14 days as a maximum) evidence of the particular shortage is usually available. The results have been checked by leaf analyses, for which spectrographic methods have proved particularly suitable. Iron, manganese, and zinc were the commonest trace element deficiencies encountered in deciduous fruit in the Western Cape Province of South Africa under similar soil conditions, often in the same orchard; magnesium deficiency also appeared to be fairly prevalent, followed by nickel, cobalt, copper, and boron. Deficiencies in the major nutritional elements, nitrogen, potassium, and phosphorus, were also included, and definite evidence was obtained of calcium deficiency in a considerable number of cases.

One of the interesting results of the tree-injection work in South Africa is the confirmation it has given of the fact, already mentioned, that total content of an element in an organ such as a leaf does not necessarily give a measure of its functional adequacy. This has been clearly established with iron, where the most marked response to iron injections has sometimes been obtained on plants the leaves of which actually contained unusually large amounts of iron, and it probably holds good also for manganese, zinc, and boron.

Much of the existing knowledge of deficiency diseases is of comparatively recent, some of very recent, acquisition. In many cases treatment has outstripped research into the real causation of the disorder. There are abundant indications that the processes leading to deficiency symptoms may be very complex, and there is hardly a case in which a clear picture can be drawn of the physiological disturbances brought about. As more knowledge becomes available, it is probable that generalisations can be made which will simplify treatment. Similarity in the soil, conditions under which shortage of manganese, zinc, and iron occur have already been noted, and where liming promotes the appearance of the symptoms (as in boron, iron, manganese, and copper deficiencies) there may be more generalised causes at work than are now apparent. So also the indications of injury to the chlorophyll apparatus of the leaf cells in so many cases may mean that some general cause or group of causes is involved, the understanding of which may help in remedying the trouble.

In the present state of knowledge it is, perhaps, justifiable to regard tea yellows as due to a deficiency of sulphur or citrus exanthema to one of copper. Yet the evidence for this is simply that the disease can be cured by supplying the element in question. The result may not be merely to give the cells the sulphur or copper they require, but to enable some other constituent to do its work or to neutralise or even prevent the formation of some toxic substance which the plant takes up from the soil. Further investigation is required before the true cause of these diseases is fully understood.

In a limited number of observations on the reaction to parasitic attack of plants suffering from deficiency diseases, the indications seem usually to be in harmony with those mentioned in an earlier chapter, namely, that the best resistance to

attack is offered by plants of which the nutrition is well balanced. Thus, it has been found that strawberries deprived of such trace elements as boron, manganese, copper, and zinc were much more susceptible to the powdery mildew, *Sphaerotheca humuli* (as well as to red spider), than those receiving a full complement of these elements. It is also of interest to note that deficiency in any one or more of a group of elements increases the susceptibility of gooseberries to ' spray injury '.

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Appendix

CLASSIFICATION OF THE FUNGI

THE fungi as a whole are divided into three classes : Phycomycetes, Ascomycetes, and Basidiomycetes. To these must be added the Deuteromycetes, or ' Fungi Imperfecti ', which are conidial fungi. Further investigation may show that some have a perfect or higher stage in the Ascomycetes or Basidiomycetes, the majority existing only as conidial forms, having probably lost their higher stages. (Prominence is given below only to the more important parasitic groups.)

Lower Fungi

Hyphae absent or rudimentary, or, hyphae not interwoven to form compact tissues, not regularly septate.

Class I. PHYCOMYCETES.—Sexual reproduction distinct, the process being usually completed within the sexual cells themselves. Asexual reproduction by zoospores, sporangiospores, sporangia or conidia.

Sub-class I. ARCHIMYCETES.—Sexual reproduction by zoospores (isogametes).

Order I. *Chytridiales*
" II. *Plasmodiophorales*

Sub-class II. OOMYCETES.—Sexual reproduction, with production of oospores.

Order I. *Monoblepharidales*
" II. *Ancylistales*
" III. *Blastocladiales*
" IV. *Saprolegniales*
" V. *Peronosporales*

Sub-class III. ZYGOMYCETES.—Sexual reproduction, with production of zygospores.

Order I. *Mucorales*
" II. *Entomophthorales*

Higher Fungi

Hyphae usually interwoven to form tissues at some stage in growth, especially in the sporophore, septate.

Class II. ASCOMYCETES.—Sexual reproduction more obscure, often in two phases, the sexual nuclei uniting in a cell derived from that in which they first came together, or sometimes reduced to the union of sister nuclei ; resulting in the production of ascospores within a mother cell or ascus. Asexual reproduction by conidia.

Sub-class I. PLECTOMYCETES.—
Order I. *Plectascales*
„ II. *Erysiphales*
„ III. *Exoascales* (*Taphrinales*)

Sub-class II. PYRENOMYCETES.—
Order I. *Hypocreales*
„ II. *Sphaeriales*
„ III. *Dothidiales*
„ IV. *Laboulbeniales*

Sub-class III. DISCOMYCETES.—
Order I. *Pezizales*
„ II. *Helvellales*
„ III. *Tuberales*
„ IV. *Phacidiales*
„ V. *Hysteriales*

Class III. BASIDIOMYCETES.—Sexual reproduction reduced to the union of sister cells or even of sister nuclei within a mother cell or basidium, resulting in the production of basidiospores. Asexual reproduction by conidia.

Sub-class I. HEMIBASIDIAE.—Basidia septate or not, often irregular in growth, basidiospores irregular in number, usually forming secondary spores on germination.
Order — *Ustilaginales*

Sub-class II. PROTOBASIDIAE.—Basidia of more strictly limited growth, septate into usually four cells, each bearing only one basidiospore.
Order I. *Uredinales*
„ II. *Auriculariales*
„ III. *Tremellales*

Sub-class III. EUBASIDIAE.—Basidia of strictly limited growth, aseptate, bearing a definite number of basidiospores, usually four.
Order I. *Hymenomycetales*
„ II. *Gasteromycetales*

Class IV. DEUTEROMYCETES (Fungi Imperfecti).—Asexual reproduction by conidia. Some are known to be conidial stages of higher fungi.
Order I. *Sphaeropsidales*
„ II. *Melanconiales*
„ III. *Hyphomycetales*

The first or lowest class of fungi is the PHYCOMYCETES or alga-like fungi. These include the aquatic fungi and the simplest land fungi. Their hyphae never unite into strands or tissues, and the mycelium is either filamentous or composed of isolated, rounded cells. Regular septation is never found, so that the living parts of the hyphae usually form a continuous cell. The characteristic asexual fructification is the sporangium. Conidia are also found in some orders. Sexual reproduction is of a distinct and simple

type, and the nuclei usually fuse in the same cell in which they come together. They are divided into three sub-classes: the ARCHIMYCETES, the OOMYCETES, and the ZYGOMYCETES.

The ARCHIMYCETES have their sexual reproduction by the union of zoospores (isogametes) or of two similar individuals. They are divided into two orders:

Chytridiales.—Usually one-celled fungi of very simple structure; the whole body in many cases consists of a rounded cell which becomes directly transformed into a sporangium. Sexual reproduction is variable in type and often unknown. No conidia are known. Mostly parasites in water plants, less often in land plants. The more important plant parasites are in the following families:

Olpidiaceae.—Parasites of low algal forms and of a few higher plants; cell-thallus forms zoospores.

Olpidium.

Synchytriaceae.—Cell-thallus forms a sorus of sporangia, or resting sporangia, both producing zoospores.

Synchytrium.

Cladochytriaceae.—A mycelium developed; zoosporangia and resting sporangia formed.

Urophlyctis.

Plasmodiophorales.—Parasites of higher plants. Thallus naked; spores produce a zoospore with two unequal flagella. One family.

Plasmodiophoraceae.

Plasmodiophora, Spongospora.

The OOMYCETES have their sexual reproduction by female cells known as oogonia, which are fertilised by male antheridia to form oospores. They are divided into four orders:

Monoblepharidales—Aquatic fungi with sexual reproduction; oogonium fertilised by antherozoids. Asexual, by zoospores. One family.

Monoblepharidaceae.

Monoblepharis.

Ancylistales.—Aquatic fungi, parasitic in algae and small water animals. The whole of a segment becomes an antheridium or oogonium or an asexual sporangium with zoospores. One family.

Ancylistaceae.

Lagenidium.

Blastocladiales.—Aquatic fungi, or soil saprophytes; septa only at reproduction. Thalli rhizoidal, differentiated into sporophyte and gametophyte; the asexual bears zoosporangia and thick-walled resting spores; the sexual is mono- or dioecious; gametangia have mono-flagellate gametes of unequal size. One family.

Blastocladiaceae.

Allomyces, Blastocladia.

Saprolegniales.—Aquatic fungi, usually saprophytic. Sexual reproduction by antheridia and oogonia, each oogonium usually bearing a number of oospores. In most cases no actual fertilisation occurs. Asexual reproduction by zoospores usually within elongated sporangia. Two families.

Saprolegniaceae.—Hyphae not constricted.
Saprolegnia, Aphanomyces.

Leptomitaceae.—With the hyphae constricted at intervals, but not truly septate.
Apodachlya, Leptomitus.

Peronosporales.—Chiefly land fungi parasitic on plants, a few also aquatic and saprophytic. Sexual reproduction by oogonia and antheridia ; fertilisation resulting in a single oospore. Asexual reproduction chiefly by zoospores within rounded sporangia. Conidia often formed from the sporangia by suppression of the zoospores. Three families.

Pythiaceae.—Parasitic or saprophytic. Sporangiophores little differentiated from vegetative hyphae.
Pythium, Phytophthora.

Albuginaceae.—Parasites ; sporangiophores arising in a layer under the epidermis ; sporangia in chains.
Cystopus, Albugo.

Peronosporaceae.—Obligate parasites. Sporangia or conidia singly at ends of branched sporangiophores.
Peronospora, Plasmopara, Bremia, Pseudoperonospora, Sclerospora.

The ZYGOMYCETES have their sexual reproduction by the union of two more or less similar cells to form a zygospore. The asexual reproduction is never by zoospores. They are divided into two orders :

Mucorales.—Land fungi, chiefly saprophytic. Zygospores formed by the union of two usually equal cells. Asexual reproduction typically by rounded air-disseminated spores formed in a sporangium, rarely also by conidia. Several families. Mucor, Rhizopus.

Entomophthorales.—Parasitic on insects, rarely on plants, or saprophytic. Sexual reproduction by zygospores formed by the union of two, often unequal cells. Asexual reproduction by conidia only. One family.
Entomophthoraceae. Entomophthora, Empusa, Basidiobolus.

The ASCOMYCETES include a great number of forms, all agreeing in having spores developed within a mother-cell, the ascus. In most, the asci are produced on, or in special sporophores ; these are known as perithecia when round or flask-shaped and enclosing the asci in the hollow cavity, and as apothecia when the asci are exposed at maturity. Conidia of the most diverse sorts are also usually borne. The mycelium is, on the whole, less aggregated into masses than in the BASIDIOMYCETES ; in the majority of cases, however, the asci are developed on a hymenium lining a sporophore formed by the union of many hyphae, and in some there is a considerable development of stromatic tissue in which a number of perithecia may be borne (compound stromata). Besides these sporophores, aggregations of hyphae to form sclerotia are found at times. The asci are usually of regular size, with eight spores. Sometimes there are less than eight spores, sometimes more, up to sixteen, thirty-two, sixty-four, or more.

Sub-class PLECTOMYCETES.—Asci formed directly on the mycelium or on special parts of it, not united in a hymenium enclosed in, or borne on a complex sporophore. In the *Exoascaceae*, which are parasites, the asci are formed under the surface of

the leaf, becoming exposed when ripe. In Endomyces they occur singly as lateral outgrowths of the hyphae. In Gymnoascus there is a rudimentary perithecium, formed of a loose web of branched hyphae within which the asci are entangled. The yeasts, or *Saccharomycetaceae*, differ from all the other higher fungi in having a much reduced gemmate mycelium, consisting of a single cell which buds off other similar cells, or may become converted as a whole into an ascus ; in some cases, at least, the formation of the ascus is preceded by a sexual act in which two cells and their contained nuclei fuse. Well-defined cleistocarps in *Aspergillaceae* and *Erysiphales*, opening irregularly. The three orders are :

Plectascales.—Arrangement of asci irregular. The principal families are :

Endomycetaceae.

Endomyces, Eremascus.

Saccharomycetaceae.—The yeasts.

Aspergillaceae.

Eurotium, Penicillium.

Erysiphales.—Asci within well-defined cleistocarps or cleistothecia, borne on a profuse superficial mycelium. The families are :

Erysiphaceae.—Obligate parasites, ascocarps more or less spherical, with appendages.

Erysiphe, Sphaerotheca, Uncinula, Microsphaera, Podosphaera.

Perisporiaceae.—Dark mycelium, ascocarps without appendages.

Meliola.

Microthyriaceae.—Flat ascocarps, ostiolate, no appendages. Conidia not known.

Microthyrium, Asterina.

Exoascales.—No complex sporophore formed. Two families.

Exoascaceae.—Obligate parasites. Asci arise below the epidermis or under the cuticle, breaking through to the surface of the host.

Exoascus (Taphrina).

Protomycetaceae.—Resting sporangia producing non-motile spores in an extruded vesicle. Protomyces.

Sub-class PYRENOMYCETES.—Asci formed usually on a hymenium lining the interior of a hollow sporophore (perithecium), which is usually flask-shaped and with a narrow mouth through which the spores escape at maturity. The perithecia are variously arranged, sometimes occurring independently on the mycelium, in others joined together into groups in compound stromata, which may be crust-like or tubercular, or erect and even stalked. Conidial forms are extremely common and varied. The four orders are :

Hypocreales.—Perithecia independent on mycelium. The families are :

Nectriaceae.—Perithecia, superficial, soft, light-coloured.

Nectria, Gibberella, Calonectria, Sphaerostilbe.

Hypocreaceae.—Perithecia more or less immersed.

Epichloe, Polystigma, Claviceps.

Sphaeriales.—Perithecia dark, carbonaceous, brittle or leathery. The principal families are :

Sordariaceae.

Sordaria, Sporomia.

Sphaeriaceae.
Rosellinia, Coleroa.
Ceratostomataceae.
Ceratostomella.
Mycosphaerellaceae.
Mycosphaerella, Stigmatea, Guignardia.
Pleosporaceae.
Pleospora, Pyrenophora, Venturia, Leptosphaeria, Didymella, Gibbellina, Physalospora, Ophiobolus.
Gnomoniaceae.
Gnomonia, Glomerella.
Valsaceae.
Valsa, Diaporthe.
Xylariaceae.
Ustulina, Xylaria.

Dothidiales.—Perithecia sub-epidermal, indefinite, more or less merging into the stromatic tissue. One family.
Dothidiaceae.
Dothidella, Plowrightia, Phyllachora.

Laboulbeniales.—A peculiar, isolated group of fungi, ectoparasitic on the softer parts of insects.

Sub-class DISCOMYCETES.—Asci formed on a hymenium exposed on the surface of the apothecium. The latter is usually cup-shaped with incurved margins which unfold as it matures, exposing the hymenium. Compound stromata are rare, but in some cases, as in the edible morels, the sporophore has a distinct stalk and the hymenium lines the outer convoluted surface of the fertile head, which may be of considerable size. In the edible truffles (*Tuberaceae*) the sporophore is an underground tuberous body with one or more internal chambers, or a series of labyrinthiform passages, lined with the hymenium. The chief orders are :

Pezizales.—Asci exposed at maturity. The principal families containing plant parasites are :
Mollisiaceae.—Apothecia becoming erumpent from host tissue at maturity, often stalked.
Pseudopeziza, Dasyscypha, Sclerotinia (conidial forms known as Botrytis or Molinia).

Phacidiales.—Asci immersed in the substratum.
Phacidiaceae. Apothecia grouped in black stromata. Rhytisma.

Hysteriales.—Apothecia elongated, opening by a longitudinal slit.
Hypodermataceae.—Apothecia, black, sub-epidermal. Lophodermium.

The BASIDIOMYCETES include a very large number of the higher fungi, representing the most diverse types. All, however, have the character that at some period in their life-history, following the completion of a sexual act, spores are formed on sterigmata produced from special, fertile cells, the basidia. They are divided into three sub-classes : HEMIBASIDIAE, PROTOBASIDIAE, and EUBASIDIAE.

The HEMIBASIDIAE include only one order, the *Ustilaginales* or smuts, parasites which attack grasses and other plants and produce spores, as a rule, only in certain organs of their hosts, generally the flowers. Those spores usually form a dense black dust,

from which the name 'smuts' is taken. On germination they give out a promycelium or short germ-tube, which is a transitional form between the ordinary type of germ-tube and the basidium of the other two sub-classes. It is either transversely septate or aseptate, and each cell bears a variable number of sporidia, on sterigmata, or the sporidia may be sessile. On germination, these may bud off secondary spores. The vegetative mycelium is filamentous, and there is no formation of compact tissues except sometimes in the spore beds. There are two families in the *Ustilaginales*:

Ustilaginaceae.—Basidia transversely septated into four cells.
Ustilago, Sphacelotheca.

Tilletiaceae.—Basidia continuous or one septate.
Tilletia, Urocystis, Entyloma.

The PROTOBASIDIAE differ from the HEMIBASIDIAE chiefly in having a more regular basidium and a definite number of sporidia, each cell of the, usually, 4-celled basidium producing a single sporidium on the end of its sterigma. They are divided into three orders:

Uredinales.—The rusts. True parasites of green plants. Spores produced in cup-, or flask-shaped, or flat spore beds and of five different kinds: spermatia, aecidiospores, uredospores, teleutospores, and sporidia. One or more of these may be absent. On germination, the teleutospore produces a promycelium like that of the smuts, but more strictly limited in growth. This represents the basidium, and from each of its (usually four) cells a single sporidium is produced on a sterigma. The mycelium is filamentous, except where it condenses to form the spore beds. The families are:

Pucciniaceae.—Teleutospores stalked.
Puccinia, Uromyces, Phragmidium, Triphragmium, Gymnosporangium, Hemileia.

Cronartiaceae.—Teleutospores closely packed together into crusts as in Chrysomyxa, or into a cylindrical body, as in Cronartium.

Melampsoraceae.—Teleutospores sessile, compacted into dark-coloured crusts.
Melampsora, Melampsorella.

Coleosporiaceae.—Teleutospore itself becomes a basidium, from each of which a sporidium develops on a sterigma.
Coleosporium.

Endophyllaceae.—Only teleutospores formed, in chains, resembling aecidiospores.
Endophyllum.

Tremellales.—Saprophytes with the hyphae united into masses, usually gelatinous when moist. The basidia are produced generally on fertile hyphae joined into a hymenium, which may be either superficial or enclosed by a protective covering of sterile hyphae. They are vertically septate, usually into four cells, each of which produces a single basidiospore. On germination, conidia may be produced almost immediately ('secondary spores') or they may be formed on the mycelium at a later stage.
Tremella, Helicobasidium.

Auriculariales.—As for *Tremellales*, but the basidia are transversely septated.
Auricularia (Hirneola).

The EUBASIDIAE include the mushrooms, puff-balls, and most of the larger fungi. The hyphae frequently unite to form large conspicuous masses, particularly in the sporophores, but also in sclerotia and rhizomorphs. The basidia are generally arranged close together into a hymenium and are aseptate, with a definite number of basidiospores, usually four, borne on sterigmata. Conidia are found in some cases. They include two orders :

Hymenomycetales.—Saprophytes or parasites. Basidia forming a hymenium, which is exposed from the first or, at least, before the spores are ripe. The principal families are :

Thelephoraceae.—Hymenium unilateral, spread over a smooth or corrugated surface.

Corticium, Stereum.

Exobasidiaceae.—Hymenium discontinuous, basidia cylindrical ; parasitic in leaves and stems.

Exobasidium.

Agaricaceae.—Hymenium spread over gills.

Psalliota, Armillaria, Lenzites, Clitocybe, Marasmius.

Polyporaceae.—Hymenium lining pores.

Polyporus, Ganoderma, Fomes, Merulius, Poria, Daedalea, Fistulina, Polystictus, Coniophora, Trametes.

Gasteromycetales.—Chiefly saprophytes. Basidia forming a hymenium within a soft gelatinous mass or gleba, which is enclosed in the sporophore by a special covering of sterile hyphae until after the spores are mature.

The DEUTEROMYCETES comprise a large number of conidium-bearing fungi, some of which have been proved to be merely stages in the life-history of ASCOMYCETES, more rarely BASIDIOMYCETES, or very occasionally PHYCOMYCETES. The vast majority, however, appear to exist habitually as independent individuals, having probably lost any other method of reproduction than that by conidia. They are entirely devoid of sexual reproduction or sporangium formation, and are usually of minute size. Many are parasitic. They are divided into three orders :

Sphaeropsidales.—Conidia borne on a hymenium, enclosed in a round, flask-shaped, or flattened sporophore (pycnidium). The conidia are set free through a narrow pore or a slit. The genera include :

Phoma, Phyllosticta, Phomopsis, Polyopeus, Cytospora, Coniothyrium, Ascochyta, Diplodina, Diplodia, Septoria, Leptothyrium, Heteropatella, Rhizosphaera.

Melanconiales.—Conidia borne on crowded conidiophores arising from an immersed stromatic base, in a cavity, of which the outer wall is formed by the tissues of the host (acervulus). The genera include :

Gloeosporium, Colletotrichum, Polyspora, Marssonina, Pseudodiscosia, Pestalozzia, Cylindrosporium, Septogloeum.

Hyphomycetales.—Pycnidia and acervuli absent ; conidiophores superficial, sometimes produced from simple free branches of the mycelium, or arising from an erect bundle of hyphae united together into a strand, or from a wart-like superficial cushion of matted hyphae or from stromatic pseudoparenchyma. The genera include :

Oospora, Cephalosporium, Sporotrichum, Verticillium, Mycogone, Ramularia, Cercosporella, Thielaviopsis, Fusicladium, Cladosporium, Clasterosporium, Heterosporium, Spondylocladium, Macrosporium, Alternaria, Cercospora, Graphium, Fusarium, Helminthosporium, Meria.

'*Mycelia sterilia*.'—To include forms in which no spores have so far been found. May include stages of Ascomycetes, Basidiomycetes, or other Fungi Imperfecti.—Sclerotium, Rhizoctonia.

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PART II

SELECTED DISEASES

Chapter X

DISEASES OF CEREALS

Black Rust or Stem Rust of Wheat and Other Cereals, *Puccinia graminis* Pers.

BLACK rust is believed to date back to very early times, and is present in all the wheat areas of the world.

Wheat is attacked by other rusts besides this one caused by *Puccinia graminis*. These are the yellow rust, due to *Puccinia glumarum*, and the brown rust, due to *Puccinia triticina*. Two, or all three of the rusts may sometimes be found together in the same crop. Of the three, yellow rust is, in some years, the most abundant and severe in Britain. Black rust is fairly common in south-west Wales, but elsewhere its attacks are usually light, though epidemics sometimes occur [23 b]. Losses caused by the rust in America are enormous. It is recorded that in the three North Central States of Minnesota, North, and South Dakota, and in Western Canada, the losses in these two areas in one year amounted to 180 and 110 million bushels respectively; and in Manitoba and Saskatchewan, during 1925 to 1935, the average annual loss was over $35\frac{1}{2}$ million bushels (11 per cent. of the possible yield), a monetary loss estimated at 30,784,000 dollars [8, 24].

Black rust appears on wheat rather late in the season, and the crop usually suffers little injury. The final effect, though the disease does not directly attack it, is to reduce the size of the grain, preventing it from filling out, and causing it to shrivel. Early symptoms are visible as elongated brown pustules on the stem, leaf stalks, and leaves (Fig. 179), but the part usually affected first and most severely is the stem, hence the name, stem rust, is often applied to black rust to distinguish it from yellow rust and brown rust, which are found chiefly on the leaves. These early pustules, which consist of uredosori, are often one-quarter of an inch or more long, and frequently join together in stripes. They arise from a mycelium under the epidermis, and when the latter is broken through from pressure of the spores below, dense masses of brown, powdery uredospores are released and dispersed by



FIG. 179.—Black or stem rust of cereals (*Puccinia graminis*). The teleutospore stage on oat (photo by Foister & Noble)

wind throughout the crop. They infect only the cereal host. Later on, the pustules assume a darker colour, because the same mycelium which gave rise to uredospores now develops the darker teleutospores, but sori of teleutospores may also break out anew, in the vicinity of the converted uredosori, and like them, break through the epidermis. The dark teleutospores are more firmly attached in their beds than the uredospores, and their stalks are more rigid and thicker; they are essentially resting spores, adapted for over-wintering on straw or stubble; they are not capable of reinfecting the cereal host, and serve only to infect the alternate host, the barberry.

Puccinia graminis requires two distinct hosts in order to carry through its complete life-cycle, and is therefore heteroecious. The uredospore and teleutospore stages occur on the cereal or other graminaceous host, for the rust also attacks oat, barley, and rye, as well as a large number of grasses. The life-history is completed on a second host, the common barberry (*Berberis vulgaris*), on which the aecidiospore stage is developed [26]. Other species of barberry and the closely related *Mahonia* are also capable of infection, but the common barberry is the only host of practical significance in the spread of this disease. Though indistinguishable in appearance, different forms or races of this fungus attack different cereals or grasses, and as already indicated in Chapter II, one race will infect wheat and barley, but not oat; the one that attacks oat fails to infect wheat and barley; and the race that is found on rye can infect barley, but not wheat and oat. On account of their selection of a particular host for attack, these are called *specialised races* of *P. graminis* [29]. Those on the cereals are separated by the addition of the generic name of the particular cereal attacked, thus *P. graminis tritici*, for the race on wheat, *P. graminis avenae*, for the one on oat, and the race which attacks rye and barley is called *P. graminis secalis*; moreover, each of these specialised races claims a number of hosts among the grasses. The existence of a large number of *physiologic races* of *P. graminis* on its various hosts and the means whereby they have been determined have already been discussed in Chapter II (p. 89); so far, over 200 races of *P. graminis tritici* have been recorded, mainly

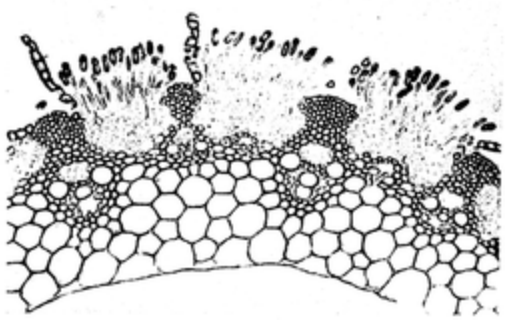


FIG. 180.—*Puccinia graminis*. Cross-section of wheat stem showing uredosori (× approx. 60) (photo by Brown; by permission of Craigie, *Dom. Can. Dept. Agric., Frmrs'. Bull.* 84)

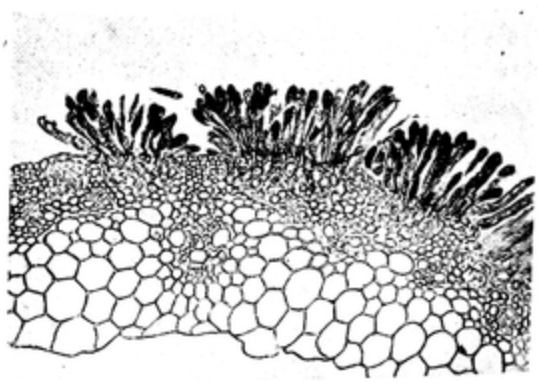


FIG. 181.—*Puccinia graminis*. Cross-section of wheat stem showing teleutosori (× approx. 60) (photo by A. M. Brown; by permission of Craigie, *Dom. Can. Dept. Agric., Frmrs'. Bull.* 84)

in the United States, of which only comparatively few have been found in Britain.

A transverse section of an uredosorus (Fig. 180) shows the mycelium to be intercellular, the hyphae, about 3·5 μ in diameter, sending into the host cells, small round or branched haustoria, without causing any apparent ill-effects on the cytoplasmic contents. *P. graminis*, like all rust fungi, is an obligate parasite, thriving only on the living tissues of its host. The uredospores (Fig. 182 A) are binucleate cells, developed singly from vertical hyphae, likewise binucleate, arising from a bed of mycelium, the cells of which are also binucleate (dikaryophytic). The uredospores are oval, 25 to 30 by 17 to 20 μ, brown, with a thick echinulose wall having four germ-pores situated equidistant in an equatorial plane (Fig. 64). These spores must have a film of water on the host before they can germinate ; a moist atmosphere is not enough [4]. Penetration, by a single germ-tube, is through a stoma over which an appressorium is formed, and from which a narrow hypha arises to invade the sub-stomatal space where it soon expands into a vesicle. From the latter an infection hypha emerges, and having penetrated a mesophyll cell and established within it its first haustorium, the hypha proceeds to develop a branching mycelium which extends in an intercellular manner, frequently sending haustoria into the cells with which it comes into contact. From such an initial infection an uredosorus with ripe spores becomes established in 8 to 14 days. With dissemination of the uredospores throughout the crop, fresh uredosori continue to be formed during the summer, but they usually cease to develop before the wheat begins to change colour and ripen. As already stated in Chapter II, uredospores of *P. graminis* are not adapted for over-wintering in colder climates [21], but they may often survive during mild winters.

The teleutospores follow the uredospores on the same or similar dikaryophytic mycelium (Fig. 181) ; the two kinds of spores may frequently be found together in the same sorus. A teleutospore (Fig. 182 B) consists of two thick-walled, smooth, superimposed cells, the top cell being rounded or blunted and thickened at the apex ; the bicellular spore is dark brown (black in the mass), 40 to 60 by 15 to 20 μ ; each constituent cell is furnished with a germ-pore, situated apically in the top cell, and just below the septum in the lower cell. Each cell during development possesses a pair of nuclei, contributed by the dikaryophytic mycelium, but when the teleutospore turns brown and matures in its sorus, the paired nuclei in each cell fuse, the process being considered to be one of deferred sexual fusion, and the ripe teleutospore represents the diploid phase in the life-history of the rust fungus, being in fact a zygote. The teleutospores do not germinate forthwith, but undergo a period of rest, and being adapted for over-wintering, remain dormant on stubble or straw for several months. They are known to be viable for at least 18 months [5], but according to some [16], they may be induced to break their dormancy by such means as freezing, or by alternately wetting and drying, but others have failed to cut short the natural period of rest [5]. Teleutospores serve, indirectly, to convey the disease to the alternate host, the barberry. In some countries, they seem to have lost the power of germination ; for instance,

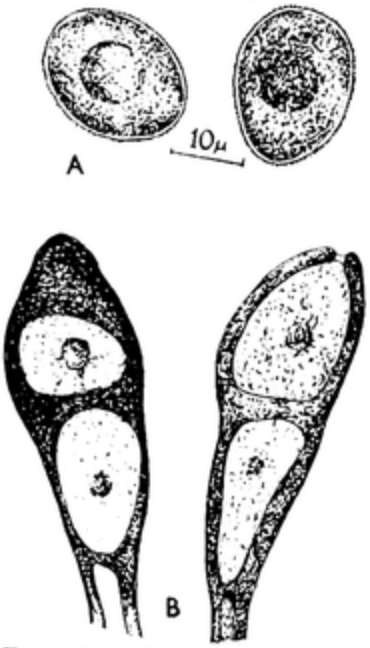


FIG. 182.—*Puccinia graminis*. *A*, uredospores showing an oil body. *B*, teleutospores with nuclei

in the plains of India [22], where barberry does not grow, and in Nebraska [28], where few barberry bushes occur south of Northern Kansas, the teleutospores do not germinate, and whether this failure is due merely to non-viability, since the teleutospores are not adapted to withstand temperatures above 26° C. [19], or to other factors inherent in races of the fungus, is not clearly known. In some localities again, where the crops ripen early, the teleutospores are developed soon enough to experience summer temperatures sufficiently high to kill them, and so the barberry in such places escapes infection.

Like the uredospores, the teleutospores must be wetted before they can germinate, the most favourable temperature for germination being 19° to 21° C. [5]. Either or both cells of a teleutospore may give rise to a stout germ-tube or promycelium (basidium), during the formation of which the diploid nucleus undergoes a reduction division, and the four nuclei which are ultimately formed in the promycelium are, therefore, haploid (Fig. 64 D). Following septation, the promycelium now consists of four uninucleate cells, each of which produces a narrow sterigma terminating in a swelling and into which the nucleus with the cytoplasm passes, to form still another type of spore, the sporidium or basidiospore. Under natural conditions teleutospore germination takes place on the wetted straw or stubble which has over-wintered on the ground, and the sporidia are carried by wind to the barberry plants. It is only the sporidia that are normally capable of infecting the second host [8 a, 26]. The process may be successfully performed by tying a truss of rusted straw to a barberry bush, the straw being wetted twice daily [5].

The sporidia germinate freely on the barberry in the presence of moisture, and infection may take place during the day or night but is favoured mostly by daylight. Penetration by the germ-tubes put forth by the sporidia is direct, through the cuticle [33]. The leaves, stems, spines, petioles, flower-sepals, and even the berries of the barberry may be attacked by the sporidia, and on all these parts, red spots, which herald the formation of the aecidial stage in the reproduction of the rust, may be developed. It is the expanded leaves, however, that catch most of the blown sporidia, and they are susceptible to infection only for about 12 days or so after unfolding [5], for soon after that period the leaves become more resistant, probably by virtue of developing a thicker cuticle [23 a]. Following penetration of the leaf on the upper surface, the mycelium becomes established in the palisade mesophyll, and while some hyphae may enter some of the host cells by means of short, beak-like processes to produce haustoria, other hyphae, which are broader, remain intercellular and increase by branching; the haustoria may be simple and spherical, or develop into 3- or 4-celled hyphae, and at a few places may appear as an intracellular mycelium, but the fungus leaves the cells almost directly to occupy the intercellular spaces of the mesophyll again. It is important to note, in view of what is stated below in relation to the sexual phase of *P. graminis*, which takes place in these infections on the barberry, that several of these infections from separate sporidia may be found close together, even within a range of two or three cells of the epidermis of the host.

Two types of fructifications, spermagonia and aecidia, which may be considered as the perfect, sexual stage in the life-history of the rust, follow upon sporidial infections on the barberry leaf (Figs. 67, 183 B). The spermagonia (or pycnia) are produced on the upper surface, and these are followed at more or less opposite places towards the lower surface of the leaf by one or more aecidia. Usually the aecidia are very numerous and aggregated close together to form dense clusters, the name 'cluster cups' being frequently applied to these fructifications, which break out at the lower surface of the leaf (Fig. 184).

The spermagonia are flask-shaped conceptacles lined with very narrow 'sper-

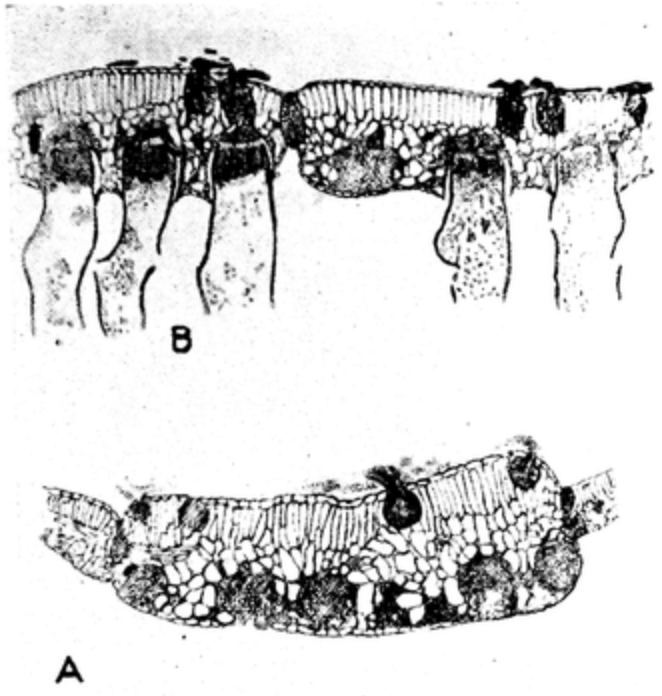


FIG. 183.—Sexuality in *Puccinia graminis*. *A*, section of barberry leaf showing a sterile infection; note, spermagonia still in active discharge on upper surface; at lower surface, numerous unfertilised aecidial primordia. *B*, leaf section showing at upper surface defunct spermagonia (fertilisation having been effected); at lower surface, five fully matured aecidia discharging aecidiospores; note at the centre, an unfertilised primordium (by permission of Ruth Allen, *J. Agric. Res.*)

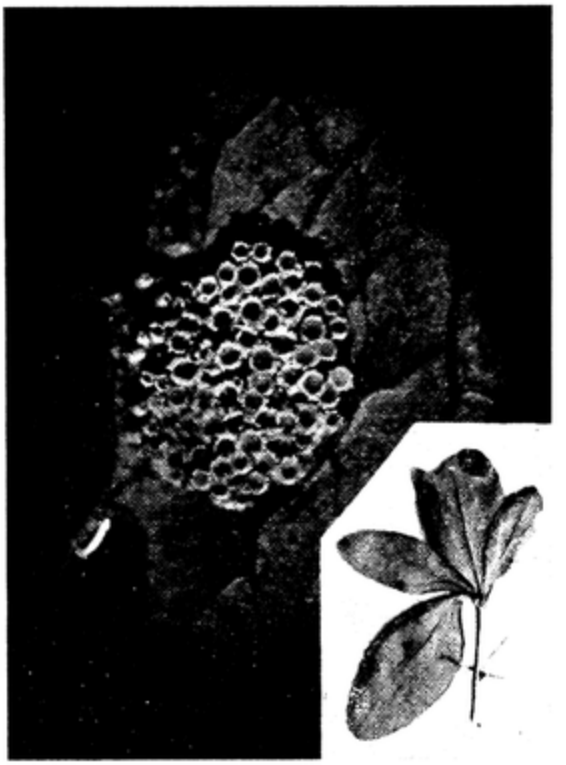


FIG. 184.—*Puccinia graminis*. Cluster cups; aecidia on the under side of a leaf of barberry (× approx. 8) (photo by permission of J. H. Craigie, *Dom. Can. Dept. Agric., Frmrs'. Bull.* 84). Inset, aecidial pustule on barberry leaf (× ½) (photo by Foister & Noble)

matial hyphae ' which converge towards the narrow neck opening out into a small ostiole at the epidermis (Figs. 59, 185). These slender hyphae cut off countless minute spores, the ' spermatia ' (pycnospores) which are exuded at the ostiole in a copious supply of sweetish ' nectar ' or ' honey dew '. As already stated in Chapter I (on reproduction of fungi), these minute spermatia are considered to fulfil the function of male fertilising cells. Spermatia have, however, been observed by some to germinate feebly by the production of short germ-tubes, and they have also been found to copulate by means of their germ-tubes, but the further history of these spermatial fusions has not been elucidated.

P. graminis is heterothallic, the four sporidia of a basidium being of opposite ' sign ', two ' + ' and two ' − ' (1, 2, 6, 7). Infections performed with sporidia of either sign give fully developed spermagonia, but only the primordia of aecidia, in the barberry leaf (Fig. 183 A). After one or more spermagonia have been formed below the upper epidermis, the same mycelium that gave rise to them traverses the mesophyll, and in one or more positions somewhat closer to the lower epidermis than the upper, a loose bed of hyphae consisting of uninucleate cells is laid down in preparation for the formation of an aecidium. But as long as an aecidial primordium remains throughout uninucleate, its development goes



FIG. 185.—*Puccinia graminis.* A thick section of a spermagonium (pycnium) on barberry leaf; note the two types of emergent hyphae, one kind being the receptive hyphae for accommodation of spermatia (pycnospores) (× approx. 350) (by permission of Craigie, *Dom. Can. Dept. Agric., Frmrs'. Bull.* 84)



FIG. 186.—*Puccinia graminis.* A compound pustule on a barberry leaf. An infection by a + sporidium has fused with an infection by a − sporidium, fertile aecidia being produced (× approx. 2·7) (photo by permission of Craigie, *Dom. Can. Dept. Agric., Frmrs'. Bull.* 84)

no further, for it is still haploid, the product of either a + or a − sporidium [12, 13]. For complete development of the primordium, a dikaryophytic condition must be established within its mycelium, and apparently there are various ways by which this can be brought about. The initiation of a binucleate condition in the mycelium of the rusts constitutes the first stage in the sexual reproduction of these fungi, and occurs when two or more sporidia of opposite ' sign ', + and −, germinate on the host close together (Fig. 186). As indicated in Chapter I, the process may be effected by the fertilising action of the spermatia on certain hyphae (which have been interpreted as ' trichogynes ', ' periphyses ', or ' paraphyses '), which emerge to the leaf surface close to, or even through the ostiole of, a spermagonium, and presumably connected to the aecidial primordium (Figs. 66, 185). In this way, ' male ' nuclei from the spermatia are believed to pass into ' female ' cells within the primordium, rendering them binucleate. Such a binucleate condition may also be brought about, at least in some of the rusts, by the mere intermingling and cell fusion of + and − mycelia, in the aecidial primordium. By either way, ' fertilisation ' is said to be attained when certain cells of the primordia come to contain ' paired nuclei ', one nucleus of a pair being descended from that of a + sporidium, and its mate from that of a − sporidium. These binucleate cells may be found more or less towards the inner boundary or base of the primordium, and with further development a well-defined layer or hymenium of female or ' fertile ' cells is formed. These cells are somewhat rectangular in shape, with the

long axis at right angles to the leaf epidermis; they are all binucleate. Some cells of the primordium may still be found uninucleate but they take no further part in the general development of the aecidium and gradually get crushed out. These sterile cells form, in fact, a kind of 'buffer' between the leaf epidermis and the fertile cells. A number of the fertile cells eventually form a well-defined group of binucleate cells, all in close lateral contact, and as they divide transversely, their nuclei meanwhile dividing conjugately, each cuts off in the direction of the lower epidermis, a binucleate aecidiospore mother-cell which, in turn, divides to form a small, flat interstitial cell, and a larger cell which is the true aecidiospore; both are binucleate (Fig. 65). Meanwhile all the developing spores are being pushed forward because each fertile cell repeats these divisions several times to form a chain of aecidiospores and interstitial cells (Fig. 67). Thus by the simultaneous development of all the fertile cells at the base of the aecidium, a cylindrical, brush-shaped mass of spores and interstitial cells arises in the host leaf, ready to break out at the lower epidermis. The outermost spore chains, forming the periphery of the cylindrical group, are sterile; they remain permanently coherent but gradually lose contact on their inner side with the fertile spore chains within, all of which tend to separate from each other in preparation for dispersal of the aecidiospores. The outer coherent cells thus form a protective cover or 'peridium' to the spore chains, and the peridium is closed over the top of the aecidium by a coherent layer of similar sterile cells. When the latter are forced outwards by the general expansion of the aecidium, the lower epidermis of the leaf is broken and this is followed by the breaking of the peridium itself, which is now bell-shaped, and it becomes much torn around the opening, where it appears like a thin paper bag with a white fringe around the now exposed orange-coloured mass of aecidiospores. The spores are readily dispersed by wind, and when quite free are spherical, but while still in the cup, especially if low down, are hexagonal in shape from the mutual pressure of the spore chains. Ripe aecidiospores are binucleate and measure from 14 to 26 μ in diameter, and have about six germ-pores in the wall (Fig. 67). These spores serve to return the rust to the cereal or other graminaceous host and cannot infect the host which produced them.

The aecidiospores germinate on the wheat in the same manner as uredospores and, like them, their germ-tubes gain access to the host tissues through stomata. The spores, being binucleate, are responsible for the establishment of a dikaryophytic mycelium within the cereal host, and we have seen how this mycelium gives rise first to uredosori of binucleate uredospores, and later to sori of teleutospores which are likewise binucleate when young, but uninucleate when mature. In the ripe teleutospores, the two nuclei, descendants of those which paired in the fertile cells (and presumably descendants of + and − sporidia) at the base of the aecidium on the barberry, fuse together. The ripe teleutospore, therefore, represents the diploid, sporophytic zygote in the life-history of the rust.

The incidence of black rust and the development of epidemics of the disease are influenced by numerous factors. In the first instance there must obviously be an abundant supply of spores for infection. We have seen that the absence of the alternate host, the barberry, makes little difference in countries of warmer climate, since the rust can survive from year to year by means of uredospores

and even in colder regions uredospores can be carried in by wind from warmer latitudes. Though these spores may often be found adhering to the grain, there is little risk of infection from this source, since the spores perish before the grain is sown; and the existence of the teleutospores on infected straw that may be returned to the soil in manure does not encourage infection, for teleutospores are innocuous to the cereal host.

Temperature plays a very important part in the incidence of rust. As already mentioned, uredospores are intolerant of very low winter temperatures, and low temperatures at uredospore infections tend to lengthen the incubation period; thus, about a week longer is required from the time of infection for the formation of uredosori on plants held at 10° than at 20° C., and much longer at 0° or 1° C. But these considerations of temperature have by no means a general application, for there are some physiologic races of *P. graminis tritici* which differ in their ability to produce uredosori even at comparatively low temperatures [23]. Again, low temperatures of 0° to 1° C. stimulate the production of teleutospores, which also germinate sooner and more abundantly than those formed at somewhat higher temperatures, but here, again, there are considerable differences observed in the rates of teleutospores formation among different races of the pathogen; some races form teleutospores quite soon after the uredospores, but other races require a longer period [16]. Somewhat higher temperatures, of 12° to 21° C. are more favourable for infection of the barberry, than low temperatures, but the latter are not always inhibitive to infection; thus, when infected bushes were kept at 0° C. for three weeks, spermagonia were developed when the temperature was raised to 18° or 20° C., and freezing destroyed the barberries before the rust fungus was killed [5]. It is clear, therefore, that since physiologic races of *P. graminis* are peculiarly sensitive to changes of temperature, it is necessary when evaluating these races in respect of their effects on the differential standard hosts (see Chapter II, p. 91) to carry out the procedure under equable conditions of the environment, and the maintenance of a constant temperature is one of the most important considerations. A temperature of 65° F. (18° C.), in conjunction with a moderate light intensity such as is experienced during spring or autumn [15], is generally agreed as being satisfactory for the purpose [25].

We have seen that infections with rust spores are only possible when the host plants are wet, and even a saturated atmosphere does not give a sufficiently humid condition. The germ-tubes require a film of water for penetration, but once this is accomplished a high humidity is not so important for parasitism of the host [8]. Taking the factors of temperature and humidity together, rust on the cereal crop is severe in seasons of abundant moisture when the temperature is about 17° or 18° C. (62° or 63° F.), a combination often experienced during wet spells alternating with bright, warm periods. Continuous cool weather is a deterrent to rust, as also are dry cool, and dry hot periods, but the latter, though protective to the host, tend to hasten unduly the ripening of the crop and to affect adversely the filling of the grain [8].

With the existence of so many physiologic races of the pathogen of black rust of wheat, and of so many different strains of the host, behaving differently in their reaction to these various races, it becomes a matter of great difficulty to determine

the true nature of resistance to this disease [3, 18]. There appear to be two types of physiological resistance, one protoplasmic, and the other functional and there is also a kind of resistance which seems to be morphological [14]. Protoplasmic resistance possessed by the host appears to assert itself soon after the plant is attacked, and while a germ-tube may penetrate a resistant or immune host, even to the extent of destroying some of the host cells, the germ-tubes are themselves early killed. The resistant principle is antagonistic and lethal, but its nature is not known, nor is it clear what the nature of the physiological properties can be with which resistant or immune plants are endowed. There is, moreover, no general agreement that any differences in the morphological, or anatomical structure, or in the nature of the nutritive contents [17], of the host, have any bearing on relative resistance or susceptibility of the cereal host to rust. While it used to be thought that the amount of sclerenchyma in the host, the size, and number per unit area of the stomata, the thickness of the cuticle and of its waxy bloom, etc., were all factors in some measure controlling resistance, it is now believed that resistance to rust, as to most other diseases, is both functional and morphological, but that no generalisation can be made. Even so-called anatomical resistance may vary in one and the same plant according to the amount and relative disposition of collenchyma and of the strands of sclerenchyma around the vascular bundles in different parts of the stem [14].

The selection of varieties of wheat resistant to, or immune from, black rust would not prove to be the difficult problem it undoubtedly is, if the numerous physiologic races of the pathogen remained stable, and the reactions of the different hosts to particular races of the fungus the same in all localities. Obviously, in countries where the presence of the barberry is essential to the carriage of the disease to the cereal crops, its eradication is of the first importance [31]. Its removal would help to stabilise the known races of the parasite by depriving these races of the opportunities for hybridisation [30]. New types of wheat favoured for their resistance to black rust are, unfortunately, not always resistant to other diseases, such as the yellow rust, brown rust, bunt, and smut. Thus, Kota wheat, introduced into Canada in 1921, found immediate favour, but fell ready victim to other diseases and has now disappeared. In Saskatchewan and Manitoba, the ' durum ' wheats, in 1917 and 1918, were more resistant to rust than the ' bread ' wheats, but in subsequent years this was not maintained. The breeding of wheat in respect of resistance to black rust (or other diseases) is a long programme, requiring at least 12 to 15 years before resistance can become established in the progeny. Recent resistant varieties are Renown, Apex, and Thatcher, the last named now replacing the one-time resistant Ceres which has become susceptible to the highly prevalent ' race 56 ' of the pathogen [32a]. It is gratifying to note that these are but the first results in the introduction of new varieties of wheat which promise to show a high degree of resistance to black rust [24, 27]. In the hard red spring wheat areas of Canada and the adjacent States, the problem of black rust of wheat has now apparently been solved by the use of resistant varieties [9a, 23c].

Although fungicidal dusts, both copper and sulphur, have proved to be highly toxic to rust spores [11], their application to the prevention of rust is hardly practicable on a wide scale. As already indicated, the eradication of barberry bushes

from hedgerows and farmsteads should be systematically carried out, as there is ample evidence in Britain that black rust breaks out every year where these shrubs are common.

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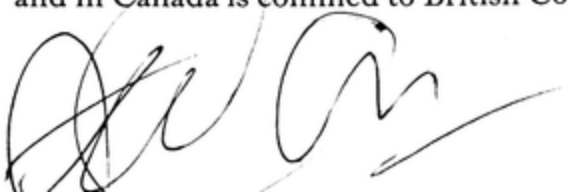
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Yellow Rust of Wheat, *Puccinia glumarum* (Schm.) Erikss & Henn.

Though yellow rust (or stripe rust) of wheat is common and more important than black rust in Britain, it rarely ruins the crop to the same extent as black rust. Throughout Europe in general, however, it appears to be the most destructive of all cereal rusts. In the United States it occurs in the Pacific and inter-mountain States [17, 29] ; and in Canada is confined to British Columbia, Alberta [31],



and west Saskatchewan [27]; it occurs also in Mexico, the Argentine [18]; south-central Asia, Russia [30], India [24, 25], China [37], and parts of Africa [8, 37].

In some years the disease is accountable for very serious losses in the field, due to destruction of the foliage, followed in some cases by sterility of spikelets or in the production of badly shrivelled grain. Though there is no substantial evidence that yellow rust is seed-borne, the germinative capacity of the grain is reduced if spores of the fungus are present in the grain-coats [19].

Puccinia glumarum occurs in Britain chiefly on wheat, is fairly common on barley, occasionally on rye, and on a large number of grasses; on some of these hosts it has become specialised, and about 14 physiologic races of *P. glumarum* are known to attack different varieties of wheat [3, 5, 8, 11, 20, 26, 27, 35, 35a].

Yellow rust appears earlier than black rust and, while in Britain and other parts winter temperatures are fatal to the uredospores of black rust, uredospores of yellow rust can tolerate very low temperatures, even many degrees of frost, and may often be found in the depths of winter [19, 24]. Thus, uredospores may be found in England on young leaves of wheat during the late autumn. The disease makes little headway, however, until the crop begins to grow actively, but by the end of March there may be quite an appreciable amount of infection which, by the end of May, has usually become well established [8]. The time of earing appears to be a factor in reaction to infection of different varieties of wheat, those ripening early being more heavily attacked than those maturing late [39]. When infected plants have reached the heading stage it is comparatively easy to pick them out, even in the absence of pustules, because infected heads are much lighter, of a yellowish green, while the healthy crop is still green in colour.

The uredosori appear as bright-yellow pustules chiefly on the leaves (Fig. 187), but in severe attacks they may also be found on the sheaths, stem, on the spikelets, covering the glumes, pales, and even the grain. Following infection, the green colour of the leaves first of all fades in long streaks, and later, as small pustules of uredospores arise along them, a distinct yellow-striped effect is imparted to the leaves. The uredosori are sub-epidermal and remain covered for a much longer time than in the other rusts, but when they finally break through, the yellow uredospores are shed and dispersed as usual by wind. They are spherical to ovate in shape, binucleate, very variable in size; from 23 to 35 by 20 to 35 μ; the spore wall is colourless, minutely echinu-

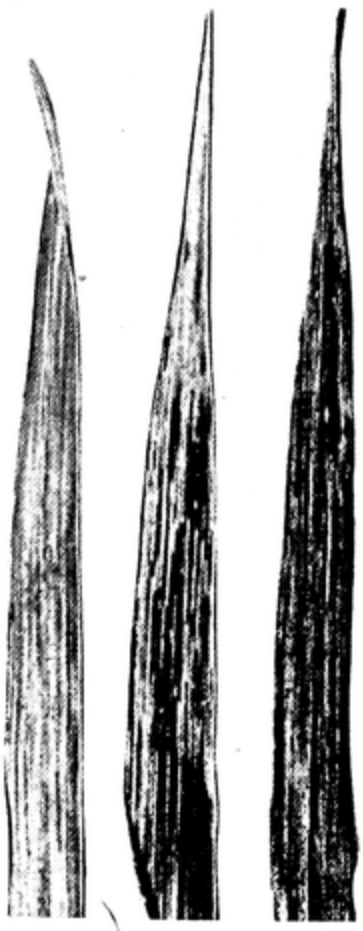


FIG. 187.—Yellow rust (*Puccinia glumarum*). On leaves of wheat (photo by Foister & Noble)

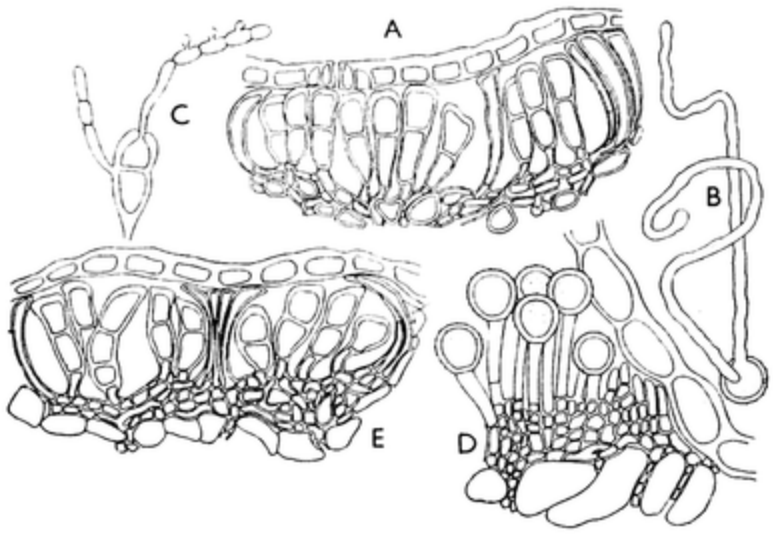


FIG. 188.—*Puccinia glumarum*. *A*, section through a teleutosorus (×240). *B*, germination of uredospore (×240). *C*, germination of teleutospore (×198). *Puccinia triticina*. *D*, section through a uredosorus. *E*, through a teleutosorus, showing 1- to 3-celled spores, and paraphyses (×240) (Fig. *C* after Jaczewski)

late, and may possess from 6 to 16 germ-pores. On the glumes the uredosori are in rows parallel with the veins and may even be found on the awns. When dehiscence takes place the uredosori on the glumes mostly open on the inner side, upon the pale, so that the spores collect in the narrow crevices among the floral parts [1]. What possible function these trapped spores can have is not known, for in such a position they cannot be wind-dispersed, and though uredospores are often found embedded even below the pericarp of the grain itself, there is as yet no confirmatory evidence that, on germination, spores from these sori may be carried up on the young shoot and, if still living, might start an attack of rust.

The teleutosori appear later, towards the maturity of the host, on the leaf sheaths, glumes, more rarely on the leaf blade, but always abundantly on the sheaths. Though they may often be found along the edges of old uredosori, it is very infrequent that teleutospores and uredospores occur together in the same sorus, as is often the case in black rust. The teleutosori are compact, dull black spots, arranged in rows like the uredosori. They do not, however, as in black rust, break through the epidermis at all, but remain as flat black crusts (Fig. 188). The teleutospores are dark brown and flattened at the top in contact with the epidermis ; they are two-celled, from 35 to 63 by 12 to 20 μ, and are interspersed with brown, unicellular paraphyses. When released they are capable of immediate germination, thus requiring no period of rest, but apparently they serve no purpose, for in the life-history of *P. glumarum* there is no intervention of a second host such as the barberry for *P. graminis* nor are the sporidia capable of infecting wheat or any other cereal or grass, so far as is known. It is probable that the fungus was originally heteroecious but that the aecidial stage, becoming of little value, has been lost. But the frequency with which *P. glumarum* gives rise to new physiologic races is strong presumptive evidence that an aecidial stage exists and on which hybridisation of known races can occur, but no aecidia-bearing host for this rust has so far been discovered [14]. Unless the new races arise by mutation it is difficult to account for them in the absence of an alternate host [12].

In most places *P. glumarum* is believed to over-winter in the form of uredospores which, as above stated, are capable of withstanding very low temperatures [24, 31, 32]. There is evidence, too, that yellow rust can survive the winter as mycelium in the host leaf, and if the infected host can live through the winter, the mycelium produces fresh sori of uredospores in the spring [1]. It is probable that resting mycelium is capable of survival in various wild-grass hosts of this

rust, such as couch and cocksfoot, and may persist during dry summer months in the tissues of these hosts, under climatic conditions which the uredospores themselves could not tolerate. Possibly some, at least, of the grass hosts harbour the same races of the fungus which attack wheat and other cereal hosts of this rust.

When the uredospores germinate, as on the wet surface of a leaf, the germ-tube forms a feeble appressorium over a stoma before the entering hypha swells up in the sub-stomatal space to form a rather firm, thick-walled vesicle. From the latter, one or more infection hyphae proceed to ramify within the mesophyll, establishing in these cells, here and there, uninucleate club-shaped, or sometimes branched, haustoria which are almost invariably to be found in contact with the nuclei of the host cells. Other hyphae emanating from the sub-stomatal vesicles, with little or no branching and without forming haustoria, run in a longitudinal manner under the epidermis, from one stomatal cavity to another, in the fashion of 'runner' hyphae, resulting in a continuous band of infection which may extend the full length of the leaf. The hyphae are very thick, 10 to 19 μ across, and may remain for a while unseptate or coenocytic, but later, with gradual development of cross-walls, short multinucleate segments are established, and as segmentation progresses further in both the ' runners ' and the short branching hyphae, there is a gradual approach to a binucleate condition of the mycelium, especially in groups of hyphae which are seen to collect beneath the stomata. These groups consist of narrow hyphae 3·5 to 6 μ in diameter and develop into uredosori. A bed of binucleate cells gives rise to a layer of elongated basal cells, the stalks of the future uredospores, and several uredospores arise in succession from the same stalk; paraphyses are also formed but they are not always present [1].

P. glumarum is intolerant of high temperatures, and both spores and mycelium grow best around 11° C.; spore germination, however, is not inhibited at temperatures up to 20°, but takes longer; the maximum temperature is about 25° C., which is considerably lower compared with 30° C. for the other rusts [19, 27, 34, 36, 38]. The importance of the temperature factor in the incidence of yellow rust may be illustrated by the gradual diminution of the disease in the Canadian prairie as the more easterly parts are reached, the high day temperatures prevailing probably preventing the growth of the fungus during the summer. In Alberta, again, the disease is prevalent only in the autumn, the spread of the rust coinciding with the shortening of the day and a reduction in the day temperature [27]. The effect of light on the development of the uredospore stage showed, however, that a 6-hours' day, in comparison with that of 12 hours, increased the incubation period [4].

Weather conditions are a determining factor in the incidence of yellow rust [4]. The disease is practically absent from areas which experience low rainfall and high summer temperatures. In localised areas, however, such as river valleys and other low-lying sites, conditions may be sufficiently humid to permit the development of epidemics [3]. In general, heavy rainfall and low average temperatures are essential to the spread of yellow rust. But apparently there are some strains of wheat that can succour the disease even in dry seasons. In England, in 1919, a very susceptible variety of wheat growing in the neighbourhood of other, moderately susceptible kinds, was attacked with great intensity during a

period of drought whereas the other varieties remained perceptibly free, the reason probably being that very susceptible varieties are attacked over a wide range of temperature, the fungus being capable of making progress in the tissues of the host even during hot weather [2]; a similar experience was recorded in Italy [28], but in these instances it is probable that other factors were also incident to these outbreaks of yellow rust in summer weather. It is known that some races of the fungus have a higher optimum temperature than others, and some strains of wheat are equally susceptible at any age, at all times of the year [33].

Plants attacked severely by yellow rust suffer mostly from impoverishment of the root system. This is a result of heavy leaf infection, and since the roots of cereals in general are so dependent upon direct translocation of elaborated food from the leaves, having little stored for their immediate needs, the roots suffer badly. Moreover, active transpiration, having the effect of cooling the leaves, favours infection by reducing the temperature, and a turgid condition in the leaf cells also assists the fungus, so that the absorptive capacity of the rooting system is still further taxed and fails to keep up with the demands of the plant. If infection of the wheat plant be followed from the seedling stage (it occurs only after the primary leaf has begun to expand) it soon becomes evident that there is a general retardation of growth, and though the stunted plants may fructify, the yield of grain and straw is reduced, and both the ears and the grain are below normal size. Obviously the adverse effect of the rust on the host diminishes, the longer infection is delayed up to heading point, but even then the roots are the parts to suffer most. It is remarkable that even on the more resistant varieties of infected wheat, on which relatively few pustules appear, there is the same general trend towards root impoverishment, with reduction in weight of roots, in total dry matter, and in the yield of grain [6].

Wheat is adversely affected, in respect of susceptibility to yellow rust, by unbalanced manurial treatment. This cereal is more open to the disease if it follows legumes and root crops in the rotation, owing to excess of nitrogen in the soil and depletion of potash and phosphoric acid, both of which confer upon wheat a high degree of resistance to disease; liming also tends to check the rust [13, 16, 23]. Infection of wheat is worse in deep loam and heavy soils than in light soils.

It is reported that the effect of yellow rust in the leaves of susceptible wheats is to induce the secretion of fat from the plastids, thus preventing normal formation of starch in the affected cells, the fungus presumably diverting the fat to its own use. When the chloroplasts become disintegrated, the fungus then attacks and finally kills the host cells. It is thought that resistant varieties of wheat are capable of preventing this degeneration of the plastids and their products by the formation of phenolic compounds in the cell sap [22], but how far this factor covers all types of resistance to yellow rust is not known, for few varieties of wheat are immune from yellow rust, under divergent conditions, especially in relation to fluctuating temperatures [10]. Even in respect of one or more physiologic races of the fungus from which a particular variety of wheat is immune, resistance of the host may be only seasonal, or it may acquire its resistance with advancing age, or it may be uniformly resistant only within certain limits of temperature [28, 33].

Wheat appears to be more liable to yellow rust if simultaneously attacked by

bunt; and indeed, this coincidence has been used to test relative resistance of wheats to yellow rust; it is suggested that in testing wheat varieties for resistance to *P. glumarum* the grain should be previously bunted and, if no yellow rust appears, it is truly resistant [9, 33].

Immunity from, and susceptibility to, yellow rust are inheritable properties in wheat; they behave as unit characters [7]. The F_1 generation of an immune with a susceptible strain is susceptible, but in the selfed F_2 generation there is a 3 to 1 susceptible to immune segregation. Resistance, therefore, is a recessive character, the immune F_2 breeding true [1, 2, 8]. By hybridisation of favourable types of wheat, resistance to yellow rust has been established in many countries, but the results have no general application, and while certain varieties of wheat so produced have high resistance in some localities, they are liable to fail in other places. In Britain the variety Little Joss has been in favour for many years; and at the Welsh Plant Breeding Station, while it was found that, in general, the differences observed in the severity of the attack by yellow rust on different varieties of wheat were due, perhaps, more to the time of sowing than to variety of wheat, few varieties proved to be truly resistant, but Yeoman possessed the valuable property of combining great powers of resistance to both black and yellow rusts; Garton's Early Cone and Percival's Blue Cone are also recommended; the varieties Swedish Iron, Dutch Million, and Red-Stand-Up have also a high degree of resistance to yellow rust but are severely attacked by black rust [21].

Experiments in Germany showed that good results against yellow rust followed the sprinkling of highly susceptible plants of winter wheat with calcium cyanamide, applied at 80 to 200 kg. hectare, in the early morning [15].

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Brown Rust of Wheat, *Puccinia triticina* Erikss.

Brown rust, also called orange rust or leaf rust of wheat, occurs in all parts of Britain but is of little economic importance in this country. Under natural conditions it is confined solely to wheat, but under experimental tests successful infections were obtained on a species of Agropyron (*A. trichophorum*); and in Kansas a number of wheat *x* rye hybrids yielded to infection when inoculated with certain races of *Puccinia triticina* causing this disease [8, 14]. In England, rye and barley also yielded to infection; on rye the reaction was different from that on wheat and the spores produced on barley would not infect barley again [10]. In Indiana, in 1931, there was a reduction in yield of from 14·8 to 28·4 per cent., and approximately three-fourths of the grain losses in this region due to the rust were attributed to a reduction in the number of grains per head and the remainder to reduced weight per grain [6]. Brown rust is common wherever wheat is grown; in certain parts of North America it can be very destructive, and in 1938 an epidemic of this rust was the worst on record in the United States, attacking varieties of wheat which had long been resistant to it and doing great damage to early hard red spring wheats [17]. On some kinds of wheat brown rust is frequently found in association with black rust, but it can also attack severely other varieties of wheat which are resistant to black rust [21].

Brown rust is mainly confined to the leaves, rarely occurring on the stems and heads. Heavy infections, therefore, greatly retard photosynthesis with the result that if the disease, as in some localities, develops early, entire leaves turn yellow and wither before the plants are about a foot high [28]. Marked interference with leaf functions and a tendency to increase of transpiration consequent upon infection cause plants to take a much longer time to head out and mature. Altogether, heavy rusting of the foliage retards normal maturity and this is reflected not only in a reduction in the grade, yield, and quality of grain [22, 27], but in the yield of straw and, in particular, as in attacks of yellow rust, in a deterioration of the rooting system. The roots appear to suffer irrespective of the time of infection, and plants which reach the heading stage free from disease may become as severely affected as if they had been associated with the fungus from the seedling stage. Heads are slow to appear on heavily rusted plants, and may either emerge slowly or fail to clear the sheath at all. As the crop ripens, susceptible plants do not turn a normal yellow but assume a greyish-green colour even after their grain has set hard [16].

The uredosori of *P. triticina* break out from under the epidermis as orange or brown specks. They do not arise in rows or stripes as in yellow rust but are grouped in small clusters or irregularly scattered. The uredospores are brown and spherical, 16 to 28 μ in diameter, the wall minutely echinulate, and furnished with 7 to 10 germ-pores (Fig. 188 D). Infection by uredospores occurs through the stomata, on either side of the leaf. Over a stoma the germ-tube forms an appressorium which, in contact with the guard cells, so affects them that the penetrating hypha appears to wait for the stoma to open, penetration being effected at one end of the opening [2]; it is also suggested that the function of the overlying appressorium may be to exert pressure between the closed guard cells, thereby prising them apart to admit the infection hypha [7]. Within the sub-stomatal cavity the invading hypha expands to form a vesicle from which branching hyphae develop to invade the leaf. Sometimes two or more germ-tubes may penetrate the same stoma, and appressoria and sometimes sub-stomatal vesicles fuse together at a common seat of infection. An intercellular mycelium with haustoria is soon established, but infection has no immediate adverse effect on the cells of the mesophyll, and the cells remain alive with their plastids full of starch for about 9 or 12 days after infection [2].

P. triticina rarely forms teleutospores (Fig. 188 E). In some years they are quite plentiful, and at other times search for them is entirely unsuccessful. Some attribute their erratic appearance to vagaries of climate, while others find that their formation appears to depend on a certain stage of maturity attained by the host [24]. The sori are scattered chiefly on the underside of the leaf and on the sheaths, and, like those of yellow rust, do not break through the epidermis. The teleutospores resemble those of yellow rust in size and shape but the sorus is divided up into small groups by the interpolation of paraphyses. The spores are smooth and brown and are said to be viable for two years [24].

An alternate host on which this rust might develop its aecidial stage remained undiscovered for a long time, until 1921, when aecidia were found, in the United States [12], on the meadow rue *Thalictrum flavum*, and other species of *Thalictrum* also yielded to sporidial infections. Aecidia are, however, not of common occurrence on *Thalictrum* in nature, and so far they have not been discovered in Britain. In this country they may possibly exist on a different host, for in East Siberia they were found to develop under natural conditions on the leaves of another member of the *Ranunculaceae*, namely *Isopyrum fumarioides*, and wheat was successfully inoculated with the aecidiospores produced on this alternate host; probably the climatic conditions in Siberia are favourable to the development and normal germination of the teleutospores [5]. Moreover, three species of *Echium*, one of *Anchusa* and two of *Cynoglossum* (of which the viper's bugloss, alkanet, and hound's tongue are species of these genera, respectively, common in this country) reacted positively to infection with a certain race of *P. triticina* in Portugal [9]. The fungus is heterothallic. In the leaves of *Thalictrum* sporidial infections are followed by the establishment of an intercellular mycelium in the mesophyll in the cells of which small, unbranched haustoria are developed; spermagonia appear on both sides of the leaf and the production of spermatia is accompanied by a copious discharge of fragrant nectar; aecidia are formed towards the lower side of the thickened leaf [3].

In the absence of an aecidial host in nature, *P. triticina*, like *P. glumarum*,

apparently depends upon the survival of its uredospores to carry the disease over from season to season, and as the spores can tolerate low temperatures they can survive on host-remains in the field as well as on volunteer plants until the new crop appears. It is probable, too, that brown rust is enabled to winter as mycelium in the host leaf, and if the infected host can survive, the mycelium may produce fresh sori of uredospores to start new infections in the spring [26].

P. triticina embraces over a hundred physiologic races, some of which have probably arisen by mutation ; about eight races are known in Britain [8, 8 a, 10, 11, 13, 23, 25]. All these races of brown rust apparently produce different, and in some cases abnormal, effects on different varieties of wheat according to changes of environment, especially if exposed to extremes of temperature and variable degrees of light intensity. There appears to be an increase in resistance in normally susceptible reactions, found in many forms, associated with low light intensity and low temperature. Thus, at temperatures of 57° to 75° F., the varieties Malakoff, Norka, and Democrat developed greater susceptibility, while Carina, Brevit, and Hussar showed greater resistance to brown rust at these temperatures [19]. In general, as regards susceptible varieties of wheat, low light intensity tends to reduce susceptibility to brown rust, and susceptibility is increased as temperature, soil moisture, and light intensity are increased. An increase in protein and sugar content, at high soil humidity, appears to increase susceptibility to brown rust [23 a]. Varieties of wheat which normally exhibit resistant reactions are on the whole less sensitive to fluctuations of the environment than those showing susceptible reactions [23].

This disease is favoured by the same meteorological conditions as those which favour black rust, but brown rust can develop over a wider range of temperature, especially at temperatures below 15° C. (60° F.) [18].

Relative resistance of wheat to brown rust is closely correlated with the age of the plant. Certain varieties of wheat, when inoculated with certain races of the fungus are highly susceptible when in the seedling stage and very resistant to the same race of the parasite at heading time. Furthermore, these same varieties showed the uppermost leaves to be more resistant than the lower, older leaves, probably because in the latter the fungus finds a food more suitable to its needs and which has not as yet been formed or stored up by the younger leaves, or it may be that phenolic compounds are present in an active toxic state in the younger tissues. In certain varieties of wheat, as in Mammoth Red, the tips of the leaves were more susceptible to the rust than the younger tissues near the base [15].

Resistance to brown and other rusts is clearly a complex problem and is affected by various factors, some of which are inherent in the host and others environmental. In general, the summer wheats are more susceptible than winter wheats to brown rust [24]. In certain localities in Canada and the United States there are several valuable wheats which are resistant, such as Carina, Brevit, Webster, Hope, and others, and hybrids between some of these and other valuable, though susceptible, forms give promise of still greater resistance in the progeny [1, 4, 8, 19, 20, 21].

The following are the chief macroscopic features whereby the three rusts of wheat can be distinguished :

Black Rust (*P. graminis*)	Yellow Rust (*P. glumarum*)	Brown Rust (*P. triticina*)
Stems most severely attacked, then leaf sheaths, leaves, and ears.	Leaves most severely attacked, then leaf sheaths, stems, and ears.	Attacks leaves almost exclusively, rarely leaf sheaths, very rarely stems.
Uredosori large, elongated, coalescing and dehiscing early, breaking up large fragments of the epidermis. Colour brown, becoming darker as teleutosori are formed in the same pustule. Found on all green parts of the plant.	Uredosori small, oval, not usually joining together, bursting late and with little displacement of the epidermis. Arranged in rows or stripes. Colour brown-yellow. Found on all green parts of the plant.	Uredosori small but often longer than in yellow rust, oval or round, not usually running together, bursting with a fringe of broken epidermis around them. Never in long rows. Colour bright orange when fresh, becoming brown. Chiefly on upper side of leaf.
Teleutosori like uredosori but black. Burst rather early. Found on all green parts of the plant, but least on the leaf blades.	Teleutosori like uredosori but more flattened, dull black, and often run together. Do not burst through the epidermis. Arranged in rows. Chiefly on under surface of leaves but also on all green parts of the plant.	Teleutosori often absent; when present resemble uredosori but more flattened and dull black. Do not burst through epidermis. Chiefly on under surface of leaves, very rare elsewhere.

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Bunt or Stinking Smut of Wheat, *Tilletia caries* (D.C.) Tul. and **Tilletia foetida** (Wallr.) Liro.

Bunt or stinking smut of wheat is a much more serious disease of wheat, where it is prevalent, than the loose smut caused by *Ustilago tritici*, described below. Owing to the efficacy of seed treatment in the control of this disease, however, bunt has gradually dwindled in importance in Britain, but there has recently been a conspicuous increase and it is imperative that protective measures should not go out of practice. The disease is caused by two allied species of the lower Basidiomycetes, *Tilletia caries* (= *T. tritici*), and *Tilletia foetida* (= *T. levis*). The former is much the commoner in Britain, where the latter is seldom met with [2] or apparently absent [32a], though there are areas in the United States and elsewhere in which *T. foetida* alone is often found [21, 44]. Bunted kernels are sometimes found on wild and cultivated grasses, e.g. *Triticum*, *Agropyron*, and *Lolium*, and four other species of *Tilletia* are recorded on grasses in Britain [5a, 11, 40].



FIG. 189.—Bunt (*Tilletia caries*), and loose smut (*Ustilago tritici*), of wheat. *A*, bunt, showing broken grains liberating the spores. *B*, *C*, loose smut, showing various stages of ear disintegration. *D*, a mixture of the spores of bunt (large) and of loose smut (small) (photos *A*, *B*, *D*, by Dillon Weston; *C*, by Foister & Noble)

Losses caused by bunt are due to the replacement of the grain contents by smut spores, the grain coats being left more or less intact. Unless controlled they can be very serious; thus, it was calculated that in 1927 bunt reduced the United States wheat crop by over 28 million bushels, an average of $\frac{1}{2}$ bushel per acre harvested [19]. In Siberian Russia the average weight of healthy wheat grains of *Triticum vulgare* var. *lutescens* was 34·578 mg. as compared with 11·159 mg. in bunted grains, and the loss of nutritive substance in infected grain was 56 per cent. of that of healthy grain of the variety *ferrugineum*, and 68 per cent. for the variety *lutescens* [33a]. The disease has the effect of shortening the straw, but tillering is often increased [11, 15, 32, 39, 43]. At maturity the ears remain erect, dark or olive green, and with open pales and glumes within

which the black bunt ball is seen (Fig. 189 A). Bearded varieties have deformed awns or the latter may be absent [8]. Bunted grain (' butts ') are plumper and shorter than healthy grain. Some ears may escape infection in diseased stools, or an infected ear may bear single healthy grains. Wheat infected with bunt has been observed in Britain to show increased susceptibility to yellow rust caused by *Puccinia glumarum* [8 b] (p. 357).

A bunt ball may contain from 1 to 4 million spores united into a greasy mass which hardens on drying, and has a pronounced smell of rotten fish due to trimethylamine. The bunted grain may remain unbroken during harvesting, but at threshing is often ruptured and may contaminate clean grain at any stage from harvest to sowing [17, 20, 25]. The spores adhere to the healthy grain, and other spores remaining in the threshing machine or sacks used for storing bunted grain serve to contaminate further stocks of clean grain. There is no spread of disease in the standing crop and infection occurs only in the soil when contaminated grains germinate.

The two species *T. caries* and *T. foetida* differ chiefly in their spores, the former having a reticulate wall, the latter a smooth one. In *T. caries* the spores, 15 to 21 μ in diameter, are rounded, while *T. foetida* has more irregular spores measuring 16 to 25 μ. Both germinate in the same way by producing a stout germ-tube (promycelium) which bears a cluster of up to 24 elongated primary sporidia at its tip [13]. The fusion of these in pairs and the subsequent formation of secondary and tertiary sporidia, which are forcibly discharged by the water drop mechanism (and are held to be the true basidiospores) has already been described (p. 41).

The haploid mycelium from the basidiospores (Fig. 63) is composed of thin non-septate hyphae, which must unite with mycelia of cemplementary sex to form a septate mycelium of stouter binucleate cells before infection can result [6]. Both *T. caries* and *T. foetida* are heterothallic, so that only mycelia from two sporidia of sexually compatible strains are able to fuse and produce the full parasitic life cycle terminating in the diploid zygote [12, 13]. Fusion of paired nuclei has been observed just prior to the maturation of the smut spores, and it is a natural assumption that the fusing nuclei are descendants of those derived from two complementary haploid sporidia, although it is not possible to trace a regularly binucleate mycelium in the intervening stages.

Growth of the sporidial mycelium is poor below 5° C. but improves from 10° up to about 20°. Spore germination is good in the soil at temperatures somewhat below those (12° to 16°) that favour rapid growth of winter-wheat seedlings and considerably below the optimum (16° to 20°) for spring-wheat varieties [5, 10, 16, 28, 34]. Infection occurs over a soil-temperature range of 5° to 20° but is greatest about 10°. This is widely believed to be due to the fact that the seedling is only susceptible up to the time of emergence of the first green leaf from the coleoptile, and the longer this period of susceptibility lasts the greater the chance of successful infection. The evidence cited below, that varieties of wheat differ from one another in this response to inoculation at different soil and air temperatures may be one of the reasons why the predominant part usually assigned to the length of the pre-emergence stage of seedling growth in the initiation of infection has been questioned by some investigators [9, 14, 30].

Following upon penetration of the coleoptile the parasitic mycelium passes to

the young shoot where, in successful infections, it keeps pace with the growth of the host, causing little interference to it until the formation and development of the ears [5, 46]. It is found chiefly in the loose cells of the pith which does not become hollow in infected, as it does in healthy plants [43]. Eventually hyphae accumulate in the ovaries, the cells of which are crushed and replaced by hyphal masses. As above mentioned, it is in these hyphae that fusion of paired nuclei takes place and there is a rapid transformation of the hyphal mass into smut spores filling all the grain internal to the pericarp to constitute a bunt ball. It has been suggested that factors stimulating growth of the host more than that of the fungus are responsible for failure of the latter to reach the ears in some tillers, but this seems difficult to establish, especially as wheat varieties react differently to such factors. Thus, while Hope wheat had only 2·4 per cent. bunt when transferred from 9° to 21° C. after emergence but was 100 per cent. bunted when grown throughout at 9°, the variety Jenkin showed a high percentage of infection under both treatments [41]. That factors stimulating the growth of the host may enable it to outgrow infection seems *a priori* to be likely, and the observation that nitrogenous fertilisers may check bunt [30] may find its explanation here. The depth of planting has been considered to be important in some cases, either because deep planting may lengthen the susceptible period of the seedling or too shallow planting may bring it up into unfavourable aerial conditions for growth [7, 24, 26].

Soil moisture is another factor of importance in infection, as bunt is inhibited in very wet or very dry soils ; in one series of experiments the percentage of infection was 10·7 in a very moist soil, 55·3 in one normally moist for sowing, and 22·3 in a dry soil. High moisture induces rapid germination of the wheat while retarding spore germination on account of deficient oxygen [34, 45]. Though the spores in bunt balls may germinate after 8 years and isolated spores after 3, if kept dry, those lodged in moist soils will mostly have germinated after a few months, so that in ordinary rotations their vitality is lost before the next crop is sown. Freezing preserves the spores, however, and bunt balls are known to survive on the winter-frozen soils of western Canada and a small part of the United States. In this area winter wheat can be attacked from the soil [14a], though in the rest of North America the intermittent heat and cold soon rids the soil of contamination [30, 31]. An exception may be found in soils rich in organic matter in which the fungus can remain as a saprophyte in the sporidial stages over long periods. The relatively lower incidence of bunt in sandy soils as compared with those rich in humus may be partly due to failure to establish a saprophytic phase in the former but is also believed to be associated sometimes with the reaction of light acid soils, since the limit on the acid side for the germination of bunt-spores in soil has been found to be about pH_5 [30, 34] ; there is evidence that soil type affects the course of germination of the spores rather than resistance of the host. It is not certain whether soil infection occurs in England, and upon this point further investigation is desirable [32a].

Numerous physiologic races of both *T. caries* and *T. foetida* are known, differing in pathogenicity, size, shape and hardness of the bunt balls, degree of reticulation of the spore wall, stunting of the host, dropping of awns, partial infection of the stool, and so on [1, 5, 11, 22 *et seq.*, 36, 38b, 47]. Owing to its nuclear history the bunt

ball comprises many diploid heterozygous units which may segregate for various characters on germination [2a, 29]; new infections may tend to screen out some of these [3, 14, 31]. This is the probable explanation of the observed fact that continual passage through a given variety of wheat sometimes tends to enhance virulence, though it is also possible that quite new strains may arise as a result of hybridisation, as has been found to occur in the cereal rusts on their aecidial hosts. Breeding against bunt meets with the familiar difficulties due to multiplicity of strains of host and parasite, and when (as is usual) resistance is first evident at the post-penetration stage, it is not always easy to determine whether this is due to a direct influence of the host cells or to such indirect factors as the relative rates of growth of host and parasite [38a]. The latter is, perhaps, the more probable explanation of the fact that some varieties are resistant when sown in the spring and susceptible when sown in the autumn [7, 28, 31a, 38]. No varieties appear to be known that are resistant to bunt in all localities under all conditions. Nevertheless a number of resistant varieties are now in cultivation in Canada and the western United States [1, 21, 37].

Breeding against bunt is of relatively minor importance except when contamination comes from the soil, owing to the ease with which the usual source of infection from spores adhering to the grain can be eliminated by seed treatment. The 'pickling' of wheat-seed grain against bunt is one of the earliest examples of the use of chemical therapeutants in the control of plant diseases.

Copper sulphate was long the standard remedy for bunt and is still occasionally recommended. It is used as a solution of 1 lb. in 5 gallons water (equivalent to 2 per cent.) and is sprinkled over the grain spread on a floor, the heap being repeatedly turned over until every grain is wetted. It is then dried, being turned over again a couple of times, and is ready for sowing. An alternative is to steep the grain by immersing it completely in a non-metal vessel (e.g. a barrel) containing the solution, stirring it well for about a minute, and skimming off the bunt balls which will rise to the surface. The solution is then drained off for further use and the grain dried quickly. Steeping is preferable to sprinkling when there are many bunt balls, as the spores inside the balls are not killed by the treatment. Copper sulphate causes some damage to the grain, as the deposit on the seed injures the delicate primary shoot and reduces germination; the injury is slight if the seed is sown within 24 hours but is quite appreciable if sowing is delayed.

When formalin was introduced, it soon replaced copper sulphate as a remedy for bunt in most countries. Sprinkling with 1 lb. commercial formalin in 40 gallons water is efficient if the grain is well wetted by turning over. Steeping by complete immersion for 10 minutes has the same advantage as with copper sulphate in enabling bunt balls to be removed; it can be done in metal vessels. Whether sprinkled or steeped, the grain must be kept moist for about 2 hours after treatment by covering the heap with sacking soaked in the same solution. Formalin is convenient, very effective, and less injurious to germination than copper sulphate, though it is still advisable to sow early, within 24 hours if possible.

Dusting with dry copper carbonate was first introduced as a remedy for bunt in Australia and has found favour there and in the western part of the United States, as the dusted grain is protected from reinfection from the soil or otherwise.

It is little used in Britain, where the moister seed beds and soils reduce its special advantages. A dosage of 2 to 3 oz. per bushel is adequate, provided the compound is of high grade and finely powdered so as to secure good admixture with the grain when both are rotated some 20 times in a revolving drum or barrel [23, 33, 42]. In Nebraska the efficacy of the treatment has been enhanced by winnowing out the bunt balls by fanning prior to dusting [26].

Where cereal-seed dressing has become a routine, the use of proprietary organomercury dusts has largely supplanted all other methods of combating bunt in the more advanced countries and, as already mentioned (Chaper VII), has received official support in England.

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General :

Holton, C. S., and Heald, F. D. : *Bunt or Stinking Smut of Wheat (a world problem).* Burgess Publ. Co., Minn. U.S.A.

Loose Smut of Wheat, *Ustilago tritici* (Pers.) Rostr.

Loose smut is common wherever wheat is grown, but, in general, is not so serious as the ' stinking smut ' or bunt of wheat described above. It is not an important disease of the crop in Britain, but in certain areas in the United States it has been reported to cause greater loss than any other wheat disease [11]; in Utah, in 1917–26, its estimated toll averaged more than 10 million bushels annually [24].

There are no signs of the disease in the crop until the ' heading ' stage is reached, when infected plants are recognised by the black smutted appearance of the ears (Fig. 189 B, C). The smutted heads consist of deformed spikelets filled with black, dry, powdery masses of spores (' brand ' spores). At first covered by a thin, grey membrane, these masses, which break out usually before the head emerges from the sheath, form dense aggregations of dark olive-brown spores which have entirely replaced all the floral parts and glumes, and only the ends of the awns usually escape transformation. Ears affected with loose smut are thus much more easily detected in the crop than bunted ears in which the spores normally remain covered throughout the growth of the crop. After a gust of wind or heavy rain nothing remains of the smutted ear except the naked stalk. Ordinarily all the heads on a plant are affected, and while the smut is confined mostly to the ears, sometimes dark streaks of spore formation may occur also on the leaves and less often on the stem.

Smutted heads emerge from the leaf sheath usually about the same time as, or very often in advance of, the heads of healthy plants. In dry weather the spores are blown in clouds throughout the crop when the heads are in bloom, and during intermittent brief intervals when spikelets are open for pollination, infection of the flowers takes place. In this way infection of wheat by loose smut is different from that caused by bunt, for though bunted grains may sometimes break open in the standing crop, they normally remain intact until after harvest, when they are threshed along with the healthy grain. Though wheat plants (and the cereals in general) are usually self-pollinating, wheat is reported to possess 3 to 4 per cent.

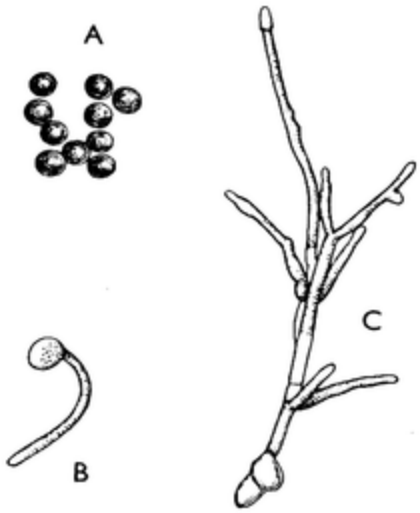


FIG. 190.—*Ustilago tritici*. *A*, the spores (×500). *B*, *C*, spore germination (×500) (after McAlpine)

'natural crossing', but this in no way implies that the self-fertilising habit of the plant interferes with the germination of smut spores on the stigmas. However, as far as is known, this is believed to be the only way by which loose smut can be conveyed to the developing grain on healthy plants. Such infected grain at harvest is, however, indistinguishable in appearance from perfectly sound grain, but when sown, systemic infection takes place, culminating in the production of smutted ears once more. This is the entire life-cycle and there is no evidence that direct infection of the seedling takes place from without, from spores or any other form that the fungus might adopt in the soil, or from spores adherent to the grain.

The olive-brown spores, a little lighter in colour on one side, are spherical, occasionally oval, finely echinulate, from 5 to 9 μ in diameter (Fig. 190). They germinate in water or nutrient solutions to form each a germ-tube which develops in the usual way to become a promycelium (basidium) of four uninucleate cells. But there is no formation of sporidia. The haploid nature of these four promycelial cells was determined after they had been induced to separate from each other at their septa by exposure to low temperatures, when the separated cells, cultured on potato dextrose agar, gave rise to colonies of different sex; these 'plus' and 'minus' strains in contact produced a distinct zone of growth quite different from the appearance of either of the haploid colonies, and in the zone hyphal fusions and hyphae with paired nuclei were found, features which were absent in colonies of the same sex [16]. When haploid cultures were used to inoculate the flowers the results were negative, but with cultures derived from compatible paired strains a high percentage of infections was obtained [2]. *U. tritici* embraces a number of physiologic races, some of which appear to predominate over others according to the locality and type of wheat attacked [8, 10, 19, 21, 31]. It is claimed that this species is not distinguishable from *U. nuda* causing loose smut of barley, except perhaps in pathogenicity, and that the two should be regarded as races, priority being given to the designation *U. tritici*. In Rumania, winter wheat was apparently attacked by only one race, while infections on summer wheat yielded three distinct races of the pathogen [20].

The spores, caught up on the protruding stigmas of the flowers, germinate on the moist stigmatic cells, to put forth their promycelial tubes, as above described, and presumably, from a dikaryophytic mycelium, infective hyphae arise which, by exploiting the intercellular spaces and pollen-tube tracks in stigmas and style, eventually reach the ovary [29]. The greatest amount of infection has been observed by some to take place during the periods of full bloom [3], but others found more infection in flowers in which the stamens were still green and immature [24]. The period during which the young ovary is liable to infection is very limited and may extend from 2 to 7 days coincident with the duration of blooming, and successful penetrations are only accomplished before the integument of the ovule

has begun to harden, which is about 10 days after blooming, penetration of the ovule usually occurring between the 7th and the 10th day. Thus, ovaries of Little Club wheat failed to develop infection 8 days after inoculation, and though they were then only $\frac{1}{3}$ of their size at maturity, they had passed the stage of susceptibility to loose smut [25]. In successful penetrations the fungus in about three weeks has reached as far as the stalk of the ovule and then proceeds to exploit the endosperm, scutellum, and the embryo. A further period of four weeks has established infection in all parts of the embryo except the root, and the scutellum is also found to harbour a considerable amount of mycelium; in the scutellum the hyphae are a little broader than in other parts, where they are about 2·5 to 3 μ in diameter, but at all times invasion occurs in an intercellular manner. The extent of fungal invasion into the various parts of the ovary varies according to the type of wheat. In some the fungus may go no farther than the pericarp, while in others all parts of the embryo, even root primordia, may contain mycelium [23]. Despite this infection the swelling of the grain is in no way impeded and the tissues remain unharmed. The fungus, becoming somewhat thicker-walled, remains dormant in the seed, and from superficial examination at harvest time, infected grain cannot be distinguished from sound grain. The fungus revives when the infected grain is sown along with the sound, the hyphae just behind the growing point of the embryo keeping pace all the while with the apical growth of the plant. The fungus invades the stem, leaf sheath, and lamina, and is present mostly in the tissues of the stem and to a greater extent in the sheath than in the lamina, in which it is sparse [15]. Finally, when the heading stage is reached, the fungus becomes active within the spikelets and, in place of the normal contents, dense masses of spores arise, as described above. Although the ultimate effect, the destruction of the grain in the ear, is the same in both bunt and loose smut of wheat, it must be borne in mind that bunt infection starts in the soil from spores adherent to the surface of the healthy grain, that is, from contaminated grains, whereas grains affected with loose smut carry the fungus from flowering to harvest; this has an important bearing on the methods for controlling these two diseases.

Some have noted in infected plants a slight hypertrophy of the tissues near the vascular bundles, with an enlargement of intercellular spaces and an increase in the number and size of the stomata in infected leaves, in which, again, the palisade cells were shorter and less compacted than in healthy leaves [13]; others have noticed on some varieties (Hope, Preston) of smut-infected wheat a distinct greyish-purple colour on the leaf sheaths and on parts of the culm [27], and a hypersensitiveness of a number of spring varieties of wheat to certain races of the fungus, expressed by inhibition of growth with chlorotic striping and curling, under greenhouse tests [30], but these are exceptional and, in general, there is no external evidence of the presence of infection prior to emergence of the heads [24].

There is no general support to the view that blossom infection is encouraged by those strains of wheat which have the capacity to open the spikelets wider and to keep them open for a longer time than others. This capacity, possessed for instance by the variety Prolific, by virtue presumably of the larger size of its lodicules and their high turgescence, is said to account for seven times as much natural infection by *U. tritici* as in the smaller-lodiculed variety du Banat [26].

On the other hand, neither the extent nor the duration of the opening of the glumes had any bearing on relative resistance to this smut of a susceptible strain of Dawson wheat and a resistant strain of Forward wheat, both of which possessed these characters to the same degree [24].

It is not easy to correlate the incidence of loose smut with the effects of environmental conditions [9]. It is said that seed sown in rich soil produced a lower percentage of smutted heads than seed in poor soil [4]. Others state that the incidence is unaffected by conditions of temperature, rainfall, or date of sowing, and apparently depends only upon the amount of infection in the seed [18]. It is reported, however, that in certain areas in the United States smut is more prevalent in the moister eastern than in the drier western parts of the country. In the presence of a low humidity, moisture for spore germination in the heads appears to be too scanty to enable infection to reach the ovary during its susceptible period. In areas where humidity is low at flowering time, smut does not occur. The important consideration for successful head infection appears to be not so much a prolonged period of heavy humidity as of the amount of moisture prevailing during the critical period of blossoming. Thus, flowers of Little Club wheat exposed for 8 days after inoculation to low (11 to 30 per cent.) and comparatively high (56 to 85 per cent.) rates of humidity yielded 21·9 and 93·96 per cent. smutted plants respectively [25]. But humidity relations are considered to be much more important in the incidence of smut than those of temperature. Some physiologic races of *U. tritici* react differently to temperature; the optimum temperature for growth for some races is 20°, for others 25°, while still others have a wider range for growth [22].

Photosynthesis is said to be more active in smutted than in healthy plants, during early growth up to about 25 days or so, after which the general building up of the plant appears to suffer a retardation, for at flowering time the average dry weight of smutted plants was only 60 to 64 per cent. normal weight [14]. Likewise, respiration of smutted plants during early growth was more active than that of healthy plants and then became more or less constant until the ears started to develop, and from that stage onward the respiratory ratio was again in favour of diseased plants [15]; the amount of soluble sugars was found to be higher in smutted plants, due possibly to the extra drain on the starch content of the host, the amount of starch being found to be proportionately less in smutted than in healthy plants [14]. There does not appear to be any relation between host resistance to loose smut and the degree of hydrogen-ion concentration of the cell sap of the plant [24].

There is no evidence of any relation between morphological characters of wheat strains and resistance to loose smut. Highly resistant and immune strains are found in many varieties of wheat, e.g. Fultz, Fulcaster, Hussar, Ridit, Preston, Junior No. 6 (Gold Coin), Leap, etc. [12, 28], and new varieties of wheat continue to be produced which show high resistance to this and other diseases of the crop [12, 27, 28]. It is claimed that resistance to loose smut in wheat is inherited as a Mendelian recessive character [8, 19].

Loose smut may be checked to a great extent by the well-known method of soaking the grain in hot water, the object being to destroy the infection within the

seed without harming the embryo. The most practical way is to carry out the treatment with small lots of grain, this being preserved for the new sowing for next season. Special seed-raising areas should, if possible, be protected from the prevailing winds at blossoming time to reduce the risk from blown spores, and no wheat fields should be nearer than 500 yards; fields of other crops and surrounding trees acting as wind-breaks will help to reduce external infection. The hot-water treatment (Fig. 191) consists of a pre-soaking for 4 to 6 hours in cold water to ensure complete wetting of all the grains. The grain is then placed in small sacks which are immersed in water at 129° F. (54° C.) for 10 minutes, and during this period it is essential to maintain this temperature, for below it the mycelium is not killed, and if it goes higher the grain may be harmed. Since the maintenance of a correct temperature is of vital importance, it is recommended to have two tubs of water, one heated to about 120° F. in which to give a brief dip of two minutes or so, and then the final immersion at exactly 129° F. for 10 minutes will ensure an equable temperature. A thermometer is essential, and hot water should be added or a jet of steam introduced if the temperature falls, the grain being stirred meanwhile [1]. The grain should be turned out at once and allowed to dry quickly to avoid mouldiness, and should be sown when dry.

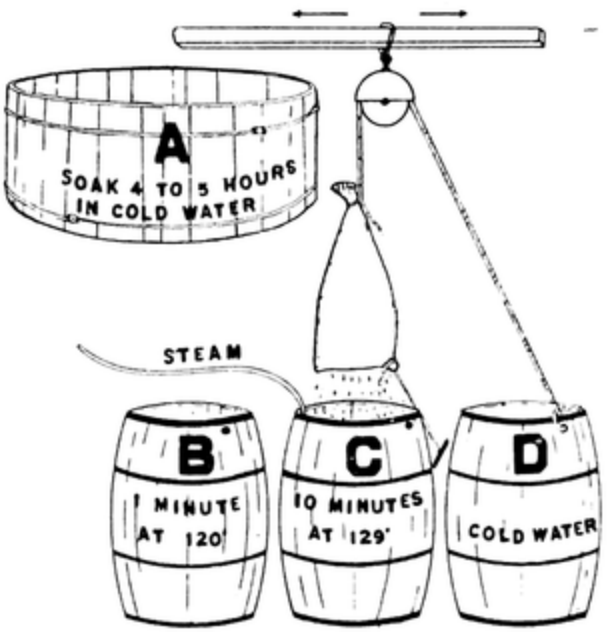


FIG. 191.—A device for treating grain with hot water (after Brentzel, *N. Dak. Agric. Exp. Stn. Circ.* 29)

It is claimed that the efficacy of the hot-water treatment is bound up largely with the exclusion of oxygen, so that by inducing intra-molecular respiration in the grain, alcohol and other katabolic products are set free which are lethal to the fungus. Accordingly, the addition of 2 to 5 per cent. alcohol during the water treatment not only curtailed the process but it is claimed that at 45° C. infection was completely eliminated after a 6-hours' treatment [7]. It is quite likely, too, that the addition of the alcohol renders the grain more easily wettable, and this in itself would shorten the process.

Growing a crop in a dry area for a season, where the conditions of humidity would be so low as to inhibit spore germination at the blossoming period, is said to give a smut-free crop without seed-treatment. But it is obviously of advantage to procure seed-grain from certified smut-free crops if possible.

Seed treatment with disinfectants such as are employed with other diseases of cereals is of no value, since the infection is internal in the grain.

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Powdery Mildew of Cereals, *Erysiphe graminis* DC.

Mildew diseases caused by various members of the Erysiphales are exceedingly common on a great variety of plants in the open and under glass. They are familiar by the white powdery effect which they produce on leaves, stems, and sometimes flowers of their hosts. The various species of mildew fungi are highly specialised in their choice of hosts, which they attack mostly when the plants are in full vigour of life, at no time thriving on their dead remains, so that they are eminently obligate parasites.

Mildew caused by the species *Erysiphe graminis* is found on a wide range of cereals and grasses ; specialised races of this parasite attack oats, barley, rye, and wheat, and also the grasses *Agropyron*, *Bromus*, *Dactylis*, *Poa*, and *Elymus* [14, 20, 26, 27, 38]. Thus, a mildewed crop of wheat offers no danger of infection to a neighbouring field of oats or barley, and vice versa. Moreover, some of these specialised races break up into distinctive physiologic races [24, 38], nine and three of which have recently been reported on barley and wheat respectively [30 a].

The damage done by mildew is difficult to assess, since the host is not usually destroyed, but the general effect is to cause discoloration and weakening of the plant and is more noticeable under glass than in the open. Losses on cereal crops grown in rotation are negligible, but when the same crop is cultivated on the same land repeatedly losses may be high in individual fields [21]. Mildew develops abundantly during the early summer and continues through the autumn to early winter. The symptoms are much alike on all the common cereal plants (Fig. 192). The fungus develops most conspicuously on the leaves, usually on the upper

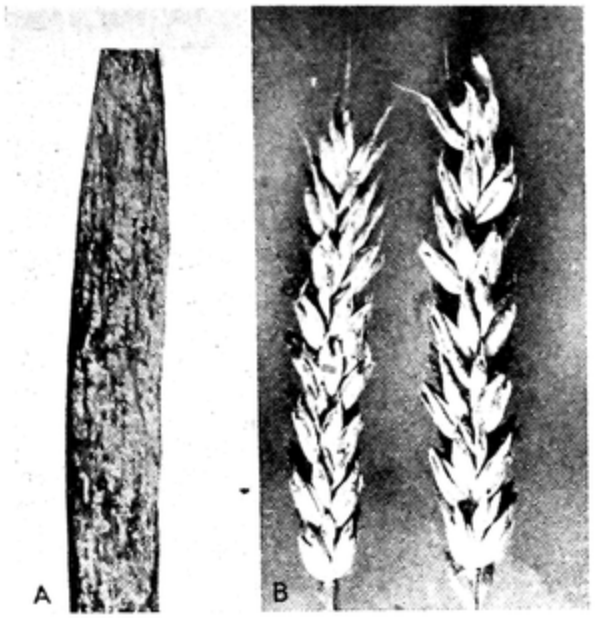


FIG. 192.—Mildew of cereals (*Erysiphe graminis*). *A*, on oat leaf. *B*, on ears of wheat (photos by Foister & Noble)

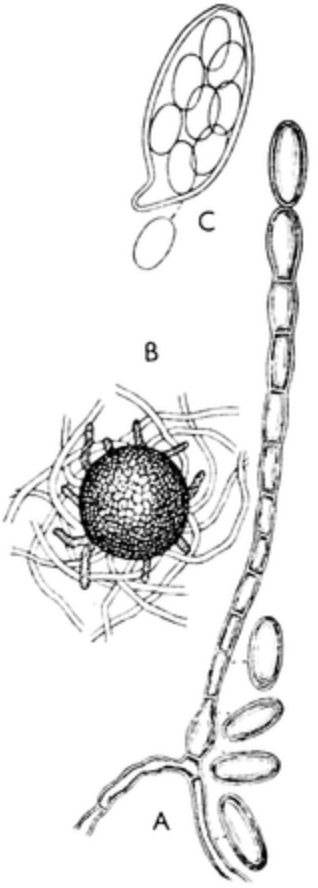


FIG. 193.—*Erysiphe graminis*. *A*, conidiophore and conidia. *B*, a cleistocarp. *C*, ascus with ascospores (*A* and *C*, after Salmon; *B*, after Delacroix)

surface but sometimes also underneath, and also on the leaf sheaths and stems. The effect on the plant at earing is very variable, and the young inflorescences may be unaffected, or sometimes be so badly mildewed as to check their development or cause them to wither completely (Fig. 192 B). The mycelium on the host is entirely superficial, forming a flocculent matted growth, at first white when the conidia are being formed, thereafter changing into a grey or reddish-brown colour when cleistocarps are developed. From much discoloration and coverage of the host-epidermis by the fungus, photosynthesis is feeble and there is much chlorosis, with a consequent weakening of the plant. In bad cases the leaves are crinkled, twisted, or variously deformed; the top of the shoot may droop and wither, and ear development may be wholly or partially checked according to the severity of attack on the leaves. In Southern Rhodesia in 1940 barley mildew reduced the yield of the crops by checking stooling of the young plants [20a].

Mildew may attack the cereal host as soon as the first leaves appear above ground, sometimes killing young seedling growth very early, but mostly the disease is in greater vigour on well-established plants. Later on, usually at earing time, when the production of conidia is waning, the superficial mycelial weft develops the perfect ascigerous stage of the fungus in the form of small, dark, spherical cleistothecia or cleistocarps (Fig. 193 B).

The superficial mycelium is anchored to the host only by the lobed haustoria which enter the epidermis, and penetration is rarely deeper into the mesophyll of the leaf (Fig. 111). The septated hyphae of uninuclear cells, 4 to 5 μ wide, are interlaced to form a web covering to greater or lesser extent large expanses of

leaf and stem. Conidiophores from hemispherical swellings on the prostrate mycelium arise from the web, at right angles to the leaf, from any cell of its hyphae, each to form a chain of about 10 to 20 conidia, the oldest terminating the conidiophore. The conidia are elliptical, hyaline, uninucleate, 25 to 30 by 8 to 10 μ; they readily fall off and are dispersed by wind (Fig. 193 A). They do not retain vitality at high temperatures [28]; and though a temperature of 20° or 21° C. appears to be best for mycelial growth, lower temperatures of 5° to 9° C. are more favourable for starting the germination of the conidia [13].

The cleistocarps are globose-depressed, from 160 to 192 by 120 to 130 μ in diameter [10]; other dimensions given are, 135 to 280 μ in diameter, usually about 200 μ [33]; black, partly immersed in the mycelial weft; furnished with simple, or slightly branched, pale-brown appendages; each contains 9 to 30 cylindrical or ovoid asci, 40 to 60 by 25 μ [10]. The asci are also described as being more or less longly pedicillate, 70 to 108 by 25 to 40 μ; ascospores 8 (rarely 4) measure from 20 to 23 by 10 to 13 μ [33]. The cleistocarps are frequently abortive. The formation of cleistocarps of *E. graminis* is precarious and variable; (their development on wheat appears to be on different lines from other cleistocarps of *Erysiphaceae* [2]). They appear to be set free from the host only after being wetted, the appendages seemingly taking no part in their release [43], though others state the contrary [30]. Moreover, it is reported that the cleistocarps do not complete the development of their spores unless supplied with abundant moisture; it is quite probable, therefore, that they remain to all appearances abortive under dry conditions on the plant which bears them, but complete their spore-development after being wetted and conveyed to a new host. Cleistocarps of *E. graminis* containing immature asci were induced to complete their development by immersing them in water cooled to 9° C., thereafter transferring them to a constant temperature of 21° C., but a preliminary freezing checked them [13, 40]. These fruiting bodies are said to appear in greater number on maturing plants or on older leaves of younger plants as a result, according to some, of a decline in the nutritive value of the host [22, 23] such as after exhaustion from conidial production, but on this point opinions seem to differ [9, 13].

It is recorded that though cleistocarps of *E. graminis* may be well developed and plentifully formed on barley they appear to play little or no part in the spread of the disease on barley [13, 19]. On wheat, however, they are said to be functional [12]. Whether all the known physiologic races of the specialised form of the fungus on barley are equally ineffective is not known, but clearly there exists some other method of spreading cereal mildews in general, other than by conidia during the summer and by survival of ascospores within cleistocarps over the winter. Though the conidia are a ready means for summer dissemination of mildew, they are ill-adapted for survival from one season to the next, owing to their inability to withstand extremes of temperature; but it is reported that in mild climates they are capable of surviving over long periods [28]. The fungus is apparently able to survive on winter wheat, in the form of dense, brown mycelial wefts on the lower leaves, these being capable of producing conidia in the spring, first on adjoining leaves of the same plant, and then spreading to others by wind-borne conidia. While it is true that cleistocarps may over-winter on stubble or debris from a previous crop, they do not appear to bring about widespread infection of either summer or winter wheat, but are responsible, perhaps, for setting up a few foci of

infection from which sufficient conidia arise to cause widespread secondary infections. If summer wheat is grown in the vicinity of a winter crop it becomes infected, in the same way, by wind-borne conidia from the latter, and the possibility of infection of summer wheat from cleistocarps, when winter wheat is in the vicinity, may thus be ruled out. In a locality where both crops of wheat are cultivated the sequence of infections appears to start from infection of winter wheat in the late autumn by ascospores from cleistocarps maturing from August to October. This is followed by the production of the resistant mycelial mats whereby the fungus over-winters on the winter wheat, on which it revives again to break out afresh in spring with production of conidia for spreading the mildew to the same crop and also to the summer wheat. On both winter and summer wheats cleistocarps are developed, first, on the winter, and later, on the summer wheat, and provide for the infection of winter wheat in the late autumn [12, 12a].

It is well known that powdery mildews, in general, appear to be worse during dry seasons, and although a certain amount of moisture is necessary to establish infection, the parasites are capable of adaptation to conditions of moderate drought. But *E. graminis* is reported to be exceptional in this sense, thriving at various degrees of atmospheric humidity with little change [15]. Young barley plants yield to infection without the presence of liquid water on their leaves. Oats, however, demand very high humid conditions [11a, 23a]. Moreover, partly perhaps through interference with the stomatal functions, and partly due to increased evaporation through the mycelial wefts present on the leaves, infected barley plants, it is recorded, transpired 67 per cent. more water per unit area than healthy plants [13]. Epidemics of mildew are reported not to occur if the host cells are in a state of high turgidity, and a reduction of osmotic pressure, as, for instance, by suspending watering or taking the plants from shade to light, is followed by infection [29, 32, 42]. Moreover, the fact that infection with powdery mildew is so closely bound up with the living host during active metabolism seems to indicate that the conditions which favour the vital processes of the host (wheat) also increase its susceptibility to the disease; thus a surplus of carbohydrate in the host leaf, considerably in excess of the normal requirements of the host, is necessary for the development of the mildew [40]. The rate of respiration in wheat was reported to be twice as high as normal, two days following infection by mildew, and the rate of increase was roughly proportional to the degree of infection, the consumption of oxygen being 650 per cent. greater than the normal intake [1].

Light appears to be essential to the development of mildew under normal conditions. Inoculated seedlings of winter wheat (Leap's Prolific) did not develop conidia when kept in the dark [40]. Reports on the effect of ultra-violet light on the fungus and infected host are conflicting; the fungus attacking barley appeared to be remarkably resistant to these rays [13]; on the other hand, the fungus attacking seedlings of Little Joss wheat was killed or rendered dormant, the fungus being suppressed only on the side of the host exposed to irradiation [16].

Mildew of cereals is undoubtedly increased by over-manuring of the crops, due probably to unbalanced application of nitrogenous and deficiency of potassic compounds; it is reduced by phosphoric acid, which also helps the host [35]. In Bavaria high resistance to mildew of barley was thought to be correlated with a

low albumen content and abundant reserves of starch [17]. Experiments have shown that the susceptibility of wheat seedlings to mildew is influenced to a high degree by the composition of the mineral solutions applied to the soil [39]. Winter rye and summer barley showed increased resistance to attack when silicon dioxide was supplied, due, it is thought, to the increased silicification of the cell membranes of the epidermis inhibiting penetration by germ-tubes [11]; cadmium nitrate [36], manganese [5a], and salts of lithium [41] are reported to produce similar results when supplied to wheat seedlings in a nutrient solution; barley in a culture solution containing traces of copper also remained free from mildew [31]. While these minerals, in all cases, may not always assist the host to defy penetration by the fungus, in some cases, by increasing the thickness of the cuticle or otherwise affecting the epidermis, infection is checked by the failure of the fungus to establish the all-essential absorptive haustoria, and even if the fungus succeeds in establishing haustoria, the latter are early encapsuled and killed.

The true nature of the resistant principle residing in the host is difficult to determine. Resistance to *E. graminis* appears to be purely relative, penetration taking place even on 'resistant' hosts, on which, however, infection is early checked. It is suggested that a substance toxic to the fungus is formed in the resistant host following penetration, but, again, there is ample evidence that factors of the environment and structural features of the host may also play their parts in favouring or checking the mildew [6]. It is clear that resistance to *E. graminis* must be studied also in relation to the various physiologic races of this organism on wheat, and especially on barley in view of the numerous races on that host. There are striking differences not only in the type but in the stability of reaction to infection exhibited by these hosts, some showing more or less uniformity of resistance, while others show considerable fluctuation [3, 4, 5, 7, 11a, 18, 25, 37].

Little can be done to control mildew of cereal crops in the field. On a small scale, mildew was checked on young plants treated with sulphur dust, or 1 per cent. liver of sulphur. In California mildew was absent on summer barley supplied with boron, but prevalent on winter wheat similarly treated [8]. Heavy manurial treatment, especially nitrogenous, should be avoided, and phosphatic compounds applied. Considerable control can, no doubt, be obtained by ascertaining the needs of particular soils for culture of cereals, and a balanced fertilising schedule should be observed. Already there are good results to be expected from selection and hybridisation of wheats and barleys that may yield new types resistant to mildew [19a, 30a, 34, 34a].

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Take-all and Whiteheads of Wheat, *Ophiobolus graminis* (Sacc.) Sacc.

This disease affects most cultivated cereals (wheat, barley, oats, maize, rye, rice) besides many wild and cultivated grasses, about a hundred species of which have proved susceptible on artificial inoculation [33, 34, 71]. Wheat suffers the greatest damage, followed in Britain by barley ; heavy attacks may occur when an infected crop of either of these two cereals precedes the other in the rotation. Rye and oats are less injured, oats being apparently immune in some areas [46, 63, 66], presumably because they are not exposed to attack by the oat variety of the fungal parasite causing this disease. This varietal form of the fungus on oats has, however, been discovered in several counties in Scotland on wheat, and the two forms of the fungus may be present in the same wheat crop [7a]. Wheat is probably affected in most of the wheat-growing countries throughout the world, and the first descriptions of the disease in England [1, 64] under the name ' straw blight ', in the United States [14, 35] and in Australia [40, 41] as ' take-all ', do not imply that it was not present in these countries at a much earlier date.

The host plant may be attacked at any stage of growth. Infected seedlings

and young plants may be destroyed (' take-all ') (Fig. 194 B), though more usually they persist and may form ears, or they may be killed while ears are developing or after heading out. The more severe infections often occur when the plants are in the seedling stage or about 6 to 8 inches high [2, 3, 4]. In heavily infected land the disease occurs in patches but it is also found on individual plants scattered throughout the crop. Usually the plants that survive attack are stunted and have few tillers [10, 12, 37]. Those that are killed or seriously injured when nearing ripeness are usually bleached, the blanched and often empty ears being then a prominent symptom (' whiteheads '), especially in dry, hot weather. In cool, moist conditions the bleaching is less marked and the attack may be checked, even enough to allow the formation of grain ; sometimes, indeed, the upper parts show no

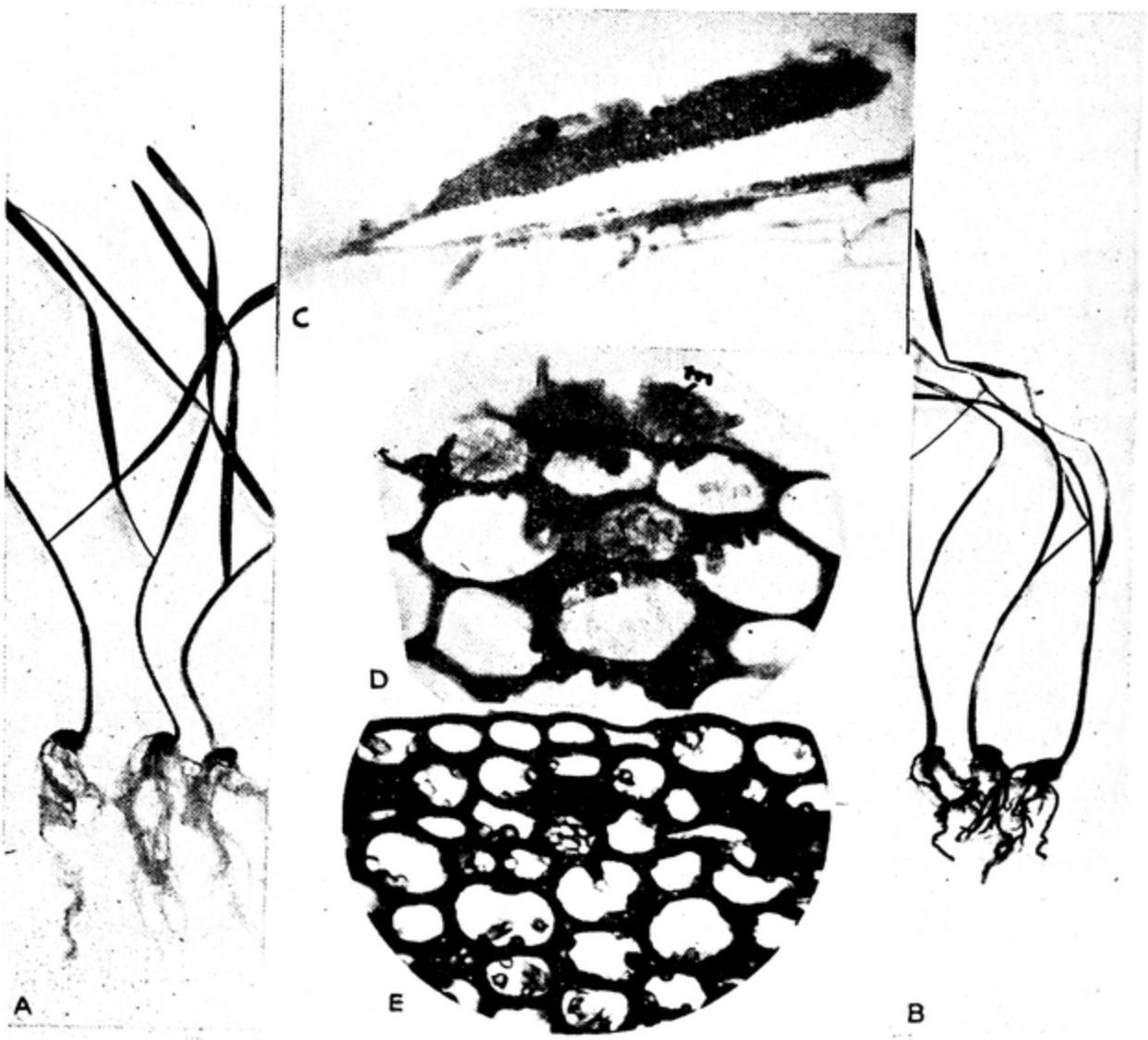


FIG. 194.—*Ophiobolus graminis*. *A*, wheat seedlings incubated but not infected. *B*, infected wheat seedlings (photos by Garrett, copyright of Rothamsted Exp. Station, *Ann. App. Biol.*). *C*, a stromatic ' mat ' or ' scale ' of the fungus, separated from the surface of a leaf sheath of oat, from the stubble. *D*, *E*, sections of root, showing in *D*, at *m*, a mycelial mat and, in both, the formation of ' lignitubers ' on the inner walls of the host cells (photos by Robertson)

obvious signs of injury though the roots may be infected and liable to carry the disease to a succeeding crop [6]. Later on, blackening and shrivelling of the bleached leaves and heads may be caused by saprophytic mould fungi, especially in wet weather. In all forms of attack the roots and often also the base of the stem (crown) are browned or blackened by the growth of the parasite, which is restricted to these parts. Affected plants break off easily at the crown when pulled, but lodging is not increased by the disease.

This disease is caused by a soil-inhabiting parasitic fungus *Ophiobolus graminis*, a member of the Pyrenomycetes [6, 14, 33, 58, 59]. It is essentially a disease of the roots which, in typical cases, are spongy and closely invested by the brown mycelium of the parasite. The mycelial investment gets denser and darker towards the base of the roots and also around the lower part of the stem, where a firm, black interwoven mycelial mat or a series of mycelial scales may be seen on removing the sheaths of the lowermost leaves (Fig. 194 C). On the surface of the roots the investing mycelium consists of long and fairly stout brown runner-hyphae (Figs. 113, 195 D), sometimes termed macrohyphae, and finer, colourless infection hyphae, or microhyphae; if the latter fail to penetrate the root, they turn brown and thicken their walls with age. The infection hyphae may enter the tissues through the epidermal layer of the roots, both primary and seminal, through the epiblast, the coleoptile and sub-coronal internode [6, 9, 13, 51, 52], and in very young seedlings may penetrate as far as the scutellar epithelium (Fig. 114) and sometimes through the leaf-sheaths and culms of young plants, below the surface of the soil, and root formation is hindered [7, 10, 56].

Penetration may or may not be preceded by the formation of 'pegs' (Figs. 124, 125, 194 D, E) (the so-called lignitubers) developed by the host cell on the inner side of the tertiary layer in the wall of the cell entered [39]; later, each infection hypha bores a passage through the peg, becoming constricted to about one-fifth of its external diameter in so doing [9, 15, 56]. The root cortex is rapidly colonised, especially in its outer part where the hyphae tend to run parallel to the root axis; deeper in, they are finer and for a time, at least, are checked at the endodermis, though the stele is penetrated later. Even in the deeper layers the first invasion of mature cells is often accompanied by the formation of pegs. In heavy infections all the tissues except the lignified xylem may be disintegrated [8].

The tissues of the crown may be reached from the epiblast, the seminal roots or the sub-coronal internode. Their invasion is more serious than that of the roots, as it must affect the whole subsequent growth, whereas killed roots can be replaced. In young stems the parenchyma and even the vessels are rapidly penetrated, but older ones resist attack and react by thickening their walls and forming a dark gummy deposit in cells and vessels [9, 12, 13, 52].

Spores are usually not produced during the actively parasitic life of *Ophiobolus graminis*. The perithecia, the only reproductive organs so far known, often begin as the host matures, and in wet weather may ripen before harvest. Ordinarily, however, their development is completed on the stubble. In the field they form almost exclusively in the lowermost leaf sheaths (Fig. 196), but they have been produced experimentally on the roots and stem. They do not seem to occur usually in the mycelial mats around the base of the stem, and are not united in stromata. The perithecia are black, pear-shaped,



FIG. 195.—*Ophiobolus graminis*. *A*, roots of healthy oat. *B*, two oat plants with infected roots. *C*, oat stem showing dark mycelial mats. *D*, ' runner hyphae ' on the root. *E*, basal part of oat stem, showing leaf sheath pulled aside, with necks of numerous small perithecia breaking through the surface of leaf sheath. (*A*, *B*, *C*, *E*, photos by Foister & Noble; *D*, by Dillon Weston)

immersed, ostiolate, the protruding neck being somewhat curved (Figs. 38, 196 B). They vary from 330 to 500 μ in diameter in the broad part, and contain numerous club-shaped asci, 90 to 115 μ long, by 10 to 13 μ wide. Towards maturity the ascus wall deliquesces except for an apical cap which persists when the eight ascospores are extruded in a bundle held together by the intersporal matrix [32]. The ascospores are long, filiform, coiled

lengthwise, and are furnished with 2 to 8 delicate septa ; they measure from 60 to 90 μ long, by 3 to 5 μ wide. Each cell in the spore and mycelium has a single nucleus [32], and the fungus is homothallic [7, 49, 69] (Fig. 55 L, M, N). On germination a germ-tube may arise from one or more of the spore cells. Cultures on artificial media grow rather slowly but are stimulated by the addition of certain growth substances [47, 49] ; a feathery, hyaline mycelium is developed, with a formation of ribbon-like or rhizomorphic strands, turning a deep neutral-grey with age. On a carrot dextrose medium numerous isolates showed an optimum growth temperature around 25° C. but varied considerably above and below this, from 12° to 30° C. ; no growth occurred at 36° C. [48]. The organism is capable of growth over a wide range of *p*H concentration in culture, depending on the physical and chemical nature of the medium [67].

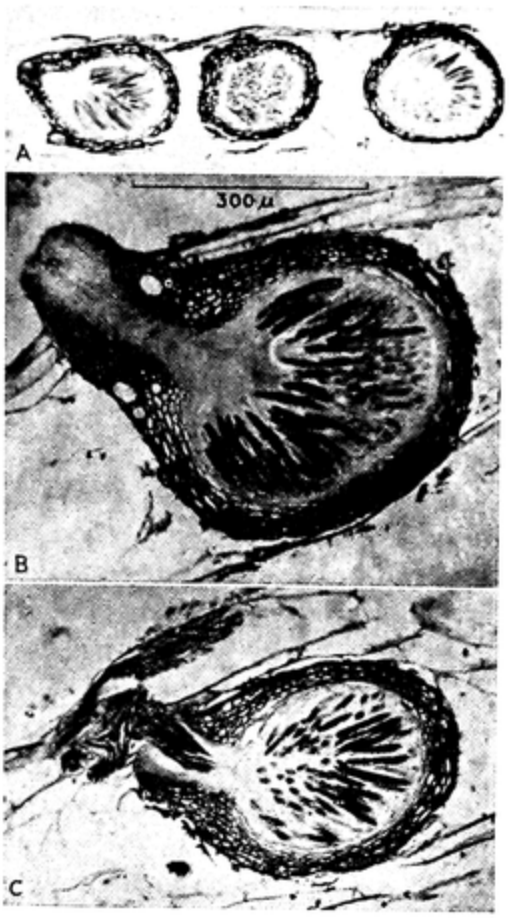


FIG. 196.—*Ophiobolus graminis* on oats. *A*, three perithecia lying in a leaf sheath of oat (from stubble). *B*, section of a ripe perithecium, the neck breaking through leaf sheath, just before spore discharge. *C*, the same, the ascospores being extruded at the ostiole in a viscid mass into the soil

The ascospores are extruded in a viscid mass from the perithecium and may be retained above the ostiole (Fig. 196 C), but when a long dry period has been succeeded by rain they may be ejected into the air at the rate of hundreds per minute [60, 61]. It is not clear what part they play in initiating infection [20], but empty perithecia are plentiful on stubble in the autumn and later. Under wet conditions the ascospores appear to be extruded into the soil in masses and under suitable conditions in the soil it is not unlikely that they germinate, thus contributing to the supply of a resting mycelium in the soil. All the evidence indicates that infection comes mainly from the soil and is much the most prevalent where the stubble from a previously infected crop has been ploughed in ; in western Canada the fungus remained viable in the soil and infected a new wheat crop after two years [56]. The full investigations carried out in England by Garrett indicate that resting mycelium is perhaps the chief source of the disease [16-26]. Ascospore infection may be responsible for isolated attacks, but is apparently difficult to bring about under ordinary field conditions. This may be due to biological antagonism to which *O. graminis* is susceptible, especially when nitrogen is in short supply, or to absence of some necessary growth substance [49, 70]. It is possible that the matrix of the spore bundles contains such a substance and that the dried spore masses in the soil may be more effective than single spores in causing infection. Spread through the soil or from plant to plant within a crop does not occur unless diseased roots touch healthy ones [11, 16, 71].

The soil conditions affecting this disease are of great importance but complex in their interaction. The fungus persists longer in light and alkaline soils than in heavy acid ones [5, 38, 43]. In heavy soil the retarding factor is believed to be the accumulation of carbon dioxide around the infected roots [18]; alkaline soils act as carbon dioxide acceptors and increase its accumulation in the root zone [16]. This accumulation is not thought, by some, to account for the check to the runner hyphae in compacted soils in which enhanced activity of soil micro-organisms is considered to be due to toxic substances produced by other soil organisms which prevent its persistence as a saprophyte in the soil [8, 73]. The decline in viability of the resting mycelium in field soils is well established, and a similar result in sterilised soil has been got where various common soil fungi, *Trichoderma*, *Fusarium*, *Penicillium*, *Aspergillus*, etc., have been inoculated into it. As somewhat similar results were produced by the filtered culture media in which these fungi had grown, a toxic action rather than food competition would seem best to account for the antibiotic action in this case [38, 62, 63 a].

The actively parasitic phase of the life of *O. graminis* is favoured by abundant aeration, a rather low soil-moisture of 35 to 40 per cent. of saturation [56] and a relatively low temperature [42]. Winter cereals in Great Britain, however, are most injured during mild winters followed by a wet spring [17], while in America pot-culture experiments showed that wheat was most injured at 12° to 16° C. and 70 to 80 per cent. moisture [42]. Strain and isolate differences account in part for these variable reactions [36, 55, 57], but the influence of soil conditions on the antibiotic action of the soil microflora must also be taken into account. After the parasitic life of *O. graminis* is over, disappearance of the resting mycelium in plant residues in the soil is most rapid where conditions favour the soil microflora [19, 45], as at relatively high temperatures and moistures, and in compact soils.

Fertiliser experiments indicate that, as in so many other diseases, infection of the stem base is least when a full balance of nutrients is supplied and is also reduced by a shortage of nitrogen [21, 22, 26, 31]. Experiments have amply shown that the survival of the fungus in root and stubble is closely related to an adequate supply of nitrogen in the soil. Root infection was most intense in pot cultures deprived of potash, and stem-base infection when the three main elements were deficient [23, 26a, 44]. Field observations confirm the increased susceptibility of cereals in soils of low fertility. Neutral or acid fertilisers are recommended in preference to those that give an alkaline reaction where the disease is feared.

No fully resistant variety of wheat or barley is known, though the red wheats are reported to be in general more resistant than the white. Control must therefore be directed largely to eliminating the sources of infection and delaying the growth of the runner hyphae. A proper rotation is of major importance, and in the light soils in which *O. graminis* causes most injury in England the succession of wheat and barley should be avoided. Temporary leys in the rotation may also be dangerous if they contain much rye grass, or other susceptible species such as couch, Yorkshire fog, and bent. Two grasses have, however, been found highly resistant to attack, namely, Timothy (*Phleum pratense*) and Tall-oat (*Arrhenatherum avenaceum*), and these, with clover, should hinder the carry-over of infection from a preceding barley to a following wheat crop [24]. It is best, however, to

follow susceptible cereals with root-crops, mustard, flax, or the like; in many areas oats can be safely interposed as it has been found that the strain of *O. graminis* on oats has a restricted distribution [65, 70 a]; in other areas, however, as above mentioned for parts of Scotland [7 a], some of the disease on a wheat crop may be due to this so-called oat variety of the parasite, but apparently barley is not attacked by this variant form.

After harvesting a diseased crop the stubble should be ploughed in as soon as possible to prevent ascospore discharge (but to what extent this may be effective or prevent the spores from producing some other form of the fungus, is not known) and reduce the chance of persistence of the mycelium in the soil until the next crop. Soil management should be directed to securing a firm seed bed. In Australia a considerable measure of control is effected by consolidating light soils, which are also ploughed to a depth of a few inches so that the seedling roots soon reach firm soil. Similar measures have been advocated in Germany [28, 68, 72]. The ploughing in of green manures has also been recommended as an aid to the destruction of the resting stage by promoting the activity of competitive and antagonistic soil micro-organisms [17, 27, 31].

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Eyespot of Wheat and Barley, *Cercosporella herpotrichoides* Fron

By causing a decay of the tissues at the base of the culms of wheat and barley, this disease brings about a collapse or lodging of the stems. It is accountable for serious losses due to death or dwarfing of tillers, or whole plants infected early in the season may be killed ; white or ' deaf ' ears may be produced or there may be a reduction in the size and number of grains (6, 11). Lodging may be fairly general throughout the crop, but very often only individual plants may be seen to have collapsed here and there in the field, resulting in a condition known to farmers as ' straggling ' or ' scrawling ', the straws falling in all directions (6).

The trouble occurs mostly on winter wheat and barley, and both may escape serious infection when sown in the spring (5). The disease has evidently been present for very many years, and has been investigated by Glynne in England, where it was first discovered in 1935 on wheat at Rothamsted (4, 5, 6, 7). In France (1a) and America (9) the trouble has been known for some years, and is widely distributed throughout Europe (9, 12). It has recently been reported in New Zealand (13).

In Britain it is most prevalent in the eastern and southern parts of England where wheat and barley are grown most frequently on the same land [5].

Symptoms of 'eyespot' in the field appear in early spring, as elliptical, dark-bordered lesions on the coleoptile near soil level, and then on the leaf sheaths, and sometimes on the leaves themselves. The fungus penetrates the leaf sheaths, and tillers and whole plants may be dwarfed or killed, so that thin crops result [6]. Small black patches of mycelium appear at the centre of the lesions and a very striking 'eyespot' effect is thus produced on the stems (Fig. 197). The spots occur near soil-level and spores are produced in abundance on these primary infections in the field in early spring, but apparently cease to be formed after about mid-April. They serve to bring about widespread secondary infections of neighbouring plants. In an advanced stage of the disease, owing to decay at the stem base, affected straws are easily broken, the stems may become twisted, and are liable to bend or break in the middle of the eyespot, under any strain [6]. This disease should not be confused with 'sharp eyespot' (Fig. 197, inset) of wheat believed to be caused by *Corticium* (*Rhizoctonia*) *solani*. Lesions of sharp eyespot may run up the stem for several inches; their well-defined margins surround pallid areas on which the brown or purple mycelium grows. Sharp eyespot is not common in England and Wales [7a]; it occurs in Holland [8], Canada, and Oregon [14].

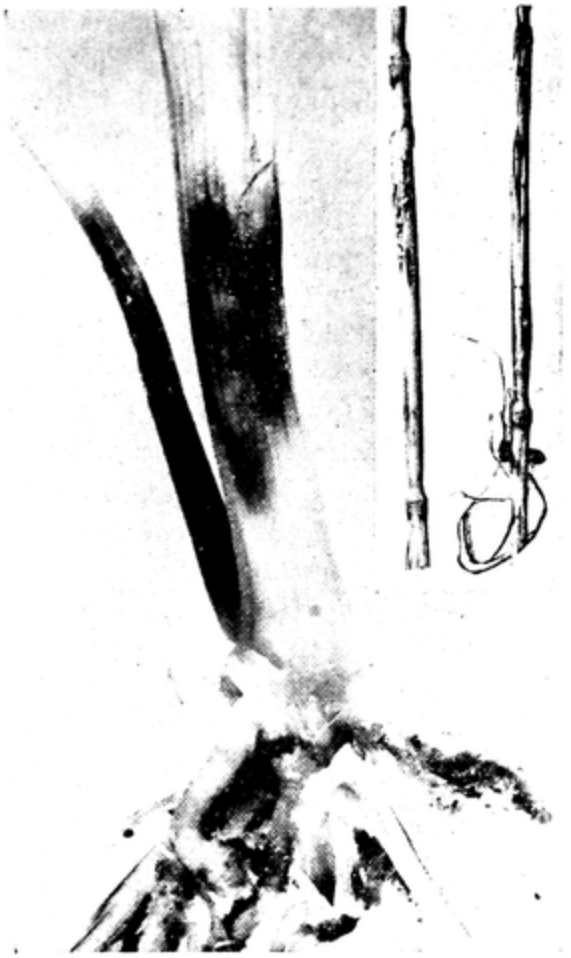


FIG. 197.—Eyespot of wheat (*Cercosporella herpotrichoides*). Lesions at the base of shoot (photo by Glynne, copyright of Rothamsted Exp. Station). Inset, sharp eyespot (*Rhizoctonia solani*) (photo by Foister & Noble)

This disease is caused by *Cercosporella herpotrichoides* (Hyphomycetes). The conidia are borne singly, or in groups of two to several, on simple or slightly branched conidiophores which are swollen at the base. The spores are acicular, curved at the apex, hyaline, obclavate, multiseptate (usually 5- to 7-celled); different authors give the dimensions as ranging from 10 to 105 μ (mean, 50 to 70 μ) by 2 to 3 μ wide at the base, and 1 to 1·5 μ wide at the apex [12]; or, from 30 to 80 μ (mean, 40 to 60 μ) by 1·5 to 3·5 μ wide [9]. On biomalt agar the mycelium is, at first, bluish-grey to mouse-coloured, and later turns olive; the hyphae are septate, and average 2 μ in width; spores have been obtained in pure culture [9], both on coremium-like structures and on sporodochia, and in pseudopionnotes. On potato dextrose agar mounds of grey, pale-edged, velvety mycelium are formed, and finally the growth gets dark on the under side [4].

In some localities the fungus is believed to survive the winter, to some extent, in the soil [11]. It is known to survive on bits of stubble left on the surface of the soil after ploughing, on which it spores freely from autumn to spring, so that infected stubble serves as a source of infection to the new crop [5]. The fungus attacks the host just above soil-level, penetrating one leaf sheath after another, and entering the stem to occupy the vascular bundles and central cavity in which it collects as a dense mass of grey mycelium. There appears to be some degree of correlation between the extent of lodging of the culms (Fig. 198) and the thickness of the ring of sclerenchyma in the affected stems, the more resistant wheats having a wider belt of this strengthening tissue than the less resistant wheats [11].

Eyespot is favoured by moist conditions and is aggravated by wet weather during the growing season. It is liable to appear on rich and heavy land, and its spread is facilitated by the luscious growth encouraged by nitrogenous fertilisers [5], which, however, help the plants to develop new tillers, and so reduce the loss caused by the disease [7 b]. Unlike the ' take-all ' disease of wheat and oats (*Ophiobolus graminis*) and other ' foot rots ' (*Fusarium culmorum*, *F. avenaceum*, etc.), this disease does not appreciably attack the roots, and may be distinguished from ' take-all ' by the fact that it does not form dark brown mats of mycelium between leaf sheath and stem (see Fig. 194 C). While *C. herpotrichoides* is not so virulent a parasite as *O. graminis*, it is reported in some parts of the Continent to be more damaging to the crops in that it attacks wheat early in the autumn, continuing during mild and wet winters, and is, moreover, favoured by having a rapid method of spreading through the production of abundant conidia [3].

To check this disease, long rotations are advised before returning to wheat or



FIG. 198.—Eyespot of wheat (*Cercosporella herpotrichoides*). Plot showing general lodging (photo by Glynne, copyright of Rothamsted Exp. Station)

barley, but little is yet known as to the longevity of the fungus in the soil or elsewhere. With improved methods of cultivation, thin sowing and wide spacing of the rows, and good drainage, late or spring sowings of the crops may escape infection [1,5]. Unfortunately, a wide range of wheats is susceptible to this disease [1,2,3], and the apparent resistance exhibited by some varieties seems to be due to ' escape ' by virtue of lateness in ripening, but the use of short-strawed varieties, which resist lodging even though infected, reduces losses from eyespot trouble [7c]. It has been established that various species of *Aegilops* and *Triticum* showed great differences in their reaction to infection by this fungus, some being markedly resistant, and hope is expressed that resistant types may eventually be developed from hybrids between wheat and certain types of related wild grasses [11].

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Black Mould of Wheat, *Cladosporium herbarum* (Link) Fr.

Cladosporium herbarum is a very common fungus on dead or dying plant tissues; it has also been found to develop on meat in cold storage [3]. Indeed, its spores are present so frequently in the air that it may be expected to appear on almost every bundle of plant specimens collected and kept without drying for a few days.

Though possessing numerous strains, this fungus is at all times a weak parasite, and its frequent occurrence during a wet season on the sub-aerial parts of wheat and other cereals, causing a black discoloration, follows invariably upon an unhealthy condition due to some other cause, insect or fungal. Wheat crops suffering from take-all (*Ophiobolus graminis*) or mildew (*Erysiphe graminis*), for instance, are very prone to develop black mould, but this affection by itself is never the cause of ' thinning out ', or premature ripening, or ' deaf ears ' of the host plants [1]. The main importance is that it reduces the milling quality of the grain.

Black mould establishes itself readily on the moist outer coats of ripe grain, but not usually on young growing grain while in the ears. Except when previously attacked by parasitic fungi or insects, or when the grain has ripened under very adverse conditions, the embryo itself is not affected by this fungus, and even badly affected grains are not prevented from germinating. Such ' mouldy ' grain, however, is obviously in a weakened condition and would hardly be selected for

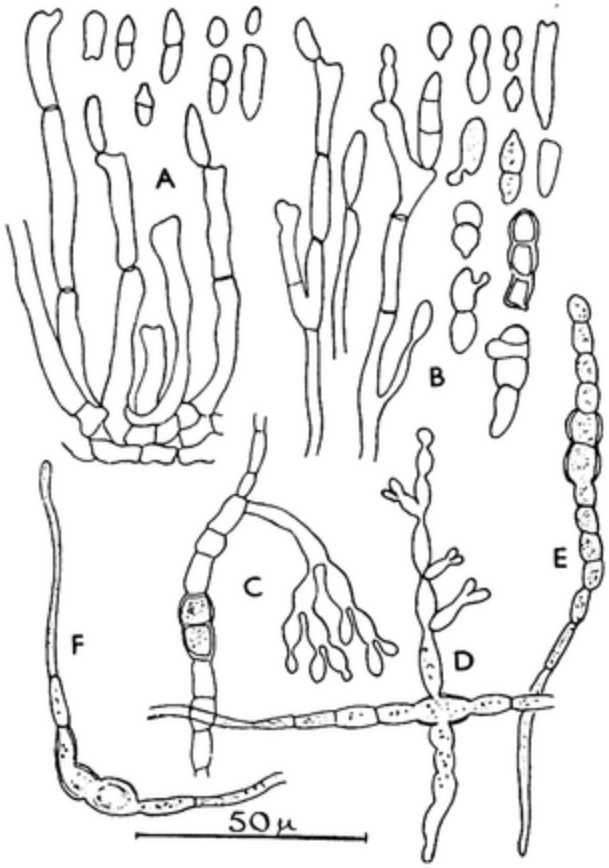


FIG. 199.—Black mould of wheat (*Cladosporium herbarum*). *A*, the fungus, from an olive-green mass on wheat leaf. *B*, a culture from beerwort gelatine (7 days). *C*, germination in tapwater. *D*, in distilled water, at 16 to 20° C. *E*, germination in 2 per cent. dextrose solution, at 16° C., showing budded conidia (the ' hormodendron ' stage). *F*, germination from a conidium in 2 per cent. saccharose solution at 10° C. (after Bennett, *Ann. App. Biol.*)

seed, for its weak seedlings are open to attack even by such a feeble parasite as *C. herbarum* [1]. The affection occurs mainly on the ears of cereals in moist localities or where the crop is growing under too humid conditions. It occurs less often on leaves and stems of wheat and other plants under similar conditions. Affected ears are covered with a greenish-black mouldy growth which is especially pronounced on lodged plants (Fig. 198). During a spell of dry weather the superficial growth of fungus may be almost absent, the surface of the outer glumes being smooth and dark brown in colour. In other cases the fungus often collects in the anthers and stigmas, and the pollen may be much reduced. In dry, harvested grain the organism is found as pieces of mycelium, and spores may also be seen frequently amongst the stigmatic hairs, and knots of hyphae or microsclerotia often occur embedded in the pericarp [1]. Some strains of the fungus such as those found on meat in cold storage are capable of enduring as low a temperature as −6° C., but prolonged freezing destroys the organism [3].

The greenish-black mycelium produces a velvety appearance on the surface of the ear, and within the tissues it tends to form layers of short-celled fungus parenchyma which force their way down between the cells of the host. At and near the surface the hyphae are greenish-brown ; and, deeper in, hyaline. Conidia are produced in tufts on erect conidiophores which usually spring from small stromata developed just below the epidermis. Similar conidiophores can also arise from any part of the external mycelium and, in addition, the hyphae themselves tend to break up, when old, into spore-like portions, the segments thus set free being capable of germinating. Typical conidiophores are greenish brown, lighter in colour at the top, septate, and one or more times bent or kneed, each bend being capable of bearing conidia. The latter are formed in branched chains terminally and laterally on the conidiophores ; the end spore of a chain is the youngest. The basal conidia in the chains are 1- to 3-septate, most frequently 1-septate, while the terminal or distal ones are usually continuous. The shape varies from cylindrical to oval, or almost round ; the colour from brown for the older conidia to nearly hyaline for the younger spores. Each conidium has a characteristic, thickened refractive cushion at the ends marking the articulations with the next spores of the chain. The

conidia are highly variable in dimensions according to the strains of the organism, and range from 5 to 15 by 4 to 10 μ, but in the forms found on storage meat the small round conidia were about 4 μ in diameter while the larger, cylindrical ones measured up to 25 by 4 to 5 μ [3] (Fig. 199).

A second type of spore, the so-called ' hormodendron ' stage, is often associated with the normal form (Fig. 199 E). In this, the conidial chains are chiefly produced at the ends of a brush-like tuft of filaments formed at the tip of a conidiophore ; these conidia are usually non-septate and almost hyaline. But all kinds of transitional stages between the two forms of spores occur, and one gives rise to the other readily in culture. *C. herbarum* possesses numerous strains [1, 2, 3] which vary in many features such as in the length and extent of branching of the conidiophores, the density of the sporing chains, in temperature relations, and in reaction to light [3]. The fungus has no perfect ascigerous stage as was once accredited to it [4], and so far remains in the Hyphomycetes (Fungi Imperfecti).

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Brown Foot Rot and Ear Blight of Wheat, *Fusarium avenaceum* (Fr.) Sacc. and *F. culmorum* (W. G. Sm.) Sacc.

Foot rot is only one phase in the history of this disease, which affects not only the basal part (' foot ') of the stem but the seedlings and the ' heads ' of fully grown plants as well. In most areas wheat and rye suffer more from this trouble than oats and barley, losses in wheat being sometimes as much as 50 to 70 per cent. of the crop ; a number of grasses which include brome and couch-grass are also attacked.

This disease is common in Britain and throughout Europe [1, 2, 3, 4, 5, 16]. In Canada [14] it is widespread, and occurs in the United States chiefly in the Northern, Pacific, and Central States [19]. In Australia [9] and New Zealand [6] this foot rot is considered to be one of the most destructive of all cereal diseases.

In Britain this disease is caused by two species of *Fusarium*, viz. *F. culmorum* and *F. avenaceum* [3, 4, 5]. Another species of *Fusarium*, *F. graminearum*, which, however, unlike these two, possesses a perfect stage, namely *Gibberella zeae*, also causes a foot rot, seedling, and head blight of wheat, but is not common in Britain ; it is described in the next section. In Canada [15] it is recorded that the specific identity of all the different Fusariums capable of causing foot rot of wheat is not known but that the greatest damage is caused by the *F. culmorum* type. The difficulty of tracing the disease to one or more specific organisms is due to the fact that the fungi concerned are capable, in the absence of a congenial host, of living indefinitely in the soil as saprophytes which, however, may resume parasitic activity at any time if a susceptible plant is available to them. Accordingly, the disease is attributed by some to one *dominant* species of *Fusarium*, other species of *Fusarium* being more or less suppressed ; in Britain, foot rot is caused mainly by the two species of *Fusarium* above mentioned. *F. nivale* is also often implicated (p. 479).

There are two sources from which primary infections may attack the host: firstly, from the presence of the fungus already within or on the grain; and secondly, from the planting of clean seed in soil already contaminated with the organisms. From either source, when the seed is sown, the fungus (or fungi) attacks the seedlings in the same way, and as the symptoms of the disease are not greatly different when caused by one or other or by both of the two Fusariums together, no distinctions are drawn here between any effects that the organisms singly, or together, may cause. Bennett [3] describes the symptoms at six stages in the development of the host:

(*a*) *Pre-emergence Blight:* The seed is so heavily impregnated with fungus that the young seedling is killed before its shoot appears above ground. This stage is responsible for considerable ' misses ' in bare wet patches of ground.

(*b*) *Seedling Blight:* The seedling has succeeded in pushing out its first leaf, but brown lesions are seen on both the coleoptile and the coleorhiza; the seedling may survive if the leaf is not bleached or browned, after emergence, otherwise death ensues. From dead seedlings or diseased parts at soil level, the fungus may grow over the soil in small patches to form a whitish ' snow mould ' which infects neighbouring seedlings and may extend its area if a suitable substrate of organic matter is present on the soil. This fungal growth around the base of seedlings sporulates freely in autumn and spring, and spores are disseminated by wind or splashing rain to neighbouring seedlings (*F. nivale* is the main cause of snow mould).

(*c*) *Spring Yellows:* If young leaves of older seedlings become a paler green than normal, turn yellow at the tips, and finally show a blighting effect, young plants usually perish in great numbers during wet weather. Should, however, a dry period with continuous sunshine intervene, a high proportion of seedlings at this stage may, by developing new roots, grow forward into useful though weakened plants.

(*d*) *Foot Rot:* This is the most destructive stage of all, for it involves the internal tissues of the ' crown ', and sets in at a critical time when the plants are approaching maturity. Following the entry of the fungus from the crown into the basal part of the stems and into the roots, the vascular system fails to function properly and wilting results in the death of the plant. When pulled up, the culm breaks off easily from the roots, which remain as a sticky brown mass with much adherent soil and fungus mycelium. The brown discoloration at the broken end of the culm does not extend much above the basal node, and while there may be a certain amount of crimson or yellowish-white mycelium at the rotted end, there is never in this type of foot rot any dense matting of brown mycelium to form scales similar to those which are characteristic of the ' take-all ' (*Ophiobolus graminis*) disease of cereals (p. 377).

The foot-rot phase of the disease may be considered as following in sequence from early attacks on seedlings as above outlined, the plants being enabled to survive until this stage, or it may start as an entirely new infection from mycelium or conidia, penetrating the host tissues from outside at soil-level, for these two species of *Fusarium* are capable of attacking the host at all stages of its development.

(*e*) ' *Deaf-Ear* ' *Stage:* This follows quickly on, and is practically coincident with, the previous stage, and if foot-rotted plants succeed in pushing out any of the ear at all, the latter is found to be empty of grain. This barren condition of the ears is not due to fungal occupation of these parts but to starvation consequent upon inter-

ference with the translocation of food substances from the infected crown below and to the low vitality of the tissues in general.

(*f*) *Blighting of the 'Head':* This stage is due entirely to secondary infections from conidia conveyed (by wind and splashing rain, and swaying of plants against each other) from plants already affected during the foregoing stages. It is as serious a phase of the disease as foot rot and more difficult to control. In wet weather infection by conidia from plant to plant spreads quickly and a whole crop may be ruined in a few days. Not only ears are attacked but conidia settle down also on the succulent nodes below the ears; these nodal infections hardly ever spread over the adjacent internodes above or below, and the foliage leaves remain unblemished. On the infected nodes a sticky mass of conidia ('mucous mould') develops, and this mould may also appear on a part or the whole of the head as a viscid mass of a deep crimson colour, in striking contrast against the bleached or yellow parts of any spikelets of the head that escape infection.

In its attack on wheat *F. avenaceum* is less virulent than *F. culmorum*, doing less damage at the early seedling stage, but in wet soil the general effects on established plants are much the same for the two species. Outside Britain, *F. avenaceum* is reported to cause only seedling blight and root rot of grown wheat plants. In the United States *F. culmorum* seldom causes blighting of wheat heads, but in Holland it appears to be the common cause of head-blight of cereal crops [2]. This species is also found in association with a stem rot and die-back of pinks and carnations [8, 27, 28] and of a rotting of Galtonia bulbs in storage [10].

The following descriptions of *F. avenaceum* and *F. culmorum* (referred to as *F.a.* and *F.c.*) are based on observations of cultures on potato-dextrose agar, and on cooked wheat grains [2, 3] (Figs. 200, 201).

Mycelium:

F.a. White, with traces of rose and yellow; on grain, yellow turning brown.

F.c. White, with yellow, rose and carmine patches; on grain, white with yellow and light carmine, later brick-red.

Sporodochia:

F.a. Rose-buff to vinaceous; on grain, few, large, up to 6 or 8 mm. in diameter, finally apricot orange in colour.

F.c. Ochraceous-brown or cinnamon-Sudan brown; on grain, carmine to ox-blood red, few, 2 mm. in diameter.

Pionnotes:

Abundant from both, sporulation occurring freely on and within the medium.

Chlamydospores:

(*a*) Pseudo-chlamydospores, i.e. chlamydospores arising from converted cells of the conidia, with thick walls.

F.a. From 5·0 to 7·5 μ in diameter.

F.c. Usually the 3 middle cells, up to 14·5 μ in diameter.

(*b*) True chlamydospores, i.e. arising in the mycelium.

F.a. Terminal, generally in chains, up to 11·6 μ in diameter. According to Atanasoff chlamydospores are absent [2].

F.c. Terminal, 8·7 to 13·8 μ, and intercalary 8·5 μ in diameter, singly, or in chains or clusters.

Sclerotia :

Formed by aggregations of segments resembling chlamydospores in dead tissues of plant bases.

F.a. Present.

F.c. Absent.

Microconidia :

Much alike, 3-septate predominant, others 0-, 1-, or 2-septate.

F.a. Spindle-shaped, 25·25 by 4·9 μ (average).

F.c. Spindle or sickle-shaped, 26·0 by 4·3 μ (average).

Macroconidia :

F.a. Slender, elliptical, curved, frequently straight for greater part of length, narrowing gradually at the ends ; wall and septa very thin ; 5-septate predominant (95 per cent.), 43·1 to 65·9 by 3·5 to 3·6 μ, average 57·0 by 3·5.

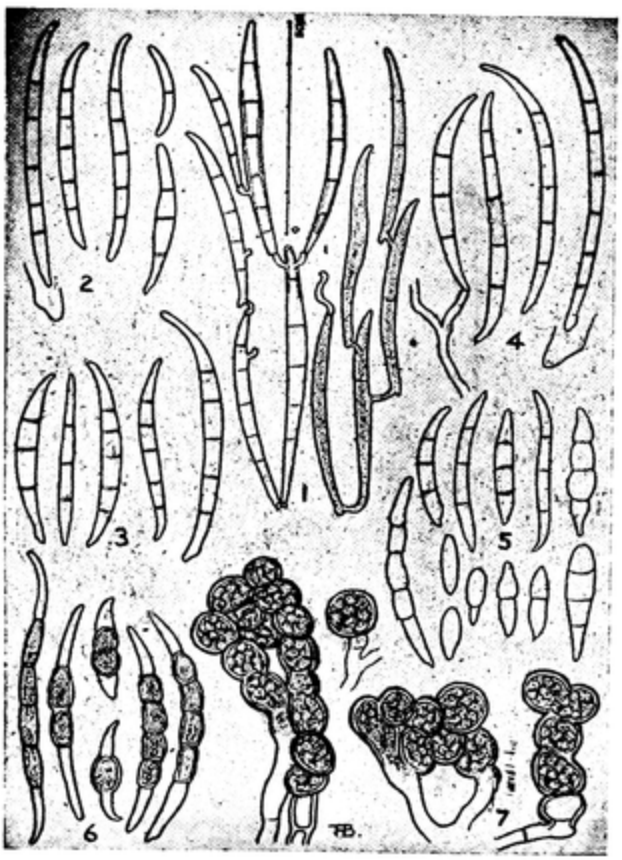


FIG. 200.—Foot rot and ear blight of wheat (*Fusarium avenaceum*). 1, sporodochial elements. 2, sporodochial conidia, from oat agar. 3, the same from salts-dextrose agar. 4, macroconidia. 5, microconidia. 6, pseudo-chlamydospores. 7, chlamydospores and sclerotial bodies (after Bennett, *Ann. App. Biol.*)

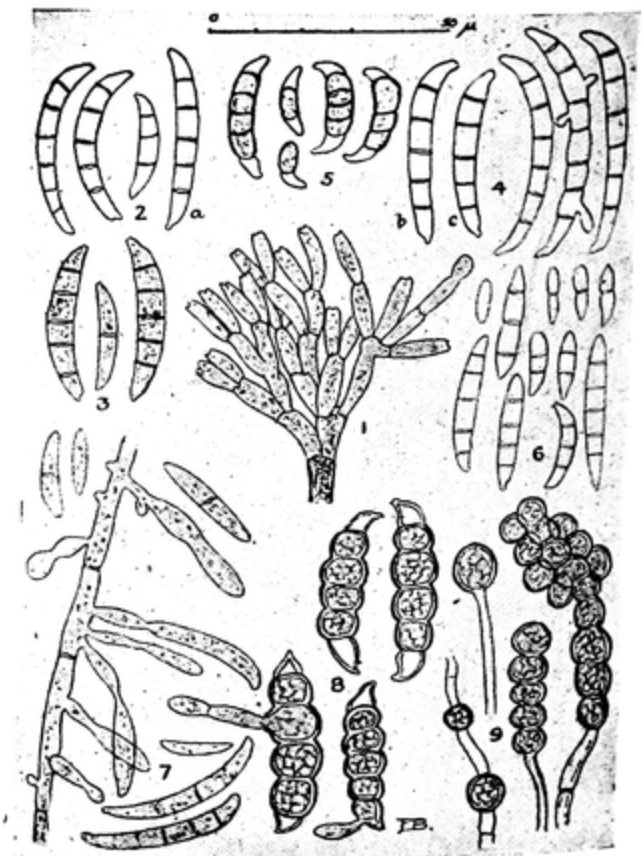


FIG. 201.—Foot rot and ear blight of wheat (*Fusarium culmorum*). 1, sporodochial elements. 2, sporodochial conidia. 3, macroconidia, abnormally wide. 4, macroconidia, including largest forms. 5, conidia from one-year-old culture. 6, microconidia. 7, conidial formation on aerial mycelium. 8, chlamydospores (conidial) from wheat agar (above), and germinating (below). 9, mycelial chlamydospores and sclerotial cluster from wheat grain ; *a*, *b*, *c*, are typical macroconidia (after Bennett, *Ann. App. Biol.*)

F.c. Sickle-shaped, apical cell sometimes constricted near apex ; pedicillate ; thick-walled and pronounced septa. Typically 5-septate (75 per cent.), 30 to 45 by 5·5 to 7 μ.

Parasitism of *F. avenaceum* and *F. culmorum* is of the unspecialised type [20], and both organisms are capable of saprophytic existence on vegetable debris in the soil. The latter organism is especially helpful in the breaking up of straw residues [21], a function which appears not to impair its parasitic propensities when a suitable host is available ; it is a common inhabitant of the compost heap and may occur in a viable condition in the soil to a depth of 20 cm. [24]. *F. culmorum*, too, has been found in soil in which it did not have any appreciable effect on wheat [22, 23]. Sporodochia which have over-wintered on wheat debris are known to retain vitality for over a year ; so also bits of mycelium and chlamydospores after many months' rest renew growth under favourable conditions. Both of these organisms may live for a year or more on straw or grain in storage. Spores, and even vegetative mycelium on plant residues of the previous year are not killed by the lowest outside temperatures in Britain [3].

The manner of infection of healthy seedlings inoculated with *F. avenaceum* or *F. culmorum* has been worked out on oat and wheat [11, 11a, 25, 26]. Conidia placed in contact with the growing oat seed (equivalent to planting in contaminated soil) germinate readily on the surface of the protruding coleorhiza and coleoptile [18]. These initial infections are apparently successful only when the inoculum consists of numerous conidia putting out their germ tubes close together on the surface about to be attacked. (Probably, as in smut and rust infections, successful penetrations can only proceed from a dikaryophytic mycelium established after fusion of germ-tubes.) Natural infections in the soil take place after the germ-tubes have formed a strand of hyphae, from the under side of which, in contact with the host, penetrating hyphae enter the host tissue. The tip of one or more of these hyphae impinging, say, on the coleoptile, first forms an appressorium usually over the vertical wall of an epidermal cell, a position which apparently facilitates the entry of the invading hypha put forth from the flattened appressorium, between the cells of the epidermis. After traversing the coleoptile the fungus increases considerably in the space between that organ and the plumule, and should the latter become thickly covered with mycelium, the young seedling is usually killed before it emerges from the soil. In general, the fungus proceeds to infect the growing seedling by penetrating chiefly the cortical tissue of the mesocotyl, and this it does in an inter- and intracellular manner ; further inward penetration into the stele appears to be held up by the endodermis for a long time. In the axis of the seedling the fungus accumulates chiefly between cortex and epidermis, especially in the sub-stomatal cavities, and here it gives rise to sporodochia which emerge either through the stomata or between disrupted epidermal cells. These fructifications develop conidia which spread the disease to neighbouring seedlings and set up secondary infections. It is at this time, too, that the fungus itself may emerge and cover the surface of small areas of soil around the diseased seedling, to form the ' snow mould ' above mentioned. The roots and crown become infected from the mesocotyl, the fungus travelling therefrom by way of the intercellular spaces of the cortex. It is not usual, however, for the fungus to travel from the crown up

into the culms for more than about 1, or 2 inches at most. Thus, as already indicated, collapse or death of the shoots is not due to the fungus keeping pace with the growth of the shoots, but to its presence in the tissues at the base interfering with plant functions.

Invasion of the crown can also be effected directly from outside, through the natural openings made by adventitious roots when they break through at the surface of the crown. Despite all these infections of the crown, however, the seedlings may yet recover, provided these adventitious roots can become established, as they often do if a dry period intervenes, and with continued growth the balance may turn in favour of the host, the fungus being confined to the parts originally infected.

So far, the fungus has been confined to the tissues outside the endodermis, and since the stelar tissues are still functional, an infected plant has good chances of recovery. Under very wet conditions of the soil, however, the fungus finally penetrates the endodermis to enter the vascular tissues of the stele. With a considerable amount of the cortical tissues of the crown already rotted, and followed now by blocking of the xylem elements with fungus, the disease enters upon its most serious phase, that of foot rot, which culminates, as already stated, not only in more or less complete destruction of the tissues at the base of the culms but in the ' bleaching ', and production of ' deaf ears ' at the heading stage. It all depends, apparently, upon the rapidity with which the crown becomes infected, whether death of the shoots occurs before heading, or is deferred until the plants are fully grown ; but with extreme infection of the crown in all its parts, and clogging of vascular elements, wilting and collapse completes the foot-rot phase of the disease.

The blighting of the heads of healthy plants and of others which outpaced internal infection is, as stated, a secondary stage, being entirely separated from the sequence of infections outlined above. If there is abundant provision of conidia from primary infections and other sources, conidia may reach the heads in diverse ways and, provided wet weather intervenes, infection of the spikelets is easily established [2]. Furthermore, the infection of the stem node below the ear also occurs at the same time as that of the head ; both of these infections are independent of each other and of any previous attacks that may have occurred on the plant. On spikelet glumes and on the nodes below, small brown spots appear which, as they increase in size develop a bleached area and numerous infections occurring together, present a mixture of light-brown and bleached patches, changing from reddish brown to carmine and bearing eventually a delicate covering of white, cottony mycelium. With the return of dry weather, the heads present a more or less bleached appearance, and the mycelium disappears except from places where it is protected within the pales, and in its place a sporodochial production of conidia appears (the mucous mould, above mentioned). The mould is of a coral hue if infection has been done with *F. culmorum*, and of an apricot tint with *F. avenaceum* [3]. According to the degree and place of infection, all the spikelets or only a few in a head may become infected, but an infection which starts low down in the axis or rachis usually results in the death of the healthy spikelets above this point, due again to the breaking-off of food supplies from below, not to actual fungal occupation of the spikelets themselves.

Spikelets which become infected by the fungus entering them from the rachis usually produce grains in which the embryos are shrunken and dead. But infection of the grain in the head may sometimes take place by direct infection of a glume from outside, the fungus penetrating right through to the surface of the pericarp where it causes a diffuse brownish patch on the side of the grain [3]. All the spikelets of a head are usually barren if infection of the head occurs at flowering time; if flowering is over before infection, infected spikelets produce only rudimentary grains which, by becoming covered with mycelium and sporodochia, are usually found with their enveloping glumes and pales stuck together. It is in this way that the grain at germination becomes infected from the presence of the fungus lurking within glumes and pales, and in the same way the fungus attacks the host at germination when clean grain is planted in contaminated soil.

We have seen that weather conditions play an important part in the development of this disease. In the soil a high degree of moisture is essential for infection and for spread of the disease into the crown and roots. A spell of dry weather will usually cut short an attack, especially that of the head-blight phase of the disease.

Initial infections by either of the two organisms concerned take place at comparatively low temperatures (about 10° C.), and since the fungus persists throughout the winter it is probable that infection in the field occurs at any minimum temperature which allows wheat seedlings to grow.

There is little information available about the effects of manurial treatment relative to this disease; a high ratio of nitrogen to potassium appears to aggravate it [20], and it is reported in the case of *F. culmorum* that infections with it occurred with equal vigour regardless of the concentrations of phosphoric acid applied to the soil [12].

Inefficient drainage, wet, acid, cloddy soils, deep sowing, too liberal applications of organic manure, are all favourable to seedling blight and foot rot. But in relation to blighting of the heads it is obvious that improved cultural conditions have no direct effect. To reduce the incidence of foot rot all infected material from previous crops should be ploughed in deeply. As cereals are the best hosts for these fungi, long periods of rotation between straw-producing crops are essential in order to eliminate soil contamination [7]. With winter wheat, while late sowing from mid-November to early December may reduce the incidence of infection, tillering and ear-weight were found to decline, necessitating the use of larger quantities of seed grain [24]. So far as spring cereals are concerned, sowing should be done at the latest date which will give a full crop. To reduce the amount of primary infection seed treatment is strongly advised and good results are reported from the application of mercurial compounds in dust or liquid form [3, 17]. A fine, firm seed bed will do much to reduce infection.

All varieties of wheat, barley, and oats are susceptible to this disease, but oats appear to suffer less than the other cereals [2, 10, 13].

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Scab of Wheat, *Gibberella zeae* (Schw.) Petch

A disease of wheat causing symptoms closely parallel to those described in the foregoing account is caused by *Gibberella zeae* (*G. saubinetii*) the conidial stage of which is also a species of *Fusarium*, viz. *F. graminearum*. In many countries only the latter stage is found. The fungus attacks wheat, oat [7], barley [8], maize, rye, rice, grasses, flax, Ipomoea, hop, horse-bean, and other plants [4, 21, 24, 27, 30, 32 33, 44, 45]. It was not observed on cereals in Britain until 1928 (though suspected on wheat in 1921 [10]), when it was discovered on imported barley which proved to be poisonous to pigs and poultry, and although the toxic properties of barley infected with this fungus have been reported from various sources [35, 37, 39], the deleterious substance is actually a product of the grain formed as a result of infection, probably an alkaloid [2]. The fungus has no such toxic effect on wheat.

The disease is much more destructive in the United States and Canada and in some European countries than in Britain, where it is, so far, of minor importance, though reported from various parts of England and Ireland [7, 9, 11-17, 19, 20, 40, 41]. In the United States losses in 1919 to wheat alone was estimated at about 80 million bushels [17], and in Eire, in three counties, in 1942, a reduction in yield per statute acre amounted to 21·8, 38·4, and 55 per cent. respectively [7].

Although *G. zeae* also causes foot rot and seedling blight of cereals like the foregoing species of *Fusarium* [8], it is as an organism causing head blight leading to grain disease that it has become prominent in its attack on wheat, especially in the United States where it is known as ' scab ' or ' scabby wheat ' disease (Fig. 202). In Pennsylvania in 1921 wheat scab was confined to the infection of the head and was prevalent in that area mostly where wheat had followed maize in crop rotation, the fructifications of *G. zeae* on the old maize stubble being the source of infection [1].

Since the symptoms of foot rot and seedling blight are much the same here

as in the previous type described, only the head blight stage is dwelt upon here. Although head infection of wheat may take place at any time from flowering up to the maturation of the grain, it takes place mostly at or soon after flowering [1a], and is most harmful at the ' soft dough ' or ' setting ' stage [7]. The heads are very resistant to infection prior to flowering [1a]. Affected spikelets present a whitish-yellow hue, as if prematurely ripened, contrasting strongly with the green colour of healthy spikelets. The discoloured glumes are covered, especially along their edges, with a whitish or whitish-pink mycelium which also keeps the glumes stuck together; later, all this fungal covering turns a red colour, especially towards the base of the glumes, and may or may not bear spores (conidia). This appearance of the shrivelled, scabby grain in the ear is, however, not distinctive of the action of *G. zeae*, for this effect, as well as the identical colour shades, may also be produced by *F. culmorum* and *F. avenaceum*, so that no reliance can be placed upon superficial examination of affected grains as a means of identifying the parasite. Sometimes, grains affected with *G. zeae* carry only a film of mycelium, without conidia, especially when occurring on barley, due, according to some, to the fact that the conidia germinate so very quickly that only the filmy mycelium is observed [3, 4, 45].

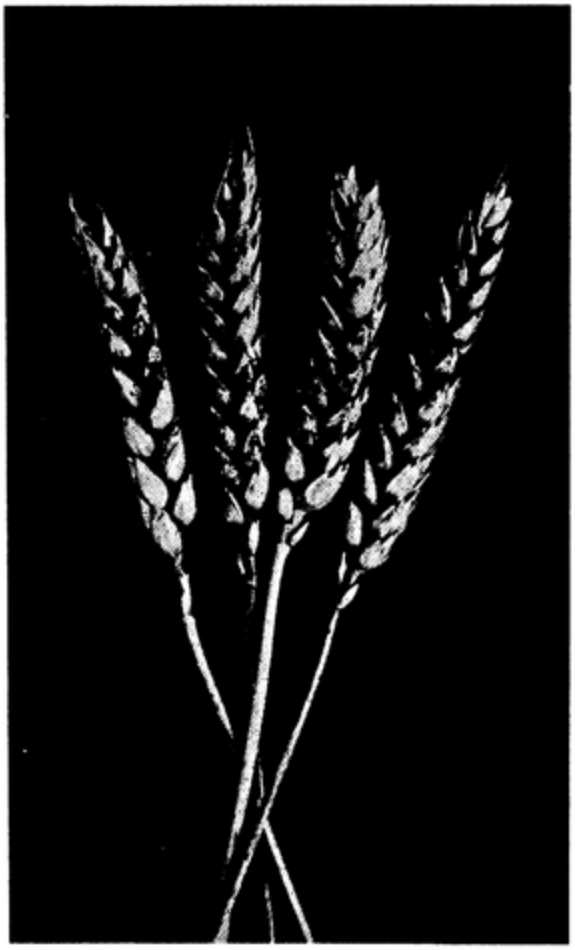


FIG. 202.—Wheat scab (*Gibberella zeae*). The black perithecia on wheat ears; affected spikelets occur in groups. On variety Queen Wilhelmina (photo by McKay, *Sci. Proc. Roy. Dub. Soc.*)

When present on wheat grain the film forms a reddish incrustation of light-pink or crimson conidia, the so-called *Fusarium graminearum* stage. On naturally infected wheat grains of English cereals, 95 per cent. of the conidia were 5-septate, measuring from 48 to 67 by 4·2 to 5·5 μ (average 56 by 4·9 μ), the remainder being 3- to 8-septate (Fig. 203). The conidia are similar to those of *F. culmorum*, but are larger and more slender with a more prominent ' foot '. In culture, they arise below the surface of the mycelium and while a few small, 1- to 3-septate microconidia may occasionally be seen, the characteristic macroconidia arise in sporodochia on the medium, or on a thin plectenchyma as tinted globules of slimy masses, pale ochraceous to salmon orange in colour; chlamydospores are absent [5].

The absence of chlamydospores and the production of perithecia distinguish this fungus from *F. culmorum* and *F. avenaceum*. Perithecia have been observed in nature on the first leaf sheath of blighted seedlings, on the culm near the soil line (in which case they

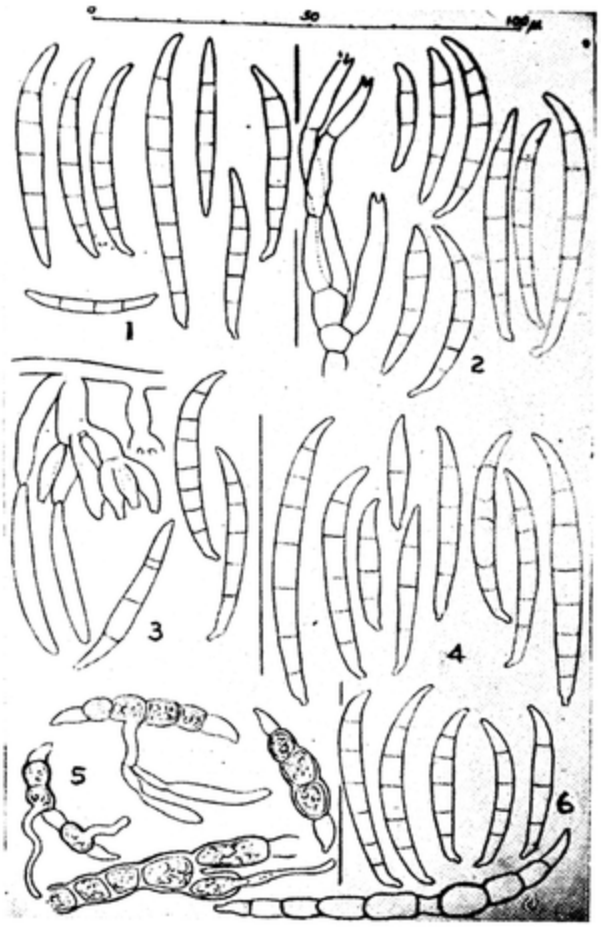


FIG. 203.—*Gibberella zeae*. 1, conidia from wheat grains. 2, conidia and part of sporodochial conidiophore, under dry conditions. 3, conidia from sporodochia on oat agar. 4, same on wheat-seedling base. 5, conidial and mycelial segments from salts-glycerine agar. 6, conidia from pseudopionnotes, on potato agar (after Bennett, *Ann. App. Biol.*)

probably also persist on the stubble after harvest [11]), on glumes and scabbed grain (Fig. 204), and on the node below the spikelets [17]. While the production of conidia is always profuse on artificial media, that of perithecia with ascospores is rare and is influenced by various factors. They have been obtained by inoculating cooked wheat grains embedded in moist, sterile soil, at 16° to 25° C.; after 6 weeks these grains bore clusters of fertile perithecia. They are crowded or scattered, black, and ovoid; on naturally infected wheat grains, under warm and moist conditions, they measure from 200 to 250 by 190 to 210 μ, and are slightly beaked at the ostiole; asci are numerous, up to 100, club-shaped, 8-spored, and interspersed with a few 3 or 4-celled paraphyses; ascospores arranged in single series, or unevenly biseriate, fusiform, straight, or commonly dorsiventral, mostly 3-septate, measure from 20 to 26 by 3·5 to 4·5 μ [5] (Fig. 204 C). Sclerotia-like bodies, probably abortive perithecia, are also formed in culture [5, 18]. Physiologic races of the fungus exist [43, 44].

The mycelium can live in the soil for at least a year on decaying plant residues, presumably as a saprophyte [11], and is capable of spreading in the soil from infected seedlings to healthy ones [28]. Conidia of *G. zeae* appear to be short-lived [45], and in summer, under field conditions, must germinate quickly or perish, but apparently under storage conditions may live for seven months or more, and ascospores survive for at least one year or longer [6]. Soil that has carried a diseased crop is said to contain conidia which may infect directly from the soil, but in Eire, such a soil did not retain infectivity to the next season despite the addition of infected debris to it [8]. Mycelium and spores on scabby heads and infected grain kept out of doors during the winter, in Minnesota, retained their vitality in the dark but not when exposed to light, so that, protected under debris or straw in the field, it is probable that the fungus is capable of living over winter on various substrates [32]. In places where perithecia are absent or rare, as in Britain, infections of spring-sown wheat probably arise from conidia produced in spring on diseased autumn-sown crops, or on residues and diseased grain [6]. Variant strains and environment no doubt account for these discrepant views about the persistence of the parasite.

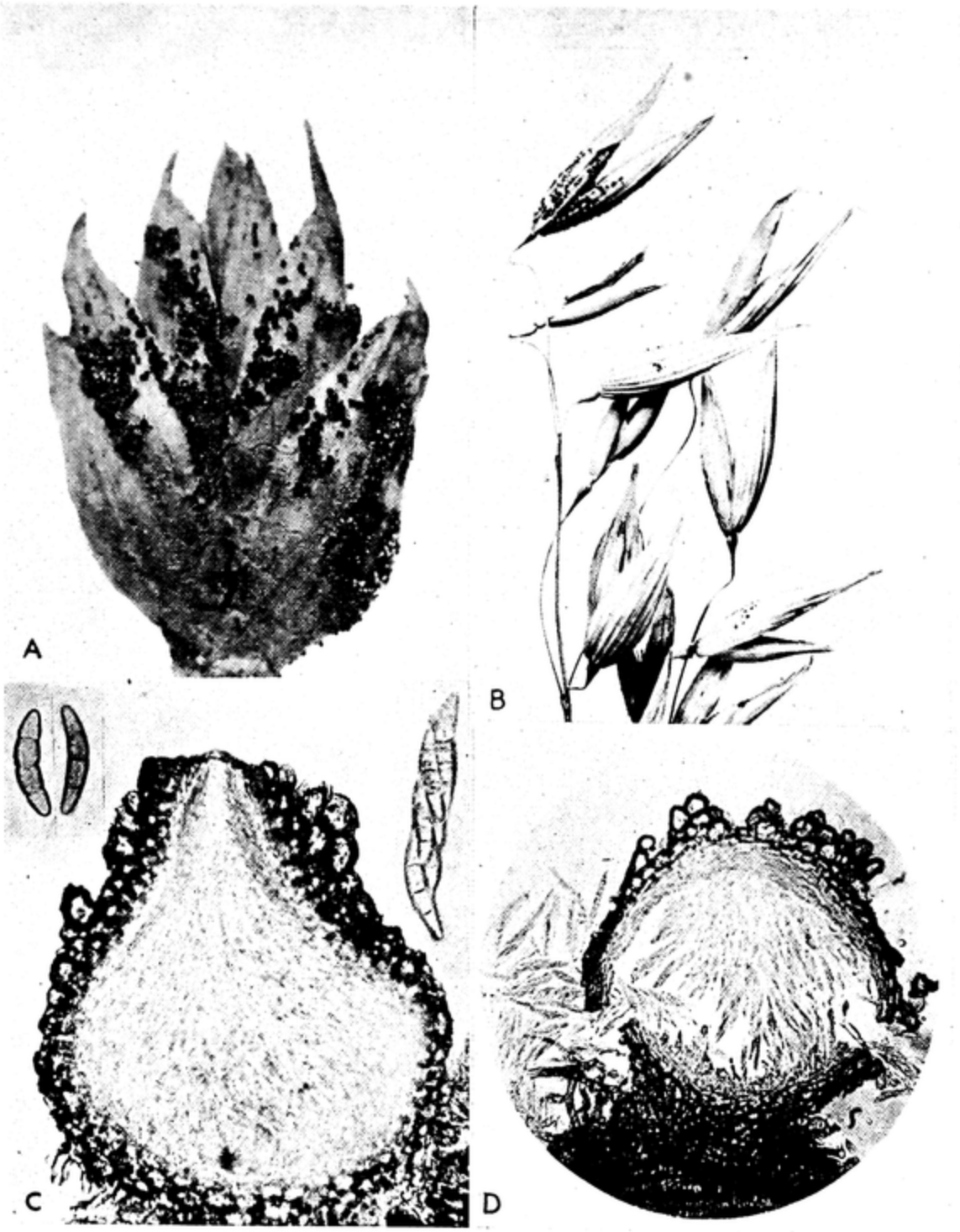


FIG. 204.—*Gibberella zeae*. *A*, spikelet showing perithecia (× 3). *B*, perithecia on spikelets of oat. *C*, median longitudinal section through perithecium showing general appearance (× 460). *D*, section of a perithecium squashed to show numerous asci and paraphyses (× 357); insets, *C*, top left, general appearance of the ascospores when removed from ascus (× 1000); top right, mature ascospores in ascus (× 1000). (*B*, photo by Foister & Noble; *A*, *C*, *D*, by McKay, *Sci. Proc. Roy. Dub. Soc.*)

Infections of wheat by *G. zeae* follow on the same lines as already described for *F. culmorum* and *F. avenaceum*, that is, primarily at seed germination from infected grain or from contaminated soil, and secondarily at the heading stage, but in this case from both conidia and ascospores conveyed by wind, including also spread of infection in the field by contact of heads, especially in wind and lashing rain. *G. zeae* does not attack the germinating seedling to any extent like *F. culmorum* and *F. avenaceum*, and except under extremely wet conditions, or in dry or poor soil impeding growth, does this parasite cause any appreciable mortality in seedlings. Again, this organism as a foot-rot fungus is not so virulent as the two Fusariums; basal attack does not cause the plant to break and fall over, neither is there failure to extrude the ears, nor are deaf ears a common feature of scab disease. Nevertheless it causes a reduction in both size and yield of grain, the extent of which is difficult to estimate, as *Gibberella* and the above-named *Fusarium* species occur so frequently together [6]. As already stated, ears of wheat are susceptible to infection from flowering-stage to maturity. Caught by the extended feathery stigma or 'brush' of the ovary, the spores germinate to penetrate the wall of the ovary in which the developing mycelium becomes intra- and intercellular [1, 38]. Early infection of a floret may inhibit the development of the embryo, which becomes replaced by mycelium, but if infection comes later, small shrivelled grains may result. The hyphae tend to become massed in the aleurone layer while in the starchy portion they form slender strands; the endosperm is disorganised, the starch grains therein forming a loose mass without, however, being attacked. There is not much infection in the pericarp and the typical grain discoloration is attributed to the occupation of the aleurone cells and modification of their contents [6]. Spread of infection from the embryo may extend to the outside of the grain, there to form a thin film of mycelium. This surface mycelium may give rise to conidia and perithecia. It usually happens that grains infected at the germ- or embryo-end are doomed from the start, while those infected at any other point, thus leaving the embryo unaffected or only slightly so, are still viable but liable to give rise to diseased plants if sown. Although the majority of infections take place at the brush end of the ovary, another type of attack, arising externally, may sometimes occur in the form of a single spot on an outer or a flowering glume whence infection passes through into the grain. Furthermore, although grains may become infected in these various ways on the plant during ripening, considerable spread of disease of wheat heads occurs after ripening when the plants are herded together in the stooks, and diseased ears laden with conidia thus come into contact with unaffected ears [6]. Infection of the glumes and the rachis of the spikelets appears to be somewhat restricted and takes place subsequent to the infection of the grain. The general view appears to be that the fungus does not travel from one spikelet to another via the rachis [1], but inoculum placed at the base of a spikelet may extend along the rachis mainly on its exterior and to some extent internally if the rachis is succulent, in which case other spikelets become affected, but it is not believed that this progressive method of axis to spikelet infection is common in nature. The fungus may live for several years within the grain [28], and with the planting of infected seed, seedling infection follows on the same lines as previously described.

The factors which influence infection by this fungus are soil temperature and moisture, and high humidity at ear-blight stage. Wheat, a low-temperature plant, makes steady growth at temperatures of, say, 8° to 16° C., increasing rapidly but with decreasing vigour with higher temperatures up to about 28° C. The fungus, too, is tolerant of low temperatures in both its vegetative and perithecial phases, so that it is not adversely affected by the winter temperatures of our British climate, but its vigour is greatly reduced in natural competition with saprophytes [6]. Since both conidia and ascospores germinate below 10° C. (a temperature which also permits of seedling germination) wheat was found to become infected in sterilised soils during February and March when the temperature did not exceed 10° C. [6], but others say that this is higher, not below 12° C. [11]. The fungus, however, is more or less subdued at low temperatures, and within a range of 16° to 24° C. blight was found to be so severe that seedlings were killed before emergence and other seedlings which had succeeded in breaking into leaf became wilted [12]. At temperatures of 28° C. and higher, blighting usually occurs only after emergence. At low temperatures, protective to the wheat, though mycelium may be found in abundance around the underground parts of the seedlings, penetrations are frustrated, but with rise of temperature the hyphae enter readily through root hairs, piliferous layer, coleoptile, and sub-coronal internode. The fungus hardly ever travels higher into the stem than the second or third internode, and infection does not reach the head, though some have suspected ear infection to take place in a systemic manner [17a]. In relation to temperature it is interesting to note that the attack of *G. zeae* on another cereal, maize, occurs under diametrically opposite conditions [26, 34]. Maize, unlike wheat, is a ' high-temperature ' plant. Whereas at 8° C. wheat plants remain disease-free, maize seedlings become blighted ; at 12° to 16° C. blight is still severe, but gets milder from 16° to 24°, being checked at the latter temperature almost entirely. It is difficult to account for these diametrically opposite effects of presumably the same strain of fungus on different hosts, and it would appear that the dominant influence of soil temperature must rest with the host rather than with the parasite. Further, it has been shown that resistance and susceptibility are correlated with the metabolism of the seedlings of the different hosts when grown at different temperatures [13, 36]. It is inferred that, under the influences of variable temperatures, moisture relations, light, etc., the degree of hydrolysis of starch and protein, of the amount of sugar in the embryo, especially in the growing point and in the coleoptile, the nature of the cell wall, whether of cellulose or pectic compounds, and especially the degree of cutinisation and suberisation, all play their part in the incidence of infection [12].

In relation to the effects of soil moisture on infection, a lowering of this factor to a point where the normal metabolism of the seedlings is inhibited predisposes both wheat and maize seedlings to blight, for at 30 per cent. moisture-holding capacity of the soil both plants were blighted at *all soil temperatures* in infected soil. But this adverse effect gradually lessened with increase of moisture content, until at 45 to 50 per cent. it ceased to be exerted. Thus, during the germination of wheat seedlings at 8° C. and 30 per cent. moisture capacity, 72 per cent. of the seedlings were blighted ; at 45 per cent. moisture, 44 per cent. ; and at 60 per cent. moisture seedling blight hardly existed. When the combined influence of

temperature and moisture content of the soil is unfavourable to the host, fungal attack seems inevitable, due probably to disturbance in the normal metabolism of the plant [12, 13]. Records of the influence on infection of variable *p*H values of the soil are meagre; a *p*H of 5·5 appears to be the minimum value for infection; at or near *p*H 9·0 the fungus is very destructive to young seedlings even before their emergence [22, 23, 42]; in cultures, and probably under natural conditions as well, calcium appears to play an important part in the growth of this fungus [31]. Consideration of the temperature factor indicates that seeding when the soil is cool, that is, of spring wheat at the earliest safe date in the spring, and of winter wheat at the latest safe date in the autumn, reduces the risk of seedling blight [11].

In the control of this disease by crop rotation it is a common observation in the United States that wheat planted on land where infected maize has grown suffers more severely than when it follows any other crop, so that wheat must not follow maize in the rotation [16, 29]. Since *G. zeae* is also harboured by wild grasses, removal of these from the vicinity of wheat areas, together with clean cultivation, is advisable. The disease is highly variable on different varieties of wheat and no variety is known to resist infection under a wide range of conditions.

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Loose Smut, *Ustilago avenae* (Pers.) Jens., and **Covered Smut,** *Ustilago kolleri* Wille, **of Oats**

' Loose ' and ' covered ' smut diseases of oats are caused by two closely related species of fungi, *Ustilago avenae* and *U. kolleri* respectively. Both types may be found wherever oats are cultivated ; the former appears to be much the commoner in Britain.

Before the control of smut by grain treatment was adopted, smut diseases of oats ranked with the most destructive plant pests in temperate climates. In the United States loss of grain through smut is estimated to be very high ; in Canada, smut is far more common on oats than on wheat and barley, and the annual losses of about 6½ million dollars are estimated to exceed those due to all the other cereal smuts put together [(6)].

It is not easy to distinguish between these two diseases in the field, and both may occur together in the same crop, but there are no visible symptoms of either until the plants have reached the heading stage.

With the appearance of the black sporing-masses (' brand ' spores) in the heads, the plants affected with either type of smut are shorter than healthy plants. Those affected by loose smut may usually be distinguished from those suffering from covered smut by the branches of the panicle standing erect and more or less closely investing the central axis, so that the smutted head presents a more compact appearance than a panicle affected with covered smut, the branches of which are lax, with the smutted spikelets dangling down just like the healthy spikelets (Fig. 205). But, in fact, it is not a safe criterion to distinguish between the two smuts from mere external appearances of the panicles and, for certainty, microscopical examination of the smut spores is essential. The spores of loose smut have a spiny (echinulate) wall, whereas those of covered smut are perfectly smooth.

Smut is usually very destructive to the protective chaffy scales, and the tissues of the ovary are entirely replaced by spores ; and other parts of the spikelets, even the glumes and awns, may also harbour them. In the covered smut, however, the spore masses are held together within the grain by a thin, whitish membrane, whereas in loose smut this protection breaks down and the spores are liable to be blown away by the wind until nothing remains of the head except the stalk. On the other hand, heads of covered smut remain more or less intact during the whole time the crop is standing, and the spores are not usually liberated until after harvest when smutted and healthy grain go together to the thresher, and from contact with the smutted grain, now broken, healthy grain becomes contaminated with the



FIG. 205.—Covered smut (*Ustilago kolleri*) and loose smut (*Ustilago avenae*) of oat. *A*, covered smut. *B*, *C*, loose smut, latter showing lesions also on the leaf. (*A*, photo by Foister & Noble; *B*, by Dillon Weston; *C*, by McKay)

spores set free. Spores from the panicles of loose smut, being free, are conveyed by wind to the spikelets of healthy panicles open for pollination, and thus reach the developing grain while the crop is standing. There is, however, no hard and fast distinction between loose and covered smuts of oats, in respect of the retention or not, of the spores within the grain. Those of covered smut may sometimes escape in the standing crop, while those of loose smut may sometimes remain in the grain until threshing. Whether the spores merely remain imprisoned and dormant within the coverings of the grain until the time of sowing or whether there is actual infection of the developing grain at the blossoming period is discussed again below, but in both loose and covered smut, grain contamination takes place, and infection in each case occurs when the grain is sown, the fungi penetrating the seedlings and keeping pace with the growth of the plants. Infection is, therefore, systemic, and with the conversion of the panicles into smutted heads, the life-cycle is complete.

Loose Smut of Oats, *Ustilago avenae* (Pers.) Jens.

Loose smut is much more common everywhere than covered smut. It is also more destructive to the spikelets, for in addition to occupying the ovary there is usually not an awn or a glume that has not been replaced by the sporing masses of this fungus. Sometimes sporulation occurs on the uppermost leaf blade as well as in the panicle (Fig. 205 C) [22]. All the heads of a stool and all the grains of an ear may become infected, though some of the upper grains may often escape.

The spores are globose to oval, olive-brown singly, with one side lighter than the other, dark, almost black, in the mass, and with a finely echinulate wall; they measure from 5 to 9 μ in diameter, and are uninucleate and diploid. Germination takes place in a few hours in water or nutrient solutions and on solid media with the production of a stout tube or promycelium (basidium) which becomes four-celled by transverse septation. Each cell of the basidium is uninucleate, and the nucleus divides, sending one daughter

nucleus into the developing sporidium (basidiospore) which arises near a septum, the top cell producing a terminal sporidium. The other nucleus remains in its basidium-cell and may divide again to provide nuclei for more sporidia (Figs. 61, 62). During the formation of the promycelium the nucleus of the spore passes through a 'reduction division' and the sporidia are therefore haploid. The spores may germinate differently at different temperatures, but whether such differences are to be attributed entirely to change of temperature or nature of the culture medium or to distinct strains of the fungus is not known, but at 0° C. the germination of certain strains was inhibited, and at 5° to 10° C., in place of forming sporidia, the cells of the promycelium produced narrow hyphae which in some cases were seen to join together; and at higher temperatures of 20° to 25° C. a few sporidia, gemmae, and narrow tubes were obtained [37]; in nutrient solutions the sporidia may bud freely after the manner of yeasts [6].

The history of the smut fungus following the fusion of pairs of sporidia or of their mycelial equivalents has already been described in Chapter I, p. 40 *et seq.* The four or more sporidia produced by a diploid spore are not all genetically alike, and for exact biological studies, monosporidial, not monospore, cultures of the fungus are essential. The sporidia are then found to fall into two groups or 'sexes', arbitrarily designated 'plus' and 'minus', two being + and two − [3, 4, 19]. Fusion of sporidia in pairs, an essential preliminary to infection, is only effective between + and − sporidia, and infection fails if the sporidia are all + or all −. There is no morphological difference between the mating sporidia. The union is not always between sporidia, and the uniting elements may be the germ-tubes or mycelium produced by them, or other cells budded from them (gametic equivalents), and the fusing elements may actually be two cells of a promycelium connected by a buckle-joint or a clamp connection [12] (Fig. 206).

The haploid sporidia or their gametic equivalents are incapable of causing infection, and it is only the mycelium that arises following the copulation of these elements that initiates the parasitic phase in the life-history of this and probably most smut diseases (see p. 100). After union of the two gametic elements, the respective nuclei, which for simplicity we may refer to as 'male' and 'female', do not fuse together but remain in association as paired nuclei. This association, though difficult to detect in the parasitic mycelium, presumably remains throughout the whole career of the parasite within the plant, from the time the fungus attacks it at the seedling stage to its final destination in the flowers at the heading stage. From the presence of the nuclei in pairs, in the mycelium which infects the host, the parasitic history is called the dikaryophytic phase, but though microscopic examination of the fungus during this phase does not always show the nuclei in close association in pairs, it is presumed that the nuclei which meet and finally fuse are of different sex. When the fungus begins its sporing in the ear, a male and a female nucleus come together again in every young spore, and it is during the ripening of the spores in the ear that the long-deferred fusion of the two gametic nuclei takes place, making every spore, therefore, a diploid reproductive cell or zygote [16]. The ripe spore alone represents the sporophyte in the life-history of the smut organism, for during its germination, as above stated, the fusion nucleus passes through the reduction stage and the promycelial cells and their sporidia return the organism to the haploid condition. Sometimes a spore produces a

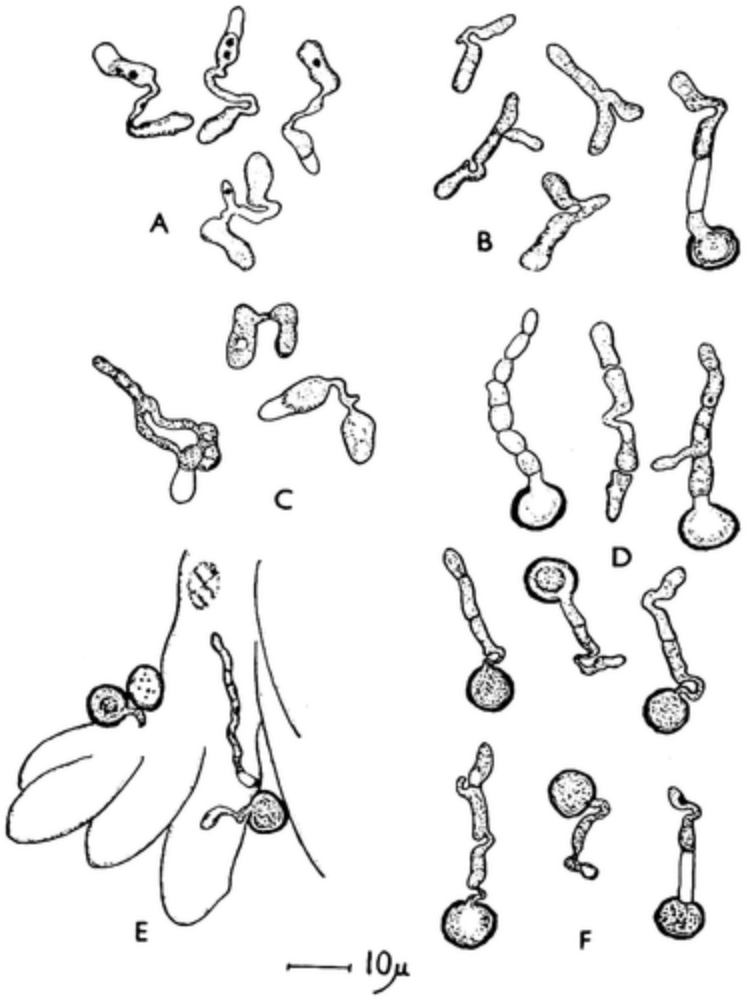


FIG. 206.—*Ustilago avenae*. *A*, fused sporidia, from artificial culture. *B*, spores germinated within the glumes, and fused segments of promycelia. *C*, fused sporidia obtained from within the glumes, after inoculation with a suspension of two monosporidial lines of opposite sex. *D*, flower infection; germinated spores on the stigmas and ovaries, showing fusion of promycelial segments and formation of gemmae. *E*, spores germinating on the stigma of Anthony oats; direct penetration by germ-tubes, as found also in seedling infection. *F*, spores germinated within the glumes at 5° C. (after Western, *Phytopath.*) (see also Fig. 121)

promycelium (usually only 2-celled) which has not passed through the reduction division and which does not produce sporidia; being still diploid, such a promycelium or its derivatives is therefore capable of establishing infection forthwith [36] (cf. Fig. 62).

There is some diversity of opinion as to the extent to which infection of the flowers takes place in the standing crop, whether it is that the wind-borne spores which find their way into the open spikelets remain ungerminated but viable throughout harvest and storage, or whether they germinate forthwith so as to penetrate the various parts of the spikelets and particularly the developing grain before harvest, in which case there is the possibility of the formation of a resting mycelium in the glumes or on the grain. There is, however, general agreement that if at the time of pollination smut spores do happen to fall on the stigma or the ovary (Fig. 206 D, E), they germinate in a very short time and the mycelium produced by them may invade the pales or the pericarp, or both. But it is by no means certain that this mycelium, which remains dormant throughout harvest and storage of the grain, can start the disease afresh when the infected grain is sown. Some say that it is only this mycelium resting in the glumes or pales that is effective [38]. The artificial inoculation of the flowers of oat by spores of loose smut is not an easy operation to perform, and while the extent of infection may depend upon various factors such as the capacity of an oat variety to open up its pales, the conditions of the environment at time of flowering, and other unknown, perhaps, inherent factors, it appears that flower infection in standing crops of oat is not nearly as common as in loose-smut infections of wheat and barley caused by *U. tritici* and *U. nuda* respectively. A great deal of experimental work, involving

various methods of inoculating the flowers has been carried out with smuts, not only as above with the hulls intact but also with grains dehulled by hand. Such shelled grain, contaminated before sowing, gave a high percentage of infection; those with hulls intact gave less, and flower-inoculated oats gave the least amount of infection [22]. While the extent of flower-infection in oats depends apparently on a variety of factors, including the type of oats and the particular strain of the pathogen experimented with, it is generally conceded that most infections which are traceable to the flowers are actually due to ungerminated spores which have been lurking within the glumes and pales or which have remained adherent to the pales. These spores remain viable *in situ* throughout harvest and storage, and revive to attack the grain when it germinates [18, 22]; occasionally, perhaps, seedling infection may occur also from resting mycelium in the pales [38, 39].

As above stated, before the fungus can infect the young seedling it must, in one way or another, have established a dikaryophytic mycelium. Under natural conditions this preliminary is no doubt effected when numerous spores germinate close together in contact with the seedling, so that when the sporidia are produced, they or their germ-tubes copulate and, by the passage of a haploid nucleus from one sporidium or its germ-tube to another, a dikaryophytic phase is initiated in the now binucleate sporidium or germ-tube, as the case may be; the empty sporidium or tube remains passive [8] (Fig. 121).

During the early stages of germination of the oat grain the plumule or first bud is pushed out by the elongation of the mesocotyl region below, and the fungus usually penetrates first just below the base of the plumule. Opportunities for penetration are brief, for after the primary shoot is about an inch long, further attacks are resisted. In about a week following infection the fungus is found in the sheath or coleoptile and in the space between that organ and the first leaf within. The infecting hyphae are intracellular in the epidermal cells, but when the deeper tissues are reached the mycelium is intercellular. The fungus, having reached the young axis, continues its growth upwards and, once the mycelium is present in the apex of the young shoot, systemic infection begins, for the fungus now keeps pace with the growth of the host until spores are formed in the heads. Within the growing point the advancing mycelium appears to be entirely intercellular and composed of somewhat broad, curved segments, some of which are uninucleate, binucleate, or even multinucleate, and even just before spore-formation it is very difficult to find evidence of conjugate nuclear division [20] and to detect at what stage the nuclei become associated in pairs prior to spore formation. During its progress within the host the fungus apparently has no adverse effect on the tissues until it accumulates in the spikelets, where eventually the tissues of the ovaries are destroyed and replaced by smut spores.

Sometimes, according perhaps to variable external conditions such as temperature, humidity, and perhaps to other factors that may induce rapid growth of the host, the infected plant may outpace the progress of the fungus, so that when the ear stage is reached the inflorescence is free from fungus and develops perfectly healthy grain. The presence or absence of smutted panicles is therefore no certain guide to susceptibility to, or immunity from, smut disease, for the infection may still exist at the base of the plant. Infected plants which gave healthy heads at harvest

may, after harvest, throw weak tillers which are completely infected with fungus which had travelled up from the stool. Even varieties of oats which are resistant to or immune from smut fail to prevent infection entirely, but with these, penetration does not usually go further than the epidermis [36], the contents of which have apparently a toxic effect on the infection hyphae, and the disease is completely checked (Fig. 121). But it is very difficult to determine the exact nature of the factors which appear to allow the fungus, in greater or lesser degree, to enter the host in some cases, with no evidence of its presence to external view, and with no inhibiting effect on the growth of the plant [2, 28, 29, 30].

There are several physiologic races of both these species of smut fungi and, as some of them have a wide geographical range, the breeding of oats for resistance to smut in particular localities is rendered very difficult, and especially so in view of the comparative ease with which the two species hybridise. Furthermore, in view of the difficulty of detecting a clear resistance to smut owing to the prevalence of incipient or latent infections in ' resistant ' oats, it is evident that the establishment of pure breeding lines resistant to or immune from smut has proved to be a matter of great practical difficulty, and, indeed, a pure-breeding resistant type of oats which has the capacity of producing consistently a low degree of infection has not so far been found [33], and varieties which have shown a high capacity of infection under experimental conditions are not always susceptible in the field [29].

Since the oat plant is only susceptible to infection during the early stages of germination, the extent of infection is obviously influenced by various factors of the soil environment, of which temperature and humidity play an important part. The optimum temperature for the germination of the spores extends rather widely from 15° to 28° C. (minimum 4° to 5° ; maximum 31° to 34° C.), and this range is practically the same for optimum growth of the host [1, 11, 15]. In general, it may be stated that a comparatively high temperature and a low moisture content of the soil are favourable to infection. The interdependence of the factors of soil temperature and soil moisture in the incidence of smut indicates a method for its control, for by the practice of early sowing of oats in the spring, when soil moistures are high and temperatures are relatively low, risk from infection is greatly reduced. Thus, in trial sowings in April and May, when soil temperature was 11·1° C. and soil moisture content ranged between 45 and 50 per cent. during the spring, the crop showed less than 0·1 per cent. of smutted plants, whereas under reverse conditions of relatively high temperatures and low soil-moistures all the crop was smutted [1]. Furthermore, any factor (such as deep planting) that retards the germination of the seedling prolongs the period of susceptibility during the pre-emergence phase, and any factor that encourages slow development of the host is favourable to the parasite, while rapid germination and growth, although not preventing infection, may help in the production of normal panicles, which thereby have outpaced the disease [5].

Little information appears to be available about the effects of temperature and humidity during the later phases of the disease, and little is known about the effects of fertilisers on the crop in relation to incidence of smut : it is known, however, that while the application of phosphates gave very high increases in the

field, there was no evidence of any reduction in the percentage of smutted panicles [33].

The great success of the treatment for control of smut disease by seed disinfection indicates clearly that infection takes place largely from spores adhering to the grain-coat, and which germinate when the seed is sown. In localities where it has been definitely established that infection of the seedling takes place from a resting mycelium in the grain, seed disinfection has also proved effective since the fungus is confined to the surface of the caryopsis. Infection appears to be mainly from spores within the glumes, and spores on the surface of the glumes are so dry and powdery that they do not stick to articles with which they come into contact so readily as the spores causing bunt (*Tilletia*) in wheat, and the danger of conveying smut by contaminated sacks and implements is not so great [38]. Although smut spores obviously fall in great numbers on the soil, there is no evidence that any appreciable amount of infection of the germinating seedlings occurs from spores in the soil.

Treatment by seed disinfection is effective against both loose and covered smut. The most popular fungicide is formalin, in solution as used for wheat bunt and covered smut of barley, or it may be used as a dusting powder. For hull-less varieties of oats copper carbonate dust is recommended ; it should contain about 50 per cent. of copper and be applied at the rate of 4 oz. per bushel [6]. This treatment has been known to give a 70 per cent. reduction of disease in the case of hull-less oats which are known to suffer severe injury if subjected to the formalin treatment [32]. Good results have also followed from the use of organic mercury dusts, both from the standpoint of smut control and effect on germination and stand [21].

Covered Smut of Oats, *Ustilago kolleri* Wille

We have seen that in loose smut, so complete is the destruction of the spikelets that the spores are easily dispersed by wind, and healthy panicles are infected in the field at flowering time, or soon after. Spikelets affected with covered smut are, however, able to preserve their shape owing to the permanence of the glumes, and whilst it is by no means unlikely that spores of covered smut may often escape from cover when the plants beat against each other in the field, and may be carried by wind, as are those of loose smut, to panicles that may be rather late in flowering, the spores of covered smut, as already mentioned, mostly remain in the heads until harvest time. When the smutted grain is threshed along with the healthy, the protective membranes are broken, and the great masses of spores thus liberated find their way under the hulls, or in varieties of hull-less oats they settle down on the grain itself. In this way sound grain becomes contaminated (Fig. 205 A).

Covered smut of oats is caused by *Ustilago kolleri* which name now replaces the older one *U. levis*. The spores are similar to those of *U. avenae* but perfectly smooth-walled, while those of *U. avenae* are spiny. The spores of both these species germinate in the same way and the mode of infection and histological features within the host are, as far as known, identical. The spores of covered smut are viable over a longer period than those of loose smut. In culture they gave the highest percentage of germination about 2 months

after collection, and preserved their viability for 2 to 2½ years, while some samples were still viable after 5½ years [28].

There are no clear characteristics whereby *Ustilago avenae* and *U. kolleri* can be distinguished on taxonomic grounds; and indeed, it has been suggested that they might well be regarded as 'spiny-' and 'smooth'-spored varieties of the same species [27, 33]. The two hybridise with the greatest of ease, the spiny character being dominant over smooth, in the simple Mendelian ratio of 3 to 1, in segregation [9, 24]. Inoculations performed with mixed sporidia of the two species resulted in the smutted heads being of variable appearance but retaining the characteristics of the loose type, with spiny spores [7]. New races of smut are believed to arise by crossing and by mutation [23]. In 1931 a 'buff' smut of oats in America was found to have arisen as a mutant of *U. kolleri* [9]. This mutant race crossed readily with the two smut species, the buff character proving recessive in inheritance. Some of these hybrids with the mutant were more virulent than either parent; one race produced spore masses entirely covered by the outer glumes, another race completely destroyed the glumes leaving the sori exposed, while other races for the most part partially destroyed the glumes, thus producing smutted panicles of an intermediate character. The spores of the buff smut, like those of *U. kolleri*, remain intact in the grain and are not readily dispersed. This new fungus may perhaps be a variety of *U. kolleri* or possibly a distinct species, and has been found in panicles smutted under natural conditions [10]. Moreover, it is claimed that *U. kolleri* is not distinct morphologically from *U. hordei*, causing covered smut of barley, and it is suggested that the two should be regarded as specialised varieties of a morphologic species, priority being given to *U. hordei* [4a].

Several authors report the existence of physiologic races of *U. kolleri* [3, 4, 26, 27, 30, 34]. One race attacks the well-known Fulghum oats, which, with the variety Red Rustproof (grown extensively in the southern half of the United States), is stated to be resistant to loose smut, but so far no race of *U. kolleri* has broken down the resistance of Red Rustproof [26]. In Wales two races were found on the variety *Avena strigosa orcadensis*; one of them was highly virulent on this host, which was, however, highly resistant to the second race, but the latter caused complete infection of the 'potato' oats, a variety of *A. sativa* [30]; and collections of *U. avenae* in the United States produced smutted ears on the normally resistant Victoria and Black Mesdag [26a, 35]. The varieties Victoria, Markton, Navarro, and Bond are highly resistant under most conditions in the United States [13].

The same physiological relations discussed in connection with loose smut appear also to apply to the parasitism of covered smut.

Both diseases are controlled by seed treatment. Some prefer formaldehyde dust as a safer treatment than the liquid formalin, owing to deterioration in keep. Good results are reported from the use of certain volatile mercury compounds such as ethyl-mercuric chloride or phosphate; the latter has the added advantage of checking other diseases of oats besides smut [21].

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Crown Rust of Oats, *Puccinia coronata* Corda

Crown rust is very common on oats, but does not occur on wheat, barley, or rye. It is, however, very frequent on grasses on some of which it is specialised. On rye grass (*Lolium perenne*) and meadow foxtail (*Alopecurus pratensis*) it causes appreciable damage in the aftermath, resulting in considerable defoliation and rendering the herbage unpalatable to stock [25]. Crown rust attacks mainly the leaves and leaf stalks, less frequently the stem. It is caused by *Puccinia coronata* [15], a heteroecious fungus which develops its uredospores and teleutospores on the graminaceous host, and spermagonia and aecidiospores on the shrubby buckthorn (*Rhamnus*) (Figs. 207, 208). In Britain, especially in the west and north, oats suffer heavily from this disease and very poor yields are obtained. In the United States crown rust is responsible for greater aggregate losses than stem rust (*P. graminis*) on oats, especially in the south ; in New York State it is the most destructive rust of cereals and causes a loss on the oat crop exceeded only by the smut diseases [14]. In Scotland, in 1933 losses were particularly severe [8a], but in later years they have not been so heavy.

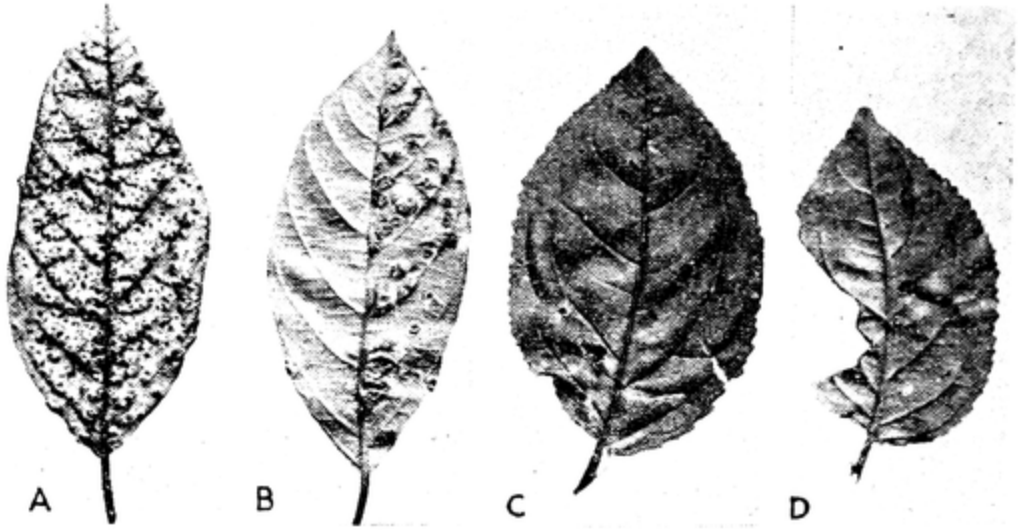


FIG. 207.—Crown rust (*Puccinia coronata*). On the aecidial hosts. *A*, *B*, on *Rhamnus frangula*. *A*, upper leaf surface showing numerous pustules. *B*, under surface showing aecidia. *C*, *D*, on *Rhamnus cathartica*. *C*, upper surface showing shrivelled infection pustules with abortive spermagonia. *D*, the same, under surface (photo by Brown (Gilsen), *Ann. App. Biol.*)

The orange-coloured uredosori, irregularly scattered on both sides of the leaf, are small, oval and sometimes confluent. The uredospores are globose to obovate, echinulate, and measure from 18 to 27 by 16 to 24 μ (Fig. 209 A). The teleutospores (Fig. 209 B) on leaf blade or sheath are covered by the epidermis for a long time ; they may be found concentrically arranged around the uredosori, or in inconspicuous raised streaks over which the epidermis remains unbroken [26]. There are numerous factors which appear to control the production of teleutospores in this rust. Different strains of the fungus vary in their capacity to produce them [18], and while some authors state that they appear independently of the resistance or susceptibility of a particular variety of oats [20], others say that the teleutospores develop earlier on a more or less resistant variety than on a susceptible one ; and some strains of the fungus produce them only on resistant plants, while other strains appear not to produce them at all [18, 19]. The teleutospores are brown and smooth, and have the upper cell considerably broadened out and flattened at the top where there are 5 to 7 dark-coloured, finger-like processes forming a ' crown ' to the spore ; the spores measure from 35 to 60 by 12 to 22 μ, and are borne on rather short, thick pedicels.

The aecidial stage, which appears on the buckthorn in May and June, follows upon infection with the sporidia produced by germinating teleutospores which have survived the winter on oat-straw or susceptible grasses. Freezing appears to assist their germination [3, 13]. On the lower surface of the buckthorn leaf, or on the petiole, yellow or purplish spots are formed, scattered or in groups, and the infected parts may show considerable distortion of growth. The aecidium which finally projects through the broken tissues is furnished with a white cylindrical peridium, torn and revolute at the margin ; the aecidiospores are delicately verrucose, orange in colour, sub-globose, and measure from 16 to 25 by 15 to 20 μ ; spermagonia are found on the upper surface of the leaf or amongst the aecidia [1]. If infections on the buckthorn are particularly heavy, aecidia may also sometimes appear on the young twigs [12].

The two species of buckthorn most commonly affected are *Rhamnus frangula* and *R. cathartica*, but *R. lanceolata* [16] and *R. alnifolia* [26] and a few other unimportant species [7, 17] are also susceptible. An unrelated host *Lepargyrea cana-*

densis of the family *Elaeagnaceae* also acts as an alternate host to that form of the rust which is found on the grass *Calamagrostis purpurescens* [8, 10]. Both the *frangula* and *cathartica* species of the buckthorn are reported in America to be susceptible; of the seven races of *P. coronata* in Britain, all except one infect one or other of the two buckthorns, not both [3]. These varieties are distinguished by their effects upon particular graminaceous hosts; the race on *Lolium perenne* is designated *P. coronata* var. *lolii*, the one on *Holcus lanatus*, *P. coronata* var. *holci*. Of the form on oats *P. coronata* var. *avenae* there are numerous physiologic races [2, 4]. In Britain it is not usual for any of the forms of this rust on the grass hosts to affect oats, and the majority of grasses are resistant to the form on oats, so that, in general, there is little to fear that infection of oats may occur from grasses infected with crown rust [3].



FIG. 208.—*Puccinia coronata*. Section of leaf of *Rhamnus frangula* showing normal spermagonia and leaf infection (×170) (photo by Brown (Gilsen), *Ann. App. Biol.*)

From experiments conducted in south Russia [11], the critical period for infection of oats with the uredospores was found to occur with the formation of the ear and up to the 'milky' condition in the development of the grain. Recent experiments in Louisiana [13a] have established that the parasitism of the leaf cells of oats by this rust depends on the liberation by the host cells of phosphoric compounds into the intercellular spaces for the nutrition of the fungus [13a]. Leaf infection is stomatal and penetration is preceded by the formation of appressoria; resistant and susceptible hosts are penetrated in the same way, and while there is very little hindrance to the progress of the fungus in susceptible leaves, in resistant hosts a reaction early sets in, and after the death of the haustoria and of the host cells invaded by them, the fungus makes no further headway [23].



FIG. 209.—*Puccinia coronata*. *A*, uredospores (×375). *B*, teleutospores (×500) (after Erikkson & Henning)

Primary infections and greater intensity of attack occur chiefly during rainy periods and are favoured by comparatively low temperatures, but the progress of the fungus in the host may continue at higher temperatures. Observations in Louisiana [9] showed that crown rust is quite prevalent from midwinter until the oats mature, and that uredospores can withstand fairly high summer temperatures. Their viability appears to

be reduced by exposure to light, this being lost within 23 days, whereas the spores were germinable after being kept in the dark for as long as 79 days [13]. Experiments in Arkansas [22] showed that at temperatures of 5 to 10° C. and a humidity value of 25 to 50 per cent., the uredospores remained viable for a whole year, while at lower humidities of 15 to 25 per cent. the spores lived only for 6 months; at temperatures above 15° C., the spores died at all humidities, and it appears that for particular localities and probably for many of the important oat areas in the United States, uredospores are of little concern in the perpetuation of this rust over the winter; in the northern areas the buckthorn provides for the primary infections, but in the southern area the rust appears to be carried over by uredospores which have survived the winter, and it is also probable that uredospores are conveyed from northern to southern areas by wind. Experiments in Winnipeg [21] have proved the importance of the influence of temperature upon reactions to crown rust shown by selected oat varieties to certain physiologic races of the fungus. While some races developed normally at all temperatures tried, on all the differential hosts, other races differed in the time of appearance of, or failure to develop, teleutospores, according to temperature [21].

Rhamnus bushes are not common in Scotland, and there is evidence that uredospores do not remain viable over the winter, so that the rust is probably brought there by wind-blown spores from England or Ireland [8a].

Although the systematic eradication of the buckthorn in the localities where it occurs would no doubt help to reduce the incidence of crown rust, in other areas where the uredospores are capable of carrying the rust over from one season to the next, the only hope of control lies in the production of resistant varieties. It must be borne in mind that it is often desirable in practice to develop varieties of the host that will prove to be resistant to one or more rusts, as here, to both stem and crown rusts, for in many localities it has been clearly demonstrated that there are varieties of oats resistant to stem rust which are susceptible to crown rust, and to the devastating smut diseases. Experiments conducted in Wales [5, 6] showed that crosses between selections of the variety Red Rustproof (*Avena sterilis*) and the highly susceptible Scotch Potato oats (*A. sativa*) infected with a strain of the rust on the latter variety, yielded two distinct groups, resistant and susceptible, and the segregation in the F_2 generation gave clear indication that resistance behaved as a simple dominant.

It may be stated, in general, that varieties of oats which mature early may actually escape heavy infection with crown rust. It has been observed in Wales [24] that the comparatively slow-growing and late-ripening varieties Goldfinder and Welsh Strigosa are usually severely diseased while Black Tartar, Ceirch du bâch, and Radnorship are amongst the least susceptible, and this despite the comparative lateness of the first two varieties; the variety American Sixty Day, one of rapid growth and early ripening, also gives good results in Wales. In the northern areas of the United States the varieties least affected by crown rust are Green Mountain, Red Rustproof, Iowar, Burt, and Richland [14, 26].

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Leaf Stripe and Seedling Blight of Oats, *Pyrenophora avenae* Ito & Kuribay (= *Helminthosporium avenae*) Eidam

Leaf stripe or leaf spot and seedling blight of oats is common and destructive in the wetter, northern and western areas of the British Isles, where oats is the principal cereal cultivated. In the United States it is not considered to be a serious disease of oats, but in Canada, Germany, Denmark, Holland, Belgium, and Italy it is reported to account for heavy losses in yield ; it also occurs in Japan, India, and South Africa.

The disease is carried by the seed and a pre-emergence stage (blight) causes much distorted growth and destruction of seedlings. There are two distinct phases in its history on the host plant. The primary, the leaf-stripe stage, involves the seedling and the young plant until the fourth or the fifth leaf is formed, and the secondary, the leaf-spot phase, appears on the plant at maturity (Fig. 210); and between these two stages there is a disease-free period of about 4 to 6 weeks [2, 13, 16].

The first leaf of an infected seedling to appear above ground usually shows a yellowish-white tip and very small yellow spots on the leaf blade (Fig. 117 B); the spots soon become elongated parallel to the long axis of the leaf or they may join together to form pale-yellow stripes. These infected parts soon turn brown so that one of the early symptoms in the field is the presence of one or two brownish stripes running from the base of the leaf but usually not reaching as far as the tip. The succeeding second and third leaves repeat the same striped appearance, but by the time the next leaf has unfolded, intensity of infection is feeble, this fourth leaf showing the disease in a much milder form as small brown patches instead of stripes as seen on the leaves below. From now on, infected plants appear to grow

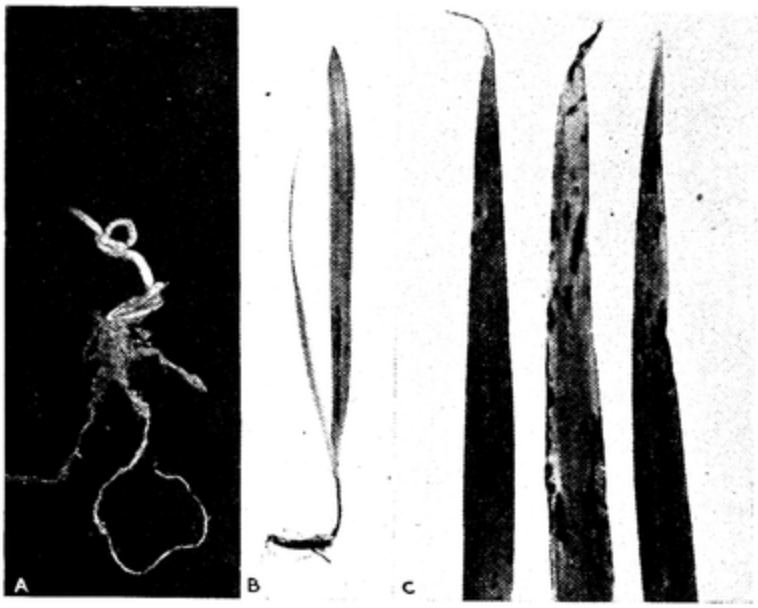


FIG. 210.—Leaf stripe and leaf spot of oats (*Pyrenophora* (*Helminthosporium*) *avenae*). *A*, a diseased, distorted seedling. *B*, the primary leaf-stripe stage. *C*, the secondary spotted stage (photos by Foister & Noble)

normally, the remaining foliage leaves looking healthy and free from disease. With the formation of the fifth and subsequent leaves, and continuing up to the 'green ear' stage, the plants enjoy a disease-free period, as above stated, of some 4 to 6 weeks. At the end of that time, however, diseased spots may be seen to break out on the upper, previously clean leaves, and their appearance heralds the second phase of the disease, that of secondary infection. This phase is not a continuation of the first, for it appears at the same time on entirely healthy plants in the crop, that is, on those which did not show primary infections. These new spots are scattered haphazardly on the fresh leaves as well as on the older leaves on which the original lesions may still be distinctive in definite longitudinal stripes. On plants healthy from the start they are, of course, the only type of infection on all or any of the leaves. The secondary spots are, at first, furnished with a yellow-green or sometimes purple margin surrounding a sunken, brown and dried area, and though these spots may later join and run together in a direction parallel to the length of the leaf, they can be distinguished from the primary stripes by their greater width and irregular contour; there is, moreover, no shredding or splitting of the lamina. The primary stage of this disease is more destructive in its effects than the secondary, for it falls at an early and critical stage in the development of the host, and must inevitably account for much general debility leading to a thin stand of seedlings [2, 7]. The second phase, while not causing appreciable crop injury, is of the character of leaf damage to foliage already mature, but is nevertheless important as it is responsible for the infection of the ear and grain [2].

The fungus which causes this disease of oats is better known by the name of the conidial stage of reproduction, *Helminthosporium avenae* [4]; the perithecial stage, *Pyrenophora avenae* [3, 9] is of rare occurrence; pycnidia of unknown function also occur in the life-history of this organism. The brown conidiophores arise on the dead discoloured spots on the leaves, singly or in groups of two or three, usually passing out between epidermal cells. The first conidium is terminal, and others arise at intervals in sympodial fashion along the septated conidiophore, at points indicated by small swellings or knee-joints (Figs. 120, 211 F). The conidia are cylindrical, at first light-coloured, becoming yellow when older; septa vary from 0 to 12 (usually 3 to 8) in number, and the basal cell is marked by a black spot or hilum; germ-tubes may be produced by any of the cells. Spores measurements given are, from 17·5 to 147 by 8·25 to 28·25 μ [2] and from 15 to 55

by 13 to 18 μ [21]. On oat- or maize-meal agar, at 25° C., a very characteristic white, cottony, tufty growth arises from a greyish aerial mycelium, the submerged portion being very dark, almost black, but different strains of the organism vary widely in appearance in culture. The young mycelium is thin-walled, colourless, septated, and multinucleate, and occasional anastomoses between hyphae may occur (Figs. 115, 118). Saltations are of frequent occurrence [2]. Conidia are produced sparingly on nutrient media but abundantly in tap-water agar [1]. Sporulation was enhanced in cultures exposed for brief

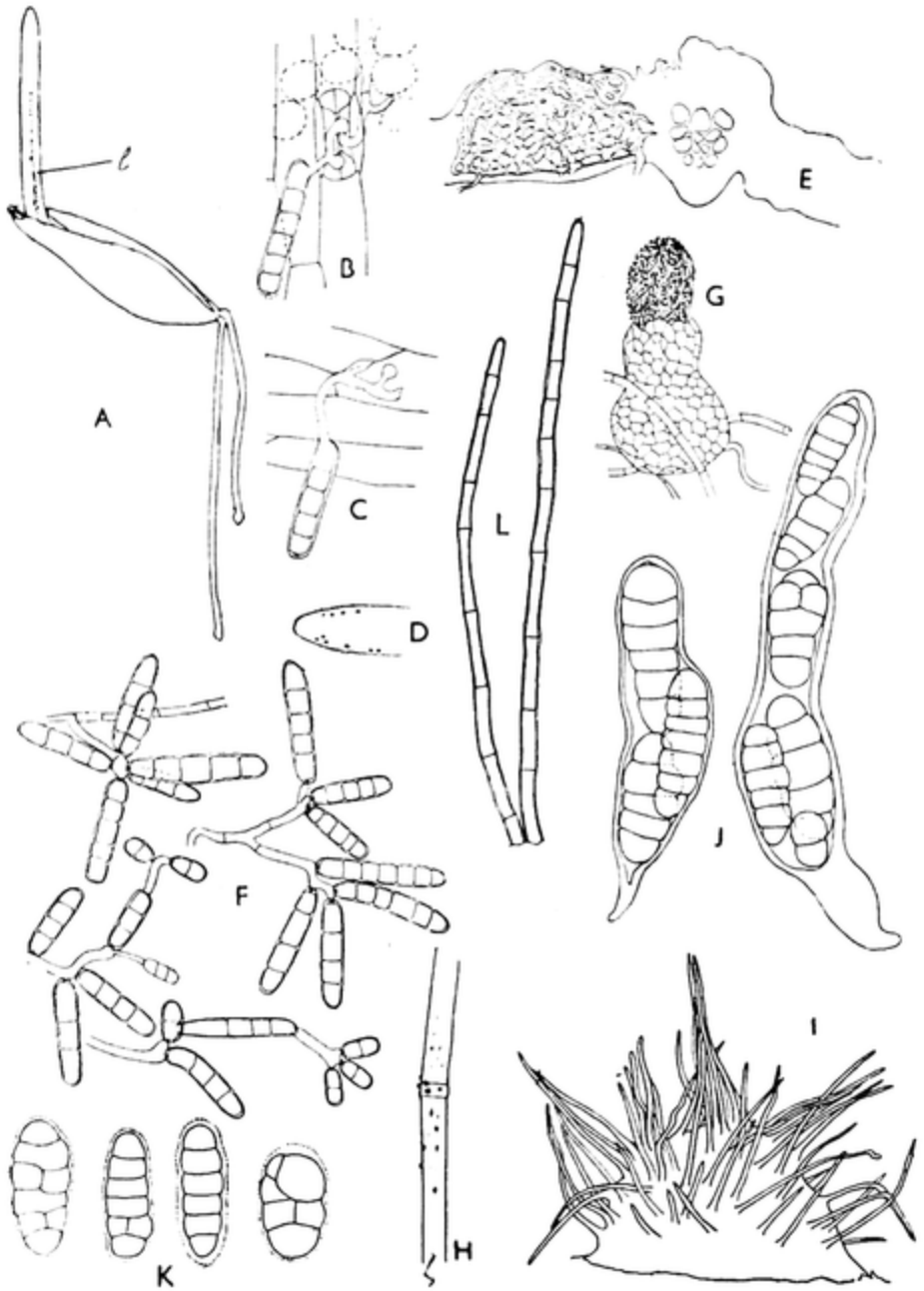


FIG. 211.—*Pyrenophora* (*Helminthosporium*) *avenae*. *A*, seedling infection, *l*, lesion on the coleoptile. *B*, *C*, penetration of oat leaves by germ-tubes, stomatal and cuticular, respectively. *D*, sclerotia on inner pale of the grain. *E*, section of a sclerotium. *F*, conidiophores and conidia (× 190). *G*, pycnidium (× 130). *H*, oat stubble bearing perithecia. *I*, immature perithecium from stubble (× 45). *J*, two asci (× 190). *K*, four ascospores (× 190). *L*, two setae from perithecium (× 190) (after Dennis, *Trans. Brit. Myc. Soc.*)

periods to white light of high intensity [8, 9] and on infected grains submitted to a 20 minutes' irradiation after an incubation of three days at 22° C. [12]. As the cultures get older, the submerged hyphae become converted into short segments resembling chlamydospores (Fig. 11 B) [1, 8]. The optimum temperature for growth is 30° C., the minimum being about 2° to 3° C.; actively growing mycelium resists temperatures below the freezing point (− 4 to − 8° C.) [2], and there is a record of as low a temperature as − 14° C. being tolerated for 56 days [17].

Resting mycelium may be found at the surface of the grain mostly at the tips of the pales and within the pericarp. On the pales it is brownish and consists of thick-walled hyphae; at intervals on the surface of the husk the resting mycelium forms small scales of cells resembling sclerotia (Fig. 115). True sclerotia may, however, be found in the field, as small black bodies embedded in leaves lying on the ground after harvest [2]. In addition to sclerotia, from which they are hardly distinguishable, small black pycnidia may be found on the chaff of the dry grain [22] and they have also been observed in culture (Fig. 211 G) [2]. They contain minute unicellular pycnospores, measuring from 2 to 3 by 2 μ, which have not been seen to germinate and their function is not known; in structure the pycnidia closely resemble those of *H. teres* (p. 443). The perithecial stage *Pyrenophora avenae* (Fig. 211 H, I), discovered independently in Germany [17] and Japan [9], is rare both in culture and under natural conditions, and although there are important differences between the descriptions from these two sources (the first, discovered in culture, and the second, naturally, on grain and stubble of oats), a description of the perithecia discovered by Dennis [3] in 1934 on stubble of King's Oat in Scotland agrees closely with that of the Japanese form. They are described as being sub-epidermal at first, later becoming erumpent, semi-globose when young, later flask-shaped or conical, with short parabloid or cylindrical ostiolar beaks; the blackish-brown wall is beset with long conidiophores and setae (Fig. 211 L). The perithecia measure from 300 to 600 μ in height, from 450 to 800 μ in long diameter, and from 350 to 700 μ in short diameter. The fasciculate-clavate or cylindrical, hyaline asci are often slightly curved, rounded at the apex, slightly stalked, and measure from 250 to 400 by 35 to 40 μ; they contain 2 to 8 (mostly 8) pale-yellowish or yellowish-brown, ellipsoid or oval ascospores furnished with 3 to 6 (mostly 5) transverse septa, sometimes with 1 to 4 vertical septa; the spores measure from 50 to 75 by 17·5 to 30 (Fig. 211 J, K) [9].

The disease is seed-borne, and the most important source of infection is the resting mycelium on the grain (Fig. 115 *fm*). The fungus is not believed to be capable of over-wintering in the soil, and it appears to be a weak competitor with the normal micro-organisms of the soil [15]. The conidia carried over on the grain have proved to be viable for at least 6 months, but they are relatively scarce and are probably of little importance in starting fresh infections [2]. The primary stage of infection is therefore believed to start when the resting mycelium becomes active at the germination of the seed. Penetration takes place at the outer surface of the coleoptile (Figs. 116, 117, 118, 119), and as the history of these early infections appears to follow the same sequence of events as that outlined in the parasitism of *H. gramineum* [19, 20], the reader is referred to the account of the stripe disease of barley caused by that organism (p. 431). Infection progresses from leaf to leaf in the same way, but in this disease comes to a halt at the fourth leaf, and henceforth leaf stripe of oats is unlike that of barley, for in the latter the fungus concerned continues unabated on its career until all the leaves and the ear are

reached and infected. Oat stripe disease, unlike the stripe disease of barley, includes a secondary phase which, as already mentioned, follows upon the primary after an interval of some 4 to 6 weeks during which the upper leaves expand and remain healthy. These secondary infections are definitely known to be due to conidia carried by wind from the primary lesions (Fig. 120) and occur on the leaves of healthy plants as well as those of plants already infected. Penetrations may be direct or stomatal, and infections soon appear as dull brown spots each with a yellow margin fading into the normal green colour of the leaf; a purplish line replacing the yellow indicates that the infection has been early checked, a phenomenon that would arise probably if the host attacked was more or less resistant, or as a result of reaction against a different strain of the parasite [2].

The freedom from disease enjoyed by infected plants from the termination of the primary until the onset of the secondary attacks is greatly favoured by a dry environment, such a condition being entirely detrimental to the germination of the conidia and infection of the leaves. With the additional growth attained during the interval, however, the foliage throughout the crop is rendered much more compact, and there is thus established a microclimate of higher humidity than before, which is conducive to the success of secondary infections; plants kept dry in exposed situations in the field or in comparative isolation show but little signs of infection beyond the primary seedling phase [2].

The influence of the temperature factor on the incidence of this disease cannot be considered apart from that of soil moisture, for despite the fact that low temperatures are favourable to the fungus in wet soil, the disease is also aggravated under higher temperatures, but only under conditions of relative drought, and in these circumstances soil moisture behaves as a limiting factor. The primary phase of this disease is of much greater importance in the colder and wetter parts of Britain than in the warmer and drier areas, a fact which indicates the importance of soil temperatures in the initial stages of the disease [11].

The secondary phase of the disease, in Britain, is at its height in late July and August, and culminates in the infection of the grain at the 'green-ear' stage. The wind-borne conidia may be found clinging to the short hairs on the pales, from which points of attachment they proceed to form a resting mycelium. It is true that the conidia may remain viable on grain stored from harvest to sowing-time, but it is the resting mycelium and not the over-wintered conidia that is mainly responsible for the primary infections when the grain is sown.

As leaf stripe of oats is carried by the seed the methods of control are on the same lines as those indicated for the stripe disease of barley. Seed disinfection with certain organic compounds of mercury has also proved highly effective in controlling this, as well as other diseases of the oat plant [2, 7, 10, 12, 14] (Fig. 163). No variety of oats is yet known to be immune from leaf-stripe disease, and all varieties are more or less susceptible. It is reported that home-grown seed is unprofitable to use for more than two years in succession and a change of seed is recommended [2].

Another seed-borne disease of oats, attributed to a new species of *Helminthosporium* (named *H. victoriae*), has recently appeared in America. It attacks the

basal parts of the plants, producing necrosis of roots and stem; the straw is rendered weak and there is excessive lodging [23].

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Grey Speck or Grey Leaf of Oats (*non-parasitic*)

Grey speck or grey leaf commonly found to attack oats also affects wheat, barley, timothy, and other fodder grasses, adversely influencing the quality of the grain or hay as the case may be. It is not attributed to any plant pathogen, and is believed to be a ' deficiency ' trouble due to the lack of manganese in the soil. Apparently it is not due so much to the complete absence of this mineral, for such a condition would probably be of rare occurrence in any soil, as to some factor or factors which render the manganese unavailable to the plant.

Grey speck is well known in Britain and has been reported from Holland, Norway, Sweden, Denmark, and Germany. Its first discovery in Britain appears to have been made in Scotland, about 1913 [29]; in 1931 it was reported in Wales on many varieties of oats, doing considerable damage in the early parts of the season [8]; it was recorded again in 1932 to be somewhat serious in certain parts of Yorkshire, to such an extent that oat crops failed almost completely [33]. The trouble was noticed in Canada in 1924 [1] but its appearance in the United States is comparatively recent [37]. The disease has been closely studied in Australia, the first case, reported in 1922, described as ' white wilt ' of oats, coming from Dwarda in Western Australia; six years later the trouble was discovered in South Australia [25], where it was popularly called ' roadside take-all ', but grey speck has no connection with the more serious fungal trouble ' take-all ' or ' whiteheads ' (see p. 377) which is also prevalent in certain parts of Australia and elsewhere. In the lower, south-eastern districts of Australia in 1932 barley was also found to suffer from a disease similar to grey speck of oats, but not nearly to the same extent as oats [25].

The earliest investigation of grey speck disease seems to have been made in Holland, where the trouble known as Dutch oat disease was attributed to the use of certain artificial manures [16]. In general, affected oats in the field are quite normal in the early stages of growth, but later the plants turn a pale-yellowish colour and soon develop small spots of a greenish-grey hue scattered over the leaves but with a tendency sometimes to collect towards the margins, thus giving a somewhat striped effect (Fig. 212). The weakened leaves soon droop and develop a crack at the fold, but while the tips of the blades may remain green and unspotted, the middle parts of the laminae are usually spotted all over. Further extensive work on the disease in South Australia [25, 26], giving details of the early symptoms in the field, states that during young stages of growth affected crops can usually be recognised from a distance by their lighter green colour than that of the normal crop, with an admixture of brown due to the death of the older leaves, the actual effect being a weakening of the plants rather than that of killing them outright. In Wales [8] the trouble was found to break out in comparatively small areas during the growing season, affected plants, stunted and yellowish, forming conspicuous bleached patches in the young crop. In this area the first symptoms appeared when the seedlings were from 4 to 8 inches high, and occurred, not on the first seedling leaf, which remained green, but on the succeeding leaves, which developed a yellow-green colour. In Western Australia it was noticed that the first leaf to become spotted was the third, followed by the second and the fourth, and then the first and the fifth, and after that in the order of their appearance [6]. Affected

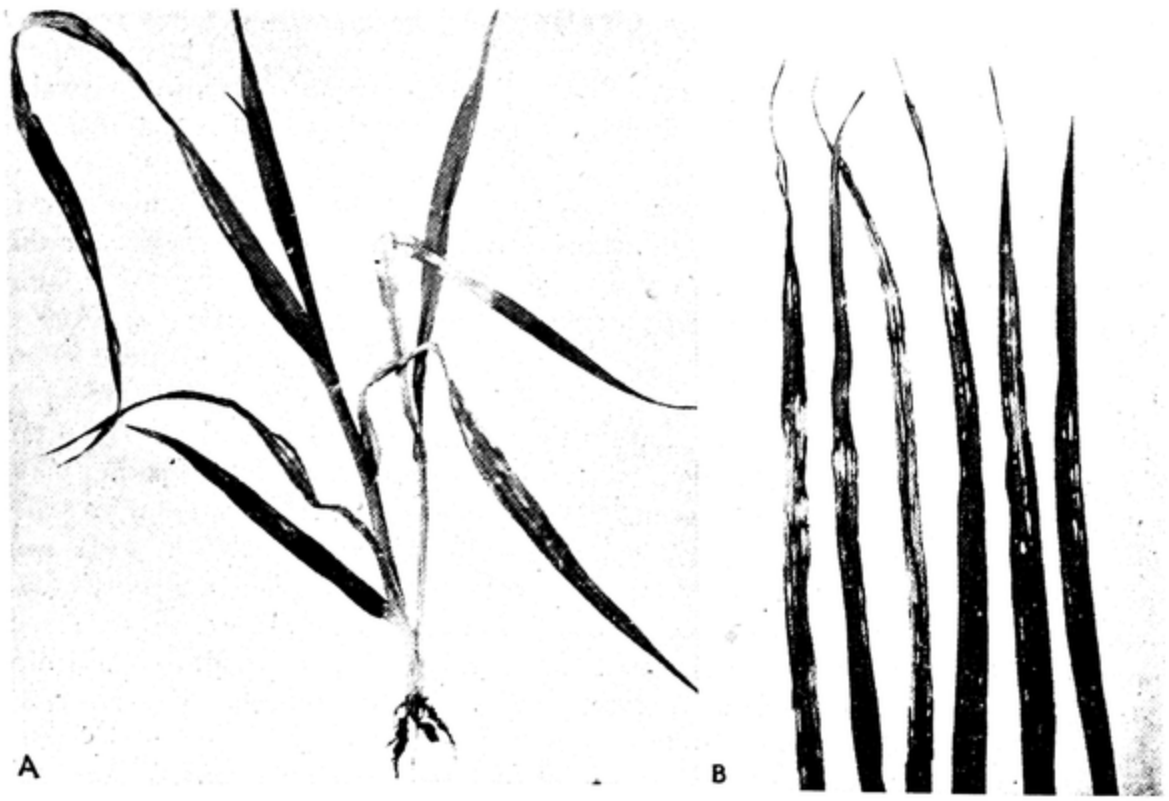


FIG. 212.—Grey leaf, or grey speck of oats. *A*, on a young plant. *B*, on older leaves (photos by McKay)

spots have a light-pinkish tinge at first but soon turn grey at the centre, at the same time developing a purplish margin, but the latter is not always present; the spots may sometimes join together to form long streaks in between the veins, or may extend from margin to margin right across the middle part of the lamina in the form of a greyish-coloured band, while the parts at the top and base of the leaf remain green, but the whole leaf eventually turns grey and dies [6, 37].

Grey speck of oats has long been known to occur chiefly on certain types of alkaline soils [28] and the application of a corrective, in the form of manganese sulphate, to the soil was first made in 1914, and carefully controlled water-culture experiments from which the minutest traces of manganese had been removed have shown that in the absence of this mineral the experimental plants repeated in every detail the characteristic symptoms of grey speck in the field [25, 26].

The absence of visible symptoms of the trouble during the early stages of growth is presumably due to a certain amount of manganese stored in the seed, this being sufficient to provide for the healthy appearance, at least, of the first one or two leaves of the seedling. But when this store is exhausted the future well-being of the plant will depend on the availability of manganese compounds in the soil. It is noteworthy that oats, in comparison with other cereals and with many other plants, demands a fairly high proportion of manganese. Thus, the comparative demands for manganese as shown by the relative dry weights of a number of plants, at the end of a series of water-culture experiments in which manganese sulphate was supplied at the rate of one part per million, were, for oats 185, wheat 150, barley 101, rye 86, broad beans 87, and peas 46 grams. In a solution of one part in ten million no grey speck symptoms appeared, and to satisfy the needs of an average oat plant it is calculated that about one part of manganese in from one to five million parts of solution is the optimum for growth in water cultures, one or two changes being made during growth until the plants reach fruition [26].

Several theories have been advanced to explain the conditions under which manganese in the soil is rendered unavailable to the plant and to account for the characteristic symptoms of grey speck disease. While some assert that the trouble is due to a disproportionate quantity of lime in the soil as compared with other minerals, thus upsetting the balance of the mineral content and resulting in an abnormal absorption of certain salts [2, 3, 19, 20], others maintain that an excess of calcium ions does not necessarily render the manganese unavailable if the latter is present in the soluble and highly ionised form of sulphate [25, 26]. Another view of the problem is based on the capacity of the roots to absorb the mineral, in relation to the activity of certain bacterial organisms in the soil [11, 12]. In a soil deficient in manganese it is stated that the roots of the oat plant develop badly, soon becoming decayed at the root tips, and under such conditions are rendered highly susceptible to bacterial infection. It would thus appear that a relationship exists between the resistance of the host and the manganese content of the soil. Moreover, a poorly developed rooting system, with a number of decaying roots and early destruction of root hairs, is one of the most striking features of serious manganese deficiency. It is interesting to note that on a piece of ground where a fire had been made (as by burning a pile of bracken) oat plants grown on the burnt

area grew perfectly healthy even though the surrounding crop was badly diseased. But the good effects of the burning did not last long, the probable explanation being that the temporary benefit was due to the killing of the harmful bacteria in the burnt patch; the effect was not permanent, because the organisms from the surrounding soil would in a short time repopulate the area and the good effects would pass off. Whether such sterilisation of the soil reduced the amount of disease by destroying the bacteria, or whether it had an indirect effect in releasing more manganese from the soil, is not clear, for the application of a germicide, such as formalin, to the soil in some cases [25, 26] did not check the trouble, whereas in other cases [12] satisfactory results with this germicide were obtained provided its vaporising properties were not dissipated too soon [12, 32].

In a series of water cultures, 3 weeks old, lacking in manganese, it was estimated that a mean population of 217,000 bacteria were found in the root tips; this number was reduced to 44,500 when manganese was added; in 9 weeks the bacterial number in the former case had increased to 1,351,000, and in the latter to 316,000. Harmful bacteria, by attacking an already weakened root system and by feeding on the decomposable material of the root tips, are enabled the more easily to convert nitrates to nitrites. Such conditions are favourable to an abundant synthesis of proteins in the root tips, and there follows an increased production of ammonia by micro-organisms. Actually, in a number of tests the ammonium content of a plant diseased through grey speck was found to be two or three times that of a normal plant. This increment is thought to be due not only to the ammonia produced as a result of bacterial activity in the diseased root tips, but partly also to the breaking down of elaborated contents in consequence of extreme carbohydrate hunger following upon enfeebled photosynthetic activity [11, 12]. It is, indeed, asserted that the primary cause of the whole trouble of grey speck lies in the shortage of sugar in the leaves, the effect being an immediate lowering of the resistance of the plant [39]. The action of the mineral, moreover, is stated to be that of a stimulant or a catalyst having an indirect effect on the photosynthetic activities of the plant [13, 14, 15].

It is well known that the quantity of oxidases in the roots is increased in the presence of manganese, the effect being most evident in the early stages of growth [4]. Unaware of the possible rôle of soil organisms in relation to grey speck disease, some observers found that the incidence of the trouble generally coincided with the presence of the nitrite ion in the roots, and perhaps also in the leaves, the application of manganese, by increasing the quantity of oxidases in the roots, having the effect of decreasing the nitrite content [9].

The presence of ammonia in the plant, by affecting the reaction of the cell sap, is believed to account for the symptoms of grey speck in the leaves. Apparently the transference of alkaline products by the sap causes a necrosis of the cells between the veins, extending at first nearly to the top and to the base of the leaf, and finally ending in the death of the leaf [11, 12].

While there is general agreement that grey speck is aggravated on alkaline soils, frequently with high organic content, not all such soils produce the full symptoms of the disease. The amount of exchangeable or soluble manganese ('manganese value') in the soil is regulated by the reaction of the soil; this

' value ' is not only influenced by the conditions of oxidation and reduction but increases as the *p*H of the soil decreases [31]. The application of acid fertilisers is a well-known treatment in the control of manganese deficiency, but it is by no means clear how soil reaction influences the availability of manganese compounds. It is suggested that the beneficial action of acid fertilisers may not be due so much to a change of the *p*H value as to the fact that, at the lower values of soil reaction, insoluble manganese compounds such as manganic oxides are reduced to soluble forms by the interaction of certain micro-organisms [30]. It is further stated that the precipitation of insoluble manganic substances in the soil is brought about by specific micro-organisms, between *p*H limits of 6·5 and 7·8, and these limits appear to coincide with values obtained in the field where grey speck is more frequent [11].

It is clear that grey speck disease of oats is not to be attributed to manganese deficiency alone, and that it must be considered in relation to the activities of certain bacteria in the soil. Some bacteria appear to have the property of precipitating manganese, thus rendering the mineral unavailable to the plant roots, and the debilitated roots, in turn, are probably attacked by another set of harmful bacteria. Experiments with cultures have established that with a minimum manganese content of from 5 to 35 parts per million, the plants remain healthy provided the roots are kept sterile, but when a sterile culture is infected with a few diseased root tips or with a mixed culture of bacteria isolated from them, the symptoms of grey speck soon appear [11]. Furthermore, in mixed bacterial cultures, containing manganese sulphate, some of the bacterial colonies were found to contain manganic oxides presumably precipitated by the appropriate organisms, but as soon as the reaction of the medium became more acid these substances were dissolved, possibly by the metabolic products of other organisms in the mixed cultures.

Good control of grey speck on oats in Wales was obtained with a dressing of 3·3 oz. of manganese sulphate in 2 gallons of water, applied with a watering-can 3 days before sowing, and the operation repeated 10 days after sowing ; in respect of the total weight of the harvest, an improvement of 50 per cent. was obtained in the weight of both straw and grain [8]. Again, in heavy fallow soils, potassium nitrate or manganese sulphate at the rate of 100 to 200 kg. is also recommended [21]. In a series of trials in Yorkshire, however, a similar treatment gave very disappointing results. While the sulphate did confer some improvement, the beneficial effects were not sustained, and it did not produce a cure, the explanation being that here, again, manganese-precipitating bacteria were in evidence, rendering the soluble sulphate unavailable to the plant [33]. In view of the findings in Wales [8] that there are varying degrees of susceptibility to grey speck among certain oat varieties, it is suggested that the disconcerting results with the mineral treatment in the Yorkshire soils may be connected with the choice of more or less susceptible varieties of the host plant. In Ontario, again, in some years no benefit was found to follow upon the application of manganese sulphate to heavy soils, either with or without sulphur, though in some seasons this treatment appeared to give some measure of control [36]. Others state that none of the various soil treatments gives any permanent control against manganese deficiency [35].

But little appears to be known as to the value and practicability of the so-called

'injection' methods for the mitigation of mineral deficiency diseases in relation to those affecting cereals. It is pointed out [24] that the function of most, if not all, of the elements are quite specific in their effects, and that one element cannot replace another. This has also been discovered in the case of grey speck, and none of the rare elements nor any combination of zinc, copper, boron, or aluminium was able to replace manganese [26].

Trials in Wales [8] showed Scotch Potato oats to be resistant to grey speck, and the varieties Radnorshire Sprig and Ceirch du bâch highly resistant; moderately susceptible varieties were Victory, Record, Black Tartar, and Golden Rain II, while the variety Orion was very susceptible. In a series of trials in Yorkshire in 1932, the variety Golden Rain showed marked susceptibility, and Record, Marvellous, and Supreme some measure of resistance to grey speck disease [33]. In Dublin in 1942 the varieties Potato (Ardee) and *Avena strigosa* proved to be virtually immune, while Glasnevin Success 10, Victor II, Ardri, Sonas Potato 7, and Star sustained moderate damage [35].

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Loose Smut of Barley, *Ustilago nuda* (Jens.) Rostr.

Loose smut is an important disease of barley in the United States and Canada [7, 9, 10, 15], but is not common in Britain, where losses from it are not so great as from the covered smut (*U. hordei*) described below. It resembles the loose smut of wheat (*U. tritici*) in all important respects.

In the field, barley plants affected with loose smut may be distinguished from those with covered smut by the earlier extrusion of the smutted heads, which, moreover, are carried up higher out of the leaf-sheath than the heads of healthy plants (Fig. 213). Heads affected with covered smut not only appear several days after those of loose smut but they are behind the healthy heads as well, in comparison with which they are also shorter [10].

The spore masses of *Ustilago nuda*, the fungus of loose smut, are not retained within the silvery membranes of the grain as are those of covered smut, but are set free at maturity as soon as the covering is broken, and after dispersal by wind nothing remains of the ear but the bare stalk.



FIG. 213.—Loose smut of barley (*Ustilago nuda*) on the two plants on left. Covered smut of barley (*Ustilago hordei*) on the two plants on right (photos by Dillon Weston)

Early symptoms of loose smut, before the spikes appear, may sometimes be detected by a discoloration of the leaves, which is yellowish-green to yellow on winter barley, but ochre to brownish on summer barley; and the awns are poorly developed if not entirely destroyed [6].

The spores of *U. nuda* are olive-brown, paler on one side than the other, finely echinulate, and almost spherical, measuring from 5 to 9 μ in diameter. At the time of pollination they are blown to healthy barley flowers and germinate on the stigmas to produce four-celled basidia, which, however, do not form sporidia (Fig. 214 B). Infection of the ovary follows on the same lines as described for *U. tritici* [3, 16] (p. 368). There are numerous races of *U. nuda*; as already mentioned (p. 368), it has been suggested that morphological differences between *U. tritici* and *U. nuda* are not sufficient to justify their separation into distinct species and that they should be considered merely as specialised varieties of a morphologic species, priority being given to *U. tritici* [6 a, 17, 30]. This species, like *U. tritici*, possesses haploid strains of different sex which exhibit cultural differences; after pairing of compatible strains a dikaryotic mycelium becomes established which in some cases has been seen to differ in certain features from those presented by either of the haplonts [28].

The optimum temperature for the germination of the spores is between 20° and 22° C. [15]. As for wheat smut, the amount of infection produced by loose smut of barley becomes greatly reduced during periods of low atmospheric humidities at flowering time. But a low rather than a moderate degree of soil-moisture encourages the development of this disease within the plant [13]. Infection does not appear to be greatly influenced by any differences in the composition of the soil, and though more smutted barley may be found sometimes on acid than on basic soils, this does not occur in all cases [11].

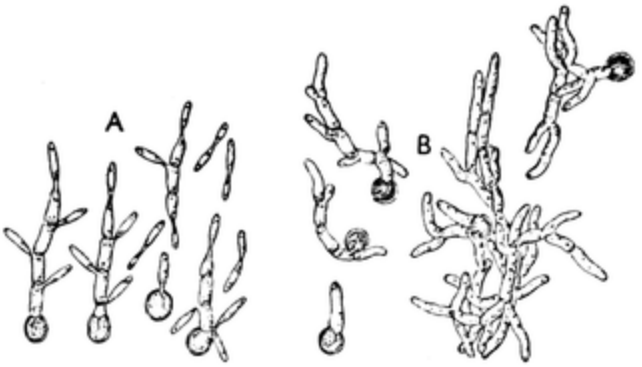


FIG. 214.—*Ustilago hordei*. *A*, germination of the spores, forming promycelia and sporidia (× 230). *B*, *Ustilago nuda*, showing spore germination without formation of sporidia (× 230) (after Brefeld)

Seed barley suspected of disease should have the same hot-water treatment as prescribed for loose smut of wheat (p. 371). But as barley is more sensitive to heat than the other cereals, the temperature of the water should not exceed 126° F., the grain being allowed to steep for 15 minutes, after which it is taken out to dry and sown as soon as possible.

Covered Smut of Barley, *Ustilago hordei* (Pers.) Lagerh.

As above stated, covered smut of barley is more important in Britain than the loose smut. In the United States the average loss through this disease is estimated at 2¼ million bushels of barley annually and in some years has exceeded twice this amount [25].

Covered smut does not become evident in the field until the ear is formed, and affected ears may emerge about the same time as healthy ears but remain shorter, and are usually retained within the sheath for a longer time before appearing, or may sometimes fail to emerge at all (Fig. 213). Unlike the loose smut of the same host, the black spore masses of this smut are held together more or less firmly by the persistent membranes of the grain and the basal parts of the glumes ; the awns also remain intact, though they may sometimes be found partly withered. The spore masses are said to be held together by a deposit of fat, a feature which renders the smutted grain difficult to treat for control by seed immersion, until the fat is removed [20].

Covered smut of barley is caused by *Ustilago hordei*, of which several physiologic races exist [26]. Apparently only a few of these races have a wide geographical distribution [4, 5]. Some varieties of barley are resistant to some of these races, thus, *Hordeum deficiens* and *H. intermedium* and a strain of the variety Pannier were found to be highly resistant to, or immune from, numerous races of the pathogen [26].

The spores are round to ellipsoid, 6 to 9, or sometimes up to 11 μ, in diameter ; they are brown (black in the mass), lighter on one side than the other, and quite smooth. They differ in their mode of germination from the spores of *U. nuda*, by the septated basidium giving rise to sporidia which develop near the septa (Fig. 214 A). The sporidia are uninucleate and, by budding, form fresh spores which develop an extensively branched

mycelium ; intersporidial anastomoses have also been observed, the nucleus passing from one sporidium to the other [32]. Such fusions, as well as mycelial anastomoses, are, no doubt, devices to secure a dikaryotic mycelium prior to infection. Growth temperatures of *U. hordei* were determined in Germany to be 5° to 6° C. minimum, 20° C. optimum, and 34° to 35° C. maximum ; differences of light intensity had no influence on the development of the fungus [18].

Owing to the strong retention of the spores within the diseased grains, it is improbable that many spores manage to work loose and so become scattered by wind or other agency as in the case of loose smut, and accordingly blossom infection is not believed to take place here. For removal from the heads, the smut-masses must await the harvest, when smutted and healthy grain are gathered together. During threshing, as in bunt of wheat, the smutted grains are broken up, and so the healthy grain becomes contaminated by contact with the spores set free ; loosened spores and broken smut grains left in sacks or thresher also serve to taint fresh stocks of hitherto clean barley.

The disease starts anew with the sowing of contaminated grain. Infection is systemic and follows on the same lines as those already described for oat smut or bunt of wheat [29]. The degree of infection of the germinating seedling is dependent on the depth of sowing, and on the amount of moisture in the soil. Since the seedling is attacked before emergence, a deeper rather than a shallow planting lengthens the period of susceptibility ; a case is recorded where the proportion of disease was 2·1 to 44·5, and another of 27 to 115 times as much, when barley was planted shallow, at $\frac{1}{2}$ inch depth, and deep, at 3 inches, respectively [27]. As soon as the seed begins to germinate, first pushing out the coleorhiza and root, and then the coleoptile, infection is only possible for a comparatively brief period during which the coleoptile must remain unsplit, for once it has developed so far as to allow the first green leaf to protrude, the chances of penetration are practically over. Accordingly, the longer the coleoptile remains in the soil, by deeper planting, the longer is leaf emergence deferred, and so the period of infectivity is correspondingly increased. Here apparently lies a practical means for reducing the risk of infection, for with the control of the soil water-level by irrigation, germination of the seed by shallow planting in a wet soil enables the plant by virtue of rapid growth to ' escape ' infection, and after the susceptible period is safely passed, a return to drier conditions offers no further risk to infection. (In Egypt, the ' mud-sowing ' method is so effective against another disease, the ' flag smut ' of wheat (it does not occur in Britain), that it is reckoned to be a much better method of checking that disease than any method of seed treatment by disinfectants [8].) The usual treatment for the control of covered smut of barley is the same as that recommended for the control of bunt of wheat or smut of oats ; but if loose smut is also present in the crop the hot water treatment may be necessary as well. In Virginia, steeping the grain in 1 in 320 formaldehyde or 0·25 per cent. germisan or tillantin gave good control [12].

A type of barley smut showing symptoms somewhat intermediate in character between the loose and covered kinds is fairly well distributed in the United States, and is caused by *Ustilago nigra* [1, 22, 24, 25]. So far, it has not been recognised in Britain. Though the symptoms shown by the emergent ears resemble mostly those of loose smut, yet all

variations may be found in the ears affected by *U. nigra*, from stages where the protective membrane is more or less intact, as in the covered smut, to others where it is loose or practically absent, as in the typical loose smut. Infected ears containing the spores of *U. nigra* appear later than those affected with loose smut and last longer, and like the latter the spores, can be carried to the flowers [23]. The outstanding differences are in the colour, which is a deep chocolate-brown, and size of the spores; the spore dimensions are 6·5 to 7 μ in diameter, as against 5·5 to 6 μ for *U. nuda*. Like those of *U. hordei* the septated basidia of *U. nigra* produce sporidia, and can also cause seedling infection when its spores are placed in contact with the grain. Experiments at Wisconsin [11] showed that infection by *U. nigra* was not favoured in wet soils, especially at extremes of temperatures of 5° and 36° C.; a dry soil produced infection at 5°, but between these extremes differences in soil moisture from 30 to 55 per cent. saturation did not appear to affect the incidence of disease. It is noteworthy that from an examination of smuts collected near Geneva, New York, and from other parts of the United States and Canada, in addition to the typical smut fungi of barley, *U. nuda* and *U. hordei*, five intermediate types were found which showed characters common to both these fungi; amongst them were what appeared to be *U. nigra* and another species *U. medians* [19], but it is not clear whether one or both of these forms should be considered as hybrids of the typical smuts or separate species [31].

In Virginia [14] covered smut (*U. hordei*) and black loose smut (*U. nigra*) were controlled by treating the seed with ceresan applied at rate of $\frac{1}{2}$ oz. per bushel of seed.

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Brown Rust of Barley, *Puccinia anomala* Rostr.

Barley, like wheat, is attacked by stem rust (*P. graminis*) and yellow rust (*P. glumarum*), but has its own brown rust (also called leaf, or dwarf, rust) caused by *Puccinia anomala* (= *P. simplex*), which also attacks a few grasses related to barley [9]. Barley is subject to this rust throughout its entire growing period but is attacked chiefly towards the heading stage.

The uredosori (Fig. 215 A) are small, lemon-yellow in colour, and irregularly scattered on both surfaces of the leaves. Uredospores are round or ellipsoid, yellow, 19 to 22 μ in diameter when round, or 22 to 27 by 15 to 20 μ when elongated ; the wall is rather thick, spiny, and is furnished with numerous germ-pores.

The teleutosori (Fig. 215 B) are scattered as small black crusts on the leaves but are somewhat more elongated on the sheaths and stem. The sori are often confluent and remain covered by the epidermis for a long time. The teleutospores are oblong-clavate or pear-shaped, the apex rounded, flattened, or drawn to one side ; the spore wall is thick and usually broader near the apex than lower down. Unicellular (mesospores) and typical bicellular spores may occur in the same sorus. The spores are chestnut brown in colour, smooth-walled, 25 to 54 by 15 to 24 μ in diameter, the single-celled spores being the smallest ; the stalk is short and brownish. The sorus is divided into compartments by clusters of brown paraphyses which spread out and become flattened under the epidermis. These and the single-celled mesospores enable the species to be readily identified.

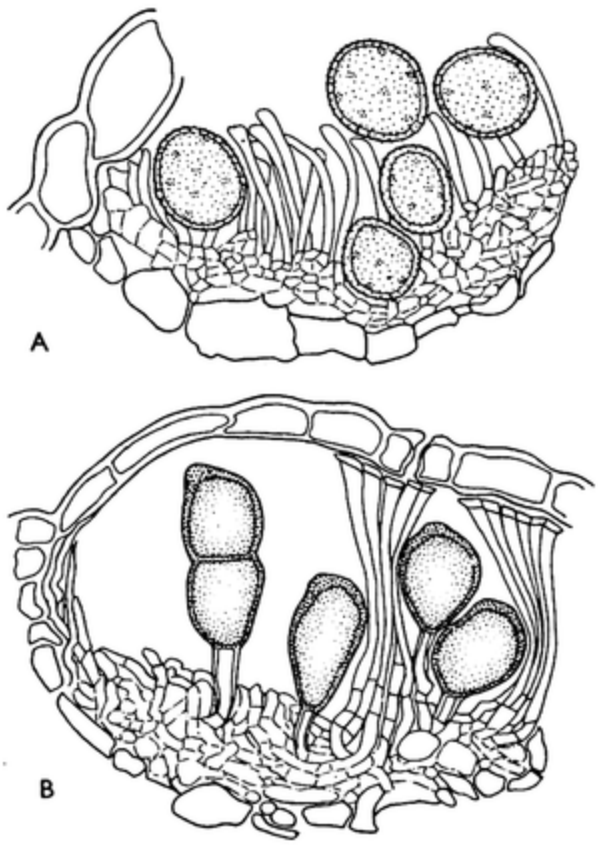


FIG. 215.—Brown rust of barley (*Puccinia anomala*). *A*, portion of uredosorus. *B*, of teleutosorus ; note the tall paraphyses, the one-celled mesopores, and the epidermis intact (× 520) (after Butler, *Fungi and Disease in Plants*)

Brown rust of barley is heteroecious. The aecidial stage has been discovered on *Ornithogalum umbellatum* [5, 6, 8, 12] (' Star of Bethlehem '), but not in Britain. On leaves of *O. pyrenaicum*, however, spermagonia and aecidia of a rust were found in Wiltshire, and inoculations on barley seedlings produced uredosori in 9 days [1, 1a]. This host may therefore initiate local outbreaks of brown rust and provide for hybridisation between known races of the barley rust and for the production of new ones [1a]. The aecidia are round, yellow, about 200μ in diameter, and furnished with a peridium ; spermagonia are found on the leaf at points opposite to the aecidia ; aecidiospores are sub-globose, 25 to 30 by 23 to 29 μ, with a finely warted surface.

At least 30 physiologic races of *P. anomala* are known. Some of these

races, e.g. race ' 12 ', are said to attack all the differential hosts, while others, e.g. race ' 17 ', are reported to be only feebly pathogenic, and other races, again, are more or less localised in their distribution [2, 4, 10].

In England and Portugal [2-5] the rust is capable of surviving the winter in the form of uredospores, and the recent discovery of a new alternate host in April in southern England is also significant [1 a]. The uredospores are unable to withstand high summer temperatures, though different physiologic races of the rust probably behave differently in this respect. In general, the most suitable conditions for the germination of the uredospores are a relatively high atmospheric humidity, of 72·5 to 100 per cent. [3], and a temperature of 16° C. [10]. Infection by uredospores is stomatal and is preceded by the formation of appressoria over or close to the stomatal apertures; haustoria are developed by the mycelium (Figs. 103, 104). In certain parts of Portugal, during the hot months, no barley leaves or tiller shoots can withstand the long drought, but at higher altitudes numerous plants are often found to carry uredo pustules. The rust is also capable of passing through the summer successfully in localised cool, sheltered places on edges of woodlands, and at sea-level near brooks and creeks in shady places. From such localities the uredospores are probably carried by wind to the plains in central Portugal where most of the winter cereals in that country are cultivated [4].

Tests carried out in Germany in 1937 showed that most varieties of barley were susceptible to this rust [11]. In Australia, however, numerous varieties of *Hordeum vulgare* proved to be highly resistant though unsuitable to local conditions in New South Wales [13, 14].

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Leaf Stripe of Barley, *Pyrenophora graminea* Ito & Kuribay (= *Helminthosporium gramineum* Rabenh.)

' Leaf stripe ' is an important and widely distributed disease of barley. It occurs almost exclusively on wild and cultivated barley, but has recently been observed, in Italy, on wheat and oats [1] and, in South Africa, maize appears to be susceptible [17].

The disease is carried by the seed. On young seedlings in the field it begins with the appearance of pallid spots which, at first, are very difficult to detect as the spots are more or less concealed by the leaf sheaths. The symptoms are more



FIG. 216.—Leaf stripe of barley. *Pyrenophora graminea* (*Helminthosporium gramineum*). On leaves and seedlings of barley (photos by Dillon Weston)

evident as the plants approach maturity, and when once a leaf is attacked, usually all the succeeding leaves, in turn, become affected, and extensive damage is done to both leaf sheath and blade (Fig. 216). Tillers which arise from a diseased shoot may escape infection altogether or be killed almost at their inception in the axil of the diseased leaves from which they contract infection, or they may persist in a more or less diseased condition. With continued growth, the symptoms on the older leaves take the form of long, yellow stripes, one to seven in number on each leaf. These stripes soon extend to form long, parallel streaks which may be continuous or intermittent, from leaf tip to base (Figs. 217 to 219). As development proceeds the stripes turn brown, though in some localities they may retain the yellow colour, and the whole leaf gradually dries up. Old lesions usually have a brown margin with a straw-coloured centre along which the spore clusters of the causal fungus appear as greyish-black spots.

The effect on the plant is considerable. The leaves often split or shred, hang limply and wither early, and growth is checked so that the plant is often only half the normal size. The development of the ears is greatly influenced by the disease, and three types may be distinguished. In the first the ear may emerge entirely from the sheath, the grains are present but are not filled, the awns and glumes are limp and darker in colour than the normal; in the second type the ear is arrested in its development, the top internode of the axis ceasing to grow before the ear is completely out of the sheath, the awns may emerge but are twisted and bent, and even when formed the grain does not mature properly, but remains small and soft; the third type is the most common, and in this there is complete absence of visible ears, which do not emerge from the sheath at all [19, 37].

Leaf stripe of barley is caused by an Ascomycete, *Pyrenophora graminea* (previously believed to be *Pleospora graminea* [4, 5, 38]), the perithecia of which, discovered in 1930, are rare and, so far, have not been found in Britain [13]. The parasite was first described in 1856, in Germany, and is more familiar as *Helmintho-*

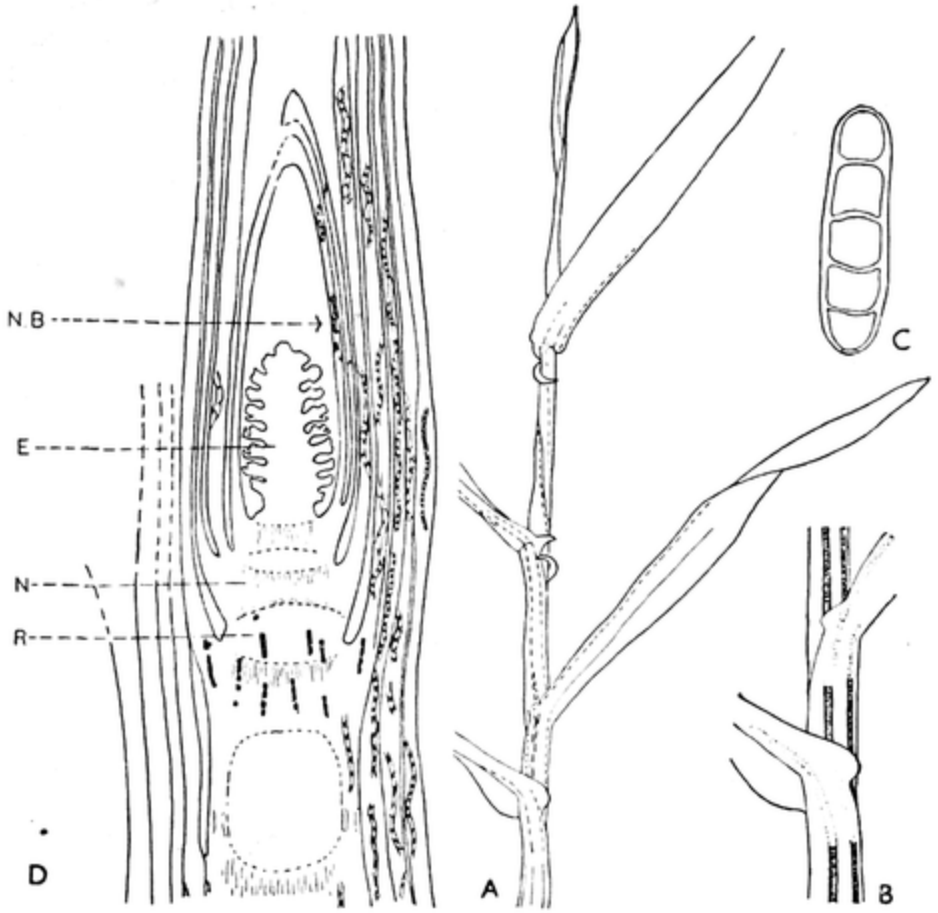


FIG. 217.—Leaf stripe of barley. *A*, *B*, the leaf-stripe effect on leaf sheath and basal part of lamina. *C*, conidium. *D*, median section of part of a young barley plant showing the growing region of stem and ear enclosed in the leaf sheaths; the nodes are indicated by light shading. *E*, ear receiving an *external application* of hyphae from the region *N.B* in the innermost leaf, long before the mycelium in the stem, as at *R*, has worked its way up from the core to damage the ear. *N*, node. *R*, internode, with mycelium (after Smith, *Ann. App. Biol.*)

sporium gramineum, the conidial stage. The fungus also forms sclerotia and pycnidia, but the latter are not common on barley in the field; little is known of the functions of the pycnidia, but they are reported to be very similar to those described in the related species *H. teres* which causes 'net blotch' disease of the same host (p. 443) [35]. The natural mycelium of *H. gramineum*, such as is present on the infected grain (this mycelium is an important source for the over-wintering of the organism) consists of thick-walled, septated hyphae, of barrel-shaped cells, yellow to brownish in colour and very variable in cell dimensions; the hyphae are so closely interwoven as to resemble mycelial clumps which later, by further thickening of the cell walls, become highly resistant, and form a resting mycelium on the surface of the grain after harvest; the same kind of mycelium may be seen in culture when the medium is drying up. These resistant clumps are of the nature of sclerotia, and they are but a stage removed from the natural sclerotia which form pear-shaped bodies on the surface of, and sometimes within, the barley grain [32, 38]; sclerotia have also been produced in pure culture, at *p*H 5·8, on a plum-agar medium [36].

The conidia (Fig. 217 C) are developed on light-brown conidiophores which are found in great profusion on lesions on the foliage and glumes ; the conidiophores arise in clusters of two to six, fascicles of three to five being common (the conidiophores of *H. teres*, and *H. sativum*, described in the succeeding sections rarely number more than three in a fascicle). The conidia are sub-hyaline when newly formed, turning yellow-brown at maturity, but never become dark-olivaceous like those of *H. sativum*. In shape they are sub-cylindrical, straight or slightly curved, widest at the base and tapering bluntly towards the apex, thin-walled, and septated from 1 to 7 cells which do not show constrictions at the septa, as do the conidia of *H. teres* [6]. The conidia measure from 50 to 125 by 14 to 22·5 μ, but there are wide differences in dimensions and number of septa shown by conidia from different races of this species ; on an average they are 3- or 4-septate [2]. Germ-tubes may emerge from any or all of the cells of a conidium, and fusion of germ-tubes or of hyphal cells is frequent [2, 10] ; this phenomenon of fusion may possibly account, at least in part, for the production, by hybridisation or mutation, of the very numerous physiologic races of this organism.

Perithecia are rarely found in nature ; they were observed in 1922, in California, on two-year-old straw of barley, and have also been produced from sclerotia in culture [21], and on the glumes of germinated grains of barley [22]. The perithecia are sub-epidermal at first, later erumpent, globose when young, subsequently flask-shaped or conical with the formation of a short, cylindrical, ostiolar beak ; the wall is blackish-brown, thick, and often beset with long setae intermixed with defunct conidiophores ; perithecia vary from 350 to 380 μ in height, and from 450 to 800 μ in widest diameter ; the fasciculate, long-clavate, hyaline asci measure from 225 to 425 by 32 to 50 μ, and contain 1 to 8 (mostly 4 or 8) yellowish-brown, ellipsoid ascospores which are furnished with one or two vertical septa in the median cells ; they are markedly contracted at the septa ; ascospores measure from 45 to 75 by 20 to 35 μ [13]. Black, sclerotial bodies resembling perithecia have been reported to occur on barley stubble in the autumn and observed to produce perithecia with viable ascospores in the spring [38], but others have not found the sclerotia to develop any further [3, 20]. The ascospores are reported not to play any part in the transmission of the disease [32, 38], which appears to take place solely by the planting of infected grain. Secondary infections by means of conidia, carried by wind or otherwise, to the foliage and to the head at flowering time or later, are not believed to play any important part in the spread of the disease in the field, but upon this point further information is desirable.

The fungus is carried on the outside of, and within the grain, and infection takes place when the seed germinates. The mycelium, which is usually found between the pericarp and the coat of the seed, can remain viable for two years or more, and under moist conditions in the soil may produce conidia and fresh hyphae. Penetrations take place through the coleoptile, or the coleorhiza, or the root itself. The path which the fungus takes within the growing seedling has been described as being systemic [2, 16, 23, 24, 36], as in the smuts, but later and more detailed studies [31, 32, 34] describe an entirely different route (Figs. 217, 218 A) in the method of infection of leaves and ear. Whether infection is systemic or not, penetrations take place chiefly through the coleoptile. The more recent interpretation of the mode of infection is that the fungus, having made a certain amount of upward and downward growth within the coleoptile, breaks through at the inner surface of this sheath to penetrate the first leaf, or more correctly, its sheathing base which is in close contact with the inner face of the coleoptile. The fungus,

now in the sheathing base of the first leaf, travels across it and breaks through at the inner surface to penetrate and infect the second leaf in contact with it. This process of penetration from outer leaf to inner leaf goes on for all the leaves in turn, and though the plant is gradually extending itself by growth, the young ear itself is also penetrated from the leaf next to it, in the same way. Infection may also pass into the internodes of the stem. During these successive penetrations the ear is still retained within, in more or less close contact with, its own protective sheath, and surrounded by the telescoping system of the older leaves or leaf sheaths, one within the other. It follows that, as the first leaf (infected as it is from the coleoptile) is pushed out during its growth from the base, it will show the presence of the fungus in one or more *stripes*. The latter, at this early stage are somewhat pale and translucent, and are described as the ' pale-stripe ' phase of infection. With continued emergence of the first leaf and fresh penetrations into it by the fungus from the coleoptile, the infected parts become more and more visible as *longitudinal stripes* corresponding to the number of penetrations made from the coleoptile (Fig. 218 A). Thus, by upward growth, each leaf in turn will brush a vertical strip of epidermis against the *externally applied mycelium*, and so penetration, followed by infection, proceeds across from one leaf to the next. The young ear in its turn receives an ' external application ' of hyphae from the uppermost leaf and, after penetrating the glumes, the hyphae reach their final destination at the surface of the ovary. Sometimes penetration may go deeper than the pericarp but hardly ever beyond the testa of the seed. It is significant that when the mature ears are examined the diseased areas on the glumes occur precisely at those parts which were in contact with the diseased portions of the enwrapping sheath, and not at any parts on the inner surface of the glumes close to the embryo, which would have been the case had infection reached the glumes from within, that is, in true systemic manner. It is noteworthy that this infection takes place at a time long before any mycelium in the interior of the stem may have worked its way up to damage the ear, as in systemic infection in the smuts [32].

Depending on the severity of the attack, the ears may escape entirely, or be heavily infected and die, or they may tolerate the fungus in greater or lesser degree. According as conditions of the environment favour the rapid development of the host, the disease may stop producing stripes on the leaves and the rest of the plant may grow away from the fungus and the ear may escape completely. Otherwise with the continued production of stripe lesions and infected ears, conidia are produced on them in abundance in the field, and these conidia might well be considered as being responsible for spreading infection by wind to the leaves and flowers of healthy plants in the crop. But such ' secondary infections ', as mentioned above, are not reported to be of ordinary occurrence in this disease, and although conidia may also be conveyed to both diseased and healthy grain during harvesting and threshing, there appears to be little certainty of infection from this source [32]. In respect of the divergent views relative to the path pursued by the fungus during infection, whether typically ' systemic ', or as outlined above, by ' external application ', it is not improbable that both methods obtain, in view of the fact that *H. gramineum* connotes so many races which differ so extensively in their degrees of parasitism and pathogenicity [29].

Little appears to be known about the possibility of seedling infection starting direct from the soil, but since the fungus is capable of forming very resistant mycelium on the glumes, as well as sclerotia, it is not improbable that these forms of the fungus may be found suitable for over-wintering in the soil in the same way as it is known to live for long periods on stored grain.

In relation to the effects of the environment on the incidence of leaf-stripe disease, it was found that when temperatures of the air and soil were kept constant, several types of stripe reaction developed under the influence of different methods of inoculation, different isolates of the fungus, and different host varieties [12, 29]. At low temperatures the proportion of diseased plants was perceptibly increased, and a dry condition of the soil — less than 20 per cent. saturation — during the period of emergence favoured the disease as compared with very wet soil. In general, the highest incidence of disease occurred at lower temperatures of 12° to 16° C., but under these conditions the symptoms were later in appearing than at higher temperatures of 20° to 24° C.; changes of temperature in the early stages of germination from low to high or from high to low, had the effect in the former instance of stimulating the appearance of the symptoms and in the latter of retarding it [28]. In Minnesota [14], early sown barley is usually more liable to infection than the late sown, and the critical period for infection appears to be incident during and immediately after germination, hence the comparative high resistance of barley in warm regions and the severity of the disease in cooler climates.

As leaf stripe of barley is a seed-borne disease, the best method of control is by seed disinfection. The well-known hot-water treatment already mentioned in the control of smut disease has also given good results here, as have also formalin and organo-mercuric applications [7, 8, 18, 25, 26, 37]. Winter-sown barley in Britain is especially liable to attack, and special care in the disinfection of the seed is essential [32].

Profound differences in pathogenicity between the numerous races of *H. gramineum* render breeding of barley for resistance and immunity very difficult [1a, 2, 9, 11, 28, 40]. Extensive studies in the United States have shown that, in general, the varieties Svansota, Manchuria, Minsturdi, Peatland, and Velvet were the most susceptible, while Lion, Glabron, Wisconsin '38', were moderately resistant, and Black Hull-less, Spartan, and Trebi were the most resistant of those tested [2, 15, 27].

Since the disease is practically confined to barley, the usual rotations may be practised, and the occurrence of the fungus on wheat and oats is exceptional; in South Africa, in place of maize in the rotation, the planting of legumes is recommended [1, 17].

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Foot Rot of Barley, *Ophiobolus sativus* Ito & Kuribay (= *Helminthosporium sativum*) Pamm., King & Bakke

'Foot rot' or 'Helminthosporium disease' of barley also attacks wheat and rye, but oats and maize are reported to be immune from it [6, 20] except in isolated cases [11 a, 24 a]. It is known to attack an unusually large number of weed and pasture grasses collected from different genera; some of these include species of *Agropyron*, *Bromus*, *Festuca*, *Hordeum*, and *Lolium*, all of which are highly susceptible and may quite possibly serve as carriers of infection in the field [2, 22, 23].

This disease is of relatively little importance in Europe and there are but few records of its occurrence in Britain [20, 23]. It is very widespread, however, in the western hemisphere, and is a serious trouble of barley and wheat in the United States, Canada, the Argentine, and Mexico [2, 6, 8, 14]. In Australia, particularly in New South Wales, it is the most destructive of all diseases that cause foot rot of wheat [1, 9, 12]. It is prevalent, too, in South Africa, where in addition to the cereals mentioned it finds congenial hosts in the weed grasses *Cynodon* and *Urochlea* [23]. In Minnesota it is reported to be the principal cause of the premature death of wheat plants after heading [4].

All parts of the host plant, including the floral parts, are liable to be attacked. A discoloration present at the germ-end of wheat grain, a condition described as 'black tip' or 'black point', is now known to be due to the same fungus which

causes this disease of barley [10]. (Other fungi believed to be associated with black-tip are *Alternaria tenuis*, *A. peglionni*, and *Helminthosporium teres*.)

Initial infections start from infected seed or with the planting of clean seed in contaminated soil. The early appearance of the disease in the field resembles a seedling blight, like a damping-off, and the young plants may be destroyed soon after emergence. In lighter infections, however, seedlings may grow into mature plants, but these are often weak and spindly, and appear in the crop in more or less circular patches which may often be several feet in diameter, though dwarfed specimens may also be found intermixed with healthy plants [2]. The internodes and leaves of affected plants are considerably shorter than those of normal plants; the primary roots are infected, and on the lower internodes of the stem chocolate-brown discoloured areas may be seen, with one or more large spots of the same hue on the blades of the first and second leaves which may also show a certain amount of curling. Unlike the 'take-all' disease of barley (*Ophiobolus graminis*) there are, in this 'foot rot' trouble, no mycelial mats or scales of mycelium between the leaf sheath and the culm in the browned basal parts of the shoot. Diseased seedlings which succeed in growing into mature plants usually have a poor rooting system, ears are poorly filled, and what seeds may be produced are small and shrunken. Diseased plants have a tendency to develop far more tillers than healthy plants; it is not infrequent to find as many as 30 to 40 tillers on an infected barley stool, but few of them develop, and the majority remain stunted; in other cases young tillers may fail to emerge from the sheath owing to being crippled by early infection. Plants which attain the heading stage may lie prostrate on the ground and easily break off at the base owing to a diseased condition of the crown.

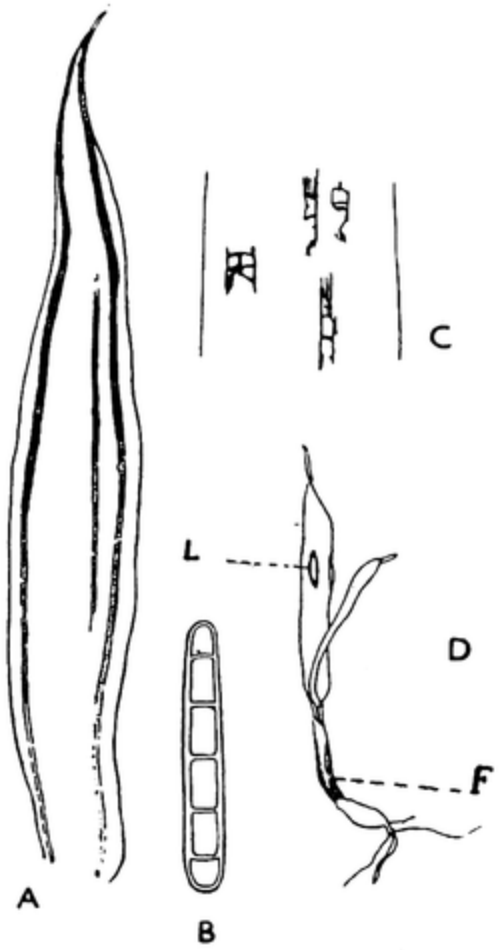


FIG. 218.—Comparison of the leaf symptoms of three *Helminthosporium* diseases of barley. *A*, the leaf stripe disease (*H. gramineum*); note the tendency for the stripes to be more conspicuous in the upper than in the lower parts of the leaf. *B*, a conidium of same. *C*, a small portion of leaf affected with the 'net blotch' disease (*H. teres*); note the netted appearance of the blotches, but this is not always evident. *D*, the leaf blotch, as at *L*, of *H. sativum*, with signs of foot rot at *F* (after Smith & Rattray, *S. Africa J. Sci.*)

The above symptoms may all result from primary infections. Unlike the stripe disease on the same host, secondary infections may come from various sources, practically all the diseased parts above ground, primarily affected, developing conidia. On the adult plants conidia may be found abundantly on the upper nodes, giving these parts a black,

velvety appearance, and secondary lesions may appear on any part of the host, chiefly on the leaves, glumes, and grain. The characteristic symptoms on the leaves consist of numerous dark-brown elongated spots of rather irregular outline and furnished with a light margin (Fig. 218 D); the spots vary in size from 3 to 20 mm. or more, and may join together so that the entire lamina is often destroyed. In general, the disease affects barley and wheat in much the same way but the leaves are the parts most conspicuously affected in barley [4].

A brief account of this disease on barley was first published in 1909 [18], and in the following year was attributed to the fungus *Helmintosporium sativum* [19] a new species, but it was not until 1914 that the same organism was found to be the cause of a similar disease on wheat in America [13]. The fungus is much more familiar under this, the name of its conidial stage, than that of the perfect ascigerous form called *Ophiobolus sativus* produced in artificial culture [15]. On potato-glucose agar, at 25° C., the fungus grows freely to form a compact, velvety layer of black or olivaceous mycelium which soon becomes covered with a dense mass of short conidiophores, but the appearance of the cultures varies greatly according to the strain of fungus, type of medium, and its degree of dilution [6, 17], and saltations are much in evidence [25]. On the host the conidiophores emerge through the stomata, or quite as frequently, from between the epidermal cells, either singly or in small groups of 2 or 3; the conidiophores are brown, septate, and bulge out in knee-fashion just above the septa. In shape and dimensions the conidia are highly variable; they are somewhat cylindrical or distinctly curved, an extreme limit in size being 134 by 30 μ and furnished with as many as 12 septa, one of minimum size being 25 by 14 μ, the average range being from 60 to 120 by 15 to 30 μ, and 3- to 10-septate conidia; sometimes oblique instead of transverse septa and, in some cases, longitudinal partitions may be seen in addition to the transverse septa, giving such conidia a muriform appearance, but these types of spore are exceptional. Germination of conidia normally proceeds from the terminal cells only. While, in general, the conidia remain viable for long periods, perhaps a year [6], their longevity appears to be closely related to their origin from particular races or strains of the fungus as well as to environmental conditions under which they were produced [4, 6].

Apart from their undoubted relationship in the life-cycle of the organism, little is known about the natural occurrence of the perithecial fructifications and their possible rôle in infections. They are described as being erumpent, black-walled, globose or subglobose, measuring from 370 to 530 by 340 to 470 μ, and furnished with ostiolar beaks; asci are numerous, cylindric or fusiform, slightly stalked, with rounded apices and containing 1 to 8, mostly 4 to 8, filiform ascospores of a pale olive-green colour, with 6 to 13 septa; the spores measure from 160 to 360 by 6 to 9 μ, and are spirally coiled within the ascus. Cultures obtained from ascospores are recorded to have given conidia typical of *H. sativum* and, moreover, inoculations with such conidia have given positive results [15].

Numerous physiologic races of this fungus exist, some of which give rise to mutant forms which differ from the parents in morphology and pathogenicity [3, 4, 7]. Certain races are also known to cause a greater amount of injury of the host at the seedling stage than to adult plants, while other races cause decidedly greater dwarfing of the mature plants of both wheat and barley [5]. The first recorded occurrence of this fungus in England, at Cambridge [20], revealed the presence of two strains collected from wheat from widely different sources. Infection experiments carried out with these showed that wheat seedlings ('Little Joss') were

yellowed and distinctly checked in growth, and a browning at the base of the stem showed the characteristic foot-rot condition associated with this disease; young seedlings which had early been killed showed the fungus within the coleoptile and in the tissues at the base of the young shoot. These particular races were only slightly infective to barley (' Spratt Archer '), and had no effect on oats (' Abundance ') [20]. Infection of young seedlings, whether by mycelium or conidia in the soil, occurs in the usual way by penetration of the coleoptile, the fungus thereafter invading the tissues of the young leaf enclosed by the coleoptile [24].

There is a marked difference in the susceptibility to primary infection of young plants at different ages, and in the case of secondary infections the plants are more liable to infection after the heading stage; spots on the stems with the characteristic blackening of the nodes are seldom seen before the plants have headed [2]. Heavy dew and rain, together with high temperatures, are greatly conducive to secondary infections on the foliage and ears, and there seems to be a distinct correlation between the amount of moisture in the air and the number of lesions on the leaves [5]. Seedling infections were found to occur within a wide range of soil temperatures, from 12° to 34° C., and were more severe between 22° to 30° C., but at the latter temperature lesions were usually checked [5]. In general, higher soil, temperatures are favourable to infection [17]. The organism thrives better in alkaline than acid soils, and the conidia can tolerate high degrees of alkalinity in culture [5]. Moreover, the disease appears to be greatly favoured on peat soils, particularly with deep drainage, which is believed to predispose the plants to infection [4].

The fungus survives in infected seed for several years, and hibernates in the field on straw and roots [2]. Barley grains naturally infected in the field have been found with mycelium in all the seed coverings and even in remains of the lodicules [17a]. Over-wintered mycelium in the soil, and contaminated seed harbouring mycelium or conidia are believed to be mainly responsible for the primary outbreaks of foot-rot disease on seedlings. Conidia in the soil may also sometimes start the disease but their viability appears to be greatly dependent on ecological factors, and unless the soil is comparatively dry they do not survive for long [16, 19]. In Canada, in 1931, the spores were very rare or absent in field soils, the fungus existing only as mycelium [11]. Secondary infections by wind-borne conidia are, as stated, widespread.

Control of foot-rot disease by the treatment of seed with hot water, formaldehyde, or organic mercury dips and dusts, give, at best, only partial protection, since the applications, being external are only destructive to any mycelium and spores on the surface of the grain, but seed treatment should, nevertheless, not be omitted. A consideration of the factor of soil-temperature indicates that sowing should be done when the soil is cool and at a time when cereal development is at its best in the matter of producing strong, vigorous roots [2, 21]. In the observance of crop rotation, such non-susceptibles as oats, maize, clover, potatoes, root crops, etc., should be grown but with strict attention to the eradication, if possible, of carrier hosts — the wild grasses — for even if healthy seed is available for sowing in clean soil there is always the risk of infection from conidia carried by wind from these congenial grasses. With regard to manurial treatment and the use of fertilisers

in the control of foot rot, little information is available, except that seedling blight is materially reduced by repeated applications of superphosphates [12].

All varieties of wheat appear to be susceptible to this type of foot-rot, and even in cases where it is thought that good resistant varieties have been established, subsequent tests under different environment showed the most promising of them liable to break down [4]. In the breeding of varieties resistant to *H. sativum* more encouraging results have been obtained in barley than in wheat, and the varieties of the former which have proved to be more or less uniformly resistant include Velvet Comfort, Glabron, Vaughan, and Manchurian Barley [14, 16].

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Net Blotch of Barley, *Pyrenophora teres* Drechsler (= *Helminthosporium teres* Sacc.)

Net blotch disease of barley is frequently reported from various centres in Britain [12], and is widely distributed in Canada and the United States [7, 8, 9, 18]. In South Africa it is the commonest of all diseases of barley, 75 to 100 per cent. of the plants being attacked in some districts [16]. Similar reports are noted from Bavaria where the losses are considered to exceed those due to 'leaf stripe' (*H. gramineum*) disease of this host [3]. In India most varieties of barley are susceptible [10]; the disease is also common in the Argentine, and is reported in Manitoba to cause a discoloration of the seeds of both oats and wheat [8].

Though this disease is often very harmful to the foliage, which at times suffers severely from secondary infections, yet only in a few localities is it reported to be a serious disease of this host, and the general impression is that 'net blotch' is

of relatively minor importance as compared with ' leaf stripe ' and ' foot rot ' diseases of barley [1, 2, 6, 14, 15]. Net blotch is also commonly found on the wild barley-grass *Hordeum murinum*, and as this plant may be found in practically all barley-growing areas it is believed to be a constant source of infection all the year round [12].

Symptoms of net blotch are not easily discernible in the earlier parts of the season because the primary lesions on the first leaves of the seedling, unless very severe, appear as mere spots or streaks, not above 1 mm. in length. Later, however, the blotches increase considerably in size, extending in a direction parallel with the long axis of the leaf, somewhat like the leaf-stripe disease, but not for more than 20 to 25 mm. or so, with relatively little increase in diameter. The affected spots soon turn brown but the colour is not evenly spread over the whole area, and at an early stage there can be seen a number of very narrow lines of a darker brown colour forming a criss-cross network effect over a background of paler brown (Figs. 218 C; 219 B); this lace-like pattern gives the disease its name of ' net blotch '. The parts of the lamina around the blotches are, at first, pale or etiolated, but later each blotch develops a narrow yellowish zone around the brown area; in the blotch itself there is never any variegation of green and yellow, as frequently seen on the leaf spots attendant on the ' foot rot ' (*H. sativum*) disease of barley, above described. When eventually the net lesions become further extended and smaller blotches due to secondary infections join together to form more or less parallel stripes on the leaf, it is not easy to distinguish this disease from the true ' leaf stripe ' (*H. gramineum*) of barley. Careful examination will show, however, that there still remain at several places along the stripes evidence of the original network effect of net blotch. Moreover, any striped effect due to the latter is rarely seen to run down into the leaf sheaths, as in the case of true ' leaf stripe ' disease, unless the leaf is old and the sheath slack. Finally, when lesions have become so numerous as to involve the greater part of the leaf, the latter begins to wither from tip to base and the spots fade from a dark-brown to a more diffused, dull-brown or grey, and it is on these dead tissues that the spores of net blotch make their appearance.



FIG. 219.—Comparison of leaf stripe of barley (*H. gramineum*), *A*, and net blotch of barley (*H. teres*), *B* (photo by Foister & Noble)

The fungus causing net blotch of barley was first discovered in 1881 in its conidial form, named *Helminthosporium teres*; the perithecial stage was not found until 1919, at Madison, on straw, stubble, and infected spikelets of barley, and proved to belong to the Ascomycete genus *Pyrenophora*, so that the correct designation of the organism is *Pyrenophora teres* [6]. This perfect stage is, however, very infrequently found. The fungus grows well in culture at 25° C., on potato-agar, but is of slow growth; the

mycelium colours the medium dark green but not to the depth of colour seen in a culture of *H. sativum* on the same medium. Greyish-white tufts of mycelium arise on the surface of the colony but the conidia are not nearly as abundant as produced by *H. sativum*. There are, however, small black sclerotia of variable size formed in abundance, but these bodies are absent or rare in *H. sativum* [5]. The brown conidiophores arising at diseased spots on the leaves emerge from between the epidermal cells or through the stomata from an intercellular mycelium below, singly, or in groups of 2 or 3 ; they are shorter and less closely septated than those of *H. gramineum* and *H. sativum*. The conidia are solitary and measure from 30 to 175 by 15 to 22 μ, and are furnished with from 1 to 10 septa at which the spores are perceptibly constricted ; the conidia, hyaline when newly formed, later turn yellow or greenish, but do not become as dark-coloured as those of *H. gramineum*, or as deeply olivaceous as the conidia of *H. sativum*. All the cells of the septated conidium may germinate.

In addition to the conidia and sclerotia above mentioned, *H. teres* sometimes forms pycnidia (Fig. 220). These have been found under natural conditions on dried and starved plant tissues, such as barley straw and chaff [13, 17]. The pycnidia are small, spherical, about 35 μ in diameter ; the pycnospores are unicellular, elliptical or round, and measure from 1 to 2 by 1 μ ; they are reported to produce normal mycelium in germination but the observations are few [13, 17].

Perithecia of *Pyrenophora teres* have been reported in Britain [14] and elsewhere, on culms, straw, stubble, and spikes of barley [4, 6, 11, 13]. These findings report the perithecia to be mostly ill-developed or abortive, and spore measurements are based largely on the examination of a few specimens only. In shape perithecia are similar to pycnidia, and furnished with short, blunted necks, but with no definite ostiolar beaks ; setae may or may not be present on the surface, where they may sometimes be found interspersed with derelict conidiophores. The asci are described as being sub-cylindrical, with a thickened

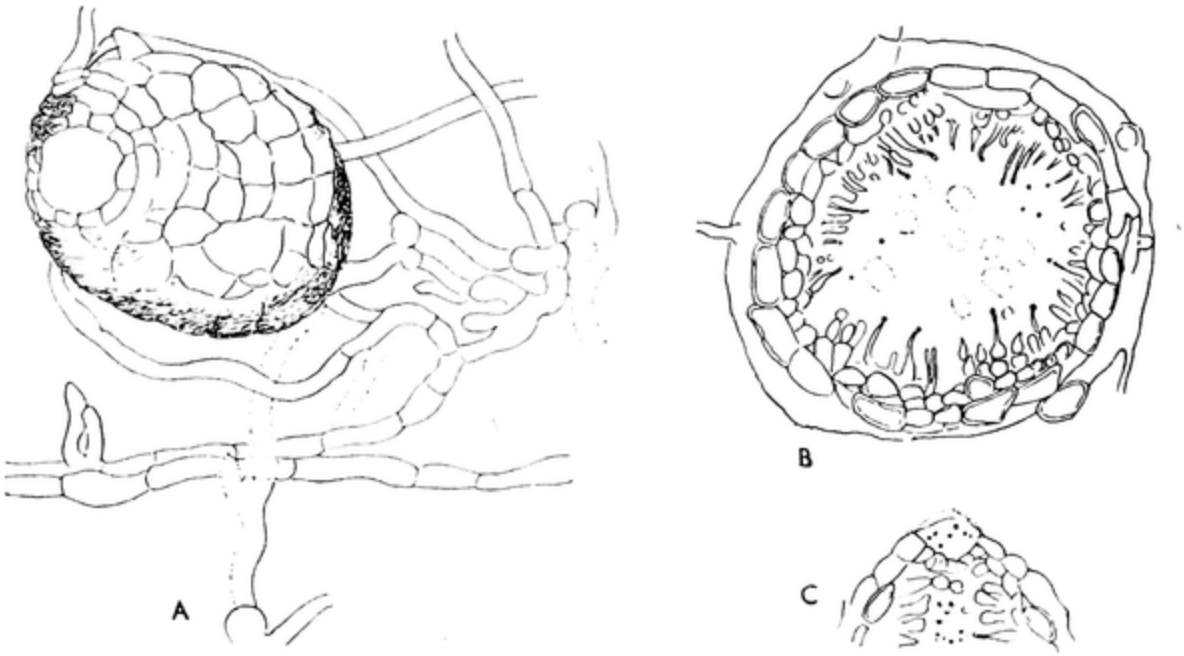


FIG. 220.—*Pyrenophora* (=*Helminthosporium*) *teres*. Diagrams of the pycnidial stage. *A*, the pycnidium arising on the mycelium, showing ostiole. *B*, the same, in section, showing the various layers of the wall and, within, the very short hyphae abstricting pycnospores. *C*, details of the ostiolar region, showing the sporophore-hyphae and spores (after Smith)

ring at the apex, and measuring from 220 to 250 by 30 to 36 μ, with 8 spores arranged in two rows ; the ascospores are light-brown in colour, and vary from 52 to 60 by 18 to 22 μ ; they are 4-celled and constricted at the septa ; one or both of the middle cells of a spore may be furnished with vertical septa [6].

The primary stage of infection of barley with net-blotch disease begins with the sowing of infected seed. It takes place in the same way as already described for ' stripe disease ' of barley (p. 434), and there is no need to repeat the observations here. With the appearance, however, of secondary lesions in isolated patches among the original stripes of the primary lesions, it is clear that net blotch differs strikingly in this effect from the true ' leaf stripe ' in which all the infections necessitate close contact of outer leaf with inner leaf during development. Moreover as the new spots in net blotch appear on all the leaves of the plant, upper as well as lower, and on the ears, it is evident that they are due to secondary infections, as a result of spores from the primary lesions having been carried by wind. Secondary infections on the glumes are often responsible for contamination of the grain.

The primary stage of infection occurs at comparatively low soil temperatures, of 10° to 15° C., the first signs appearing as small local infections on the coleoptile or on the first seedling leaf. If, however, the soil temperature is raised, the disease gradually disappears until at a temperature of 20° C., infection appears to be checked. Experiments showed that on plants grown from infected seed sown in July, the disease was virtually absent, whereas a high percentage of infection was obtained from sowings made in March and April [13]. But the influence of fluctuating temperature seems to have little effect on the incidence and continuance of secondary infections and, as higher temperatures do not check these later infections, it is clear that they account for a great deal of disease on the leaves. It is noteworthy that in certain localities where the mean summer temperature is relatively high, as in the region around Grahamstown, South Africa, secondary infections are said to be entirely responsible for outbreaks of net-blotch disease. In such latitudes it is not necessary that the disease should be introduced into the field with the seed, that is, the primary stage is not essential for its propagation. In these regions of high temperature this fungus with its spores may often be seen on self-sown, ' volunteer ' barley or on the ubiquitous wild species, *Hordeum murinum*, practically all the year round. From these sources, fields of cultivated barley easily become infected by wind-borne spores.

From the foregoing considerations it is clear that seed treatment with disinfectants has, in general, but little control over net blotch disease. Such treatment must not, however, be omitted, for under conditions of greater climatic contrast the kind of infection that may be expected to occur, whether the primary or the secondary, or both, will depend largely on the locality [16]. The treatment is the same as indicated for leaf-stripe disease.

The amount of infection with net blotch proved to be comparatively low when nitrogenous manures were withheld, though there was no significant difference when these were sparingly applied, but potassic and phosphoric fertilisers reduced the amount of disease appreciably [3].

There are no records of the possibility of *H. teres* being able to pass the winter

in the soil in the form of a resting mycelium. The sclerotial bodies, however, have been found on the dead leaves during the winter and may possibly give rise in the spring to conidia or on rare occasions to perithecia, in which case both conidia and ascospores would contribute to secondary infections. But details of the formation of conidia and perithecia under such conditions are not, so far, known. In any case, sanitary measures, such as turning-in of the stubble or other barley debris, and crop rotation should be adopted as precautionary measures.

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Ergot of Rye, *Claviceps purpurea* (Fr.) Tul.

The disease known as ergot, though associated for centuries with its attack on rye, is very common on a large number of wild and cultivated grasses; it also occurs occasionally on Rivet wheat and on Rivet × *Triticum vulgare* crosses but very seldom on oats or barley (4a, 8, 21). In Britain the disease is most prevalent on rye in the northern parts of the country. It confines its attacks to the inflorescence which, however, is rarely wholly destroyed, and an infected head of rye usually shows only some two or three grains replaced by the dark-coloured horn-like sclerotia or ergots which are so characteristic of this disease (Figs. 221, 222).

An insidious type of poisoning presumably caused by the ingestion of ergots in fodder or ergotised rye bread has been known for a long time in animal and human pathology, causing in some cases a vascular gangrenous condition and in others a nervous convulsive type of disorder, and sometimes both occurring together in the malady known as ergotism.

The sclerotial ergots contain valuable therapeutic properties and considerable advances have recently been made in defining the molecular composition of about a dozen different alkaloids yielded by ergots (1, 2, 3). In some countries the production of sclerotia for pharmaceutical purposes is carried out on a large scale by artificial inoculation of the rye-heads with the spores of the parasitic fungus causing this disease. Large quantities of ergots are annually imported into Britain, chiefly from Spain, Portugal, Russia, Poland, and the Baltic States.



FIG. 221.—Ergot (*Claviceps purpurea*). *A*, the sclerotia on three plants of slender foxtail. *B*, on cocksfoot (left) and on two plants of perennial rye-grass. *C*, on rye (*A*, *B*, photos by Dillon Weston ; *C*, by Foister & Noble)

Claviceps purpurea, the fungus of ergot, is widely distributed in Europe, America, and Australasia. The parasite attacks only the inflorescence of its host, black sclerotia being produced instead of grains. The loss of yield from rye crops appears to be due not only to the replacement of grain by sclerotia but also to the fact that infection of a flower and sclerotial development is usually followed by sterility of neighbouring flowers which, though occupied by the fungus mycelium, remain empty without production of sclerotia. It has been ascertained that a single sclerotium in a head of rye gives a clue to about 1 per cent., with an increase to about 20 per cent., sterility for the presence of some 14 or 15 sclerotia per ear [10].

Early signs of infection in the inflorescence are seen in the form of a sticky yellow fluid secreted from infected flowers and, embedded in the sweetish fluid, or ' honey-dew ' as it is called, is an immense number of minute conidia ; this stage is known as the ' Sphacelia ' condition (Fig. 223 A) and was originally named *Sphacelia segetum* before the connection between it and the ascigerous perfect stage, *Claviceps purpurea*, was discovered. The mycelium in the flowers producing the conidia consists of intricately woven hyphae which emerge from the surface of the ovary in contorted slimy masses. They give rise to a more or less close palisade of short conidiophores which abstrict minute conidia in such profusion that, when liberated into the sticky exudate, they accumulate on the surface of the inflorescence like drops of dew. This viscous secretion has been found equal in concentration to a 2·33 molar solution of sugar, and when the conidia are examined in this natural matrix they are elliptic in shape and vary in size from 3·5 to 6·0 by 2·5 to 3 μ, but when the fluid is diluted the conidia increase their dimensions, showing a range of 6·5 to 7·5 by 4·2 to 4·8 μ. It appears that the conidia will not germinate unless the honey-dew is diluted. The sweetish fluid is attractive to insects, and conidia may thus be carried from infected to healthy plants and infection results if they are conveyed to the open spikelets. The swaying of the

plants in wind and splashing rain no doubt also helps in the dissemination of conidia.

A decline in the production of conidia is early followed by a progressive swelling and hardening of the ovary due to fungal penetration and, instead of maturing into a grain, the enlarging ovary, while still retaining, more or less, its natural shape, is gradually converted into a sclerotium or ergot. Typical sclerotia are violet or dark purple in colour, with a remnant of the twisted hyphae of the sphacelial stage frequently persisting for a long time as a tawny wisp at the apex of the enlarging body. The sclerotia are larger than the normal grains, but they vary considerably in size according to the host bearing them. Those of rye may range from 1 to 3 cm. long and 8 mm. thick; those of *Molinia* from 4 to 6 mm. long by 1 to 1·15 mm. thick; those of *Agrostis* as small as 2 mm., while those of *Festuca elatior* may be as long as 18 to 20 mm. (16). The interior of a sclerotium consists of a hard white mass of fungal tissue from which any remains of ovary or ovule tissue can no longer be distinguished from the pseudoparenchyma of the sclerotium. In general, the sclerotia are fully formed in the affected ears at about the same time as grains are produced in the healthy flowers. They may fall and hibernate on the ground or may be gathered at harvest with the crop and at threshing become an admixture with the seed. In some cases sclerotia may be dispersed through the agency of wind after the fashion of pappus fruits; thus the sclerotia from the flowers of *Calamagrostis epigeios*, like the healthy grain, are enclosed within the floral pales, and by virtue of the fine circlet of silky hairs at the base of the pales expanding in dry weather, may be carried by the wind; the sclerotia of *Brachypodium sylvaticum*, enclosed within the awned investment of the flower, may be picked up on the coats of passing animals.

Sclerotia which fall to the ground at harvest may germinate in the following spring, but in general they do not remain viable on the ground for more than a year (13). They can withstand very low temperatures and, while freezing appears to be conducive to their germination, it is not essential (14), and the minimum temperature for sclerotial growth is about 10° to 12° C. (9, 22); it is also recorded that a range of 9° to 15° C., following one month of freezing, is most favourable to germination, which, however, is checked at temperatures of 18° C. and upwards (11). Even portions of broken sclerotia which may pass



FIG. 222.—Ergot. *A*, the sclerotia on two plants of barley. *B*, on three plants of Rivet wheat; note the 'nectar' ('honey-dew' stage) on the middle specimen (photos by Dillon Weston)

undetected in threshed grain or seed samples may grow quite as well as whole ones [6].

Favoured by a moist environment in the previous autumn and a suitable spring temperature of about 11° C., the sclerotia on the ground show signs of reviving by developing a number of small cracks arranged longitudinally here and there at various parts of the surface, and tiny semicircular flaps of the coloured rind may be seen turned back at these places [14]. From the white internal fungoid tissue thus exposed, a small white, club-shaped stalk or ' clava ' emerges at each point and finally each stalk expands at the apex into a flesh-coloured, round, or thimble-shaped head or stroma (Fig. 223 B). The stroma is covered with numerous closely set papillae, each papilla surrounding an ostiole leading down to a perithecium. A sclerotium may give rise to as many as 60 of the stalked stromata [1] but the average number is usually about 6. The stalk may vary in length from 5 to 25 mm., according to the depth of the sclerotium in the soil. A whole sclerotium with its complement of stromata looks like a small black pin-cushion on which the pinkish heads of the pins, the stromata on their white stalks, are inserted at various points on the cushion with the heads directed towards the light, and those stromata emerging from the sides and under parts of the sclerotium may often have their stalks considerably twisted in order that their heads may attain this position. Apparently the stromata must be completely elevated above the soil for efficient discharge of their spores. During the development of the stromata from the sclerotia there is no production of external mycelium, but sometimes, and only after the stromata are mature, a small circlet of rhizoid-like hyphae may often be found around the base of the stalks [14]. From the first appearance of a stroma at the head of its clava, it usually takes about 5 to 7 days for the perithecia to reach full maturity [19]. The perithecia are completely sunken in the stromatal head, forming a single layer of numerous flask-shaped cavities which follow closely the contour of the dome-shaped papillate stroma (Figs. 42 A, 223 C). From a group of specialised cells at the base of each cavity numerous club-shaped asci develop and, interspersed with slender paraphyses, line the base and sides of the perithecia which open to the exterior by narrow ostioles situated, as already stated, at the papillae [7]. An ascus contains 8 very slender filiform spores measuring from 50 to 76 by 0·6 to 0·7 μ in diameter. The ascospores are extruded under conditions of high humidity of about 76 to 78 per cent. saturation of the atmosphere [22] and may either collect at the ostioles in viscid masses for dispersal in splashing rain-drops, or by insects, or if showers are followed by sunny periods such conditions would liberate the spores from the perithecia and cause them to be forcibly ejected into the air and so may be carried further afield by the wind to any spikelets that may be open for the reception of pollen.

The discharge of ascospores from the perithecia appears to synchronise with the early anthesis (escape of pollen) of the particular host plant attacked. These primary infections by ascospores are said to be more successful when the flowers have just opened, presumably because the spores have a better chance to grow on the stigmatic brushes of the flowers when there is no hindrance from a previous deposit of pollen ; but fresh infections may continue for about 15 days after flowering has begun. Infections are naturally heavier the longer the flowers are

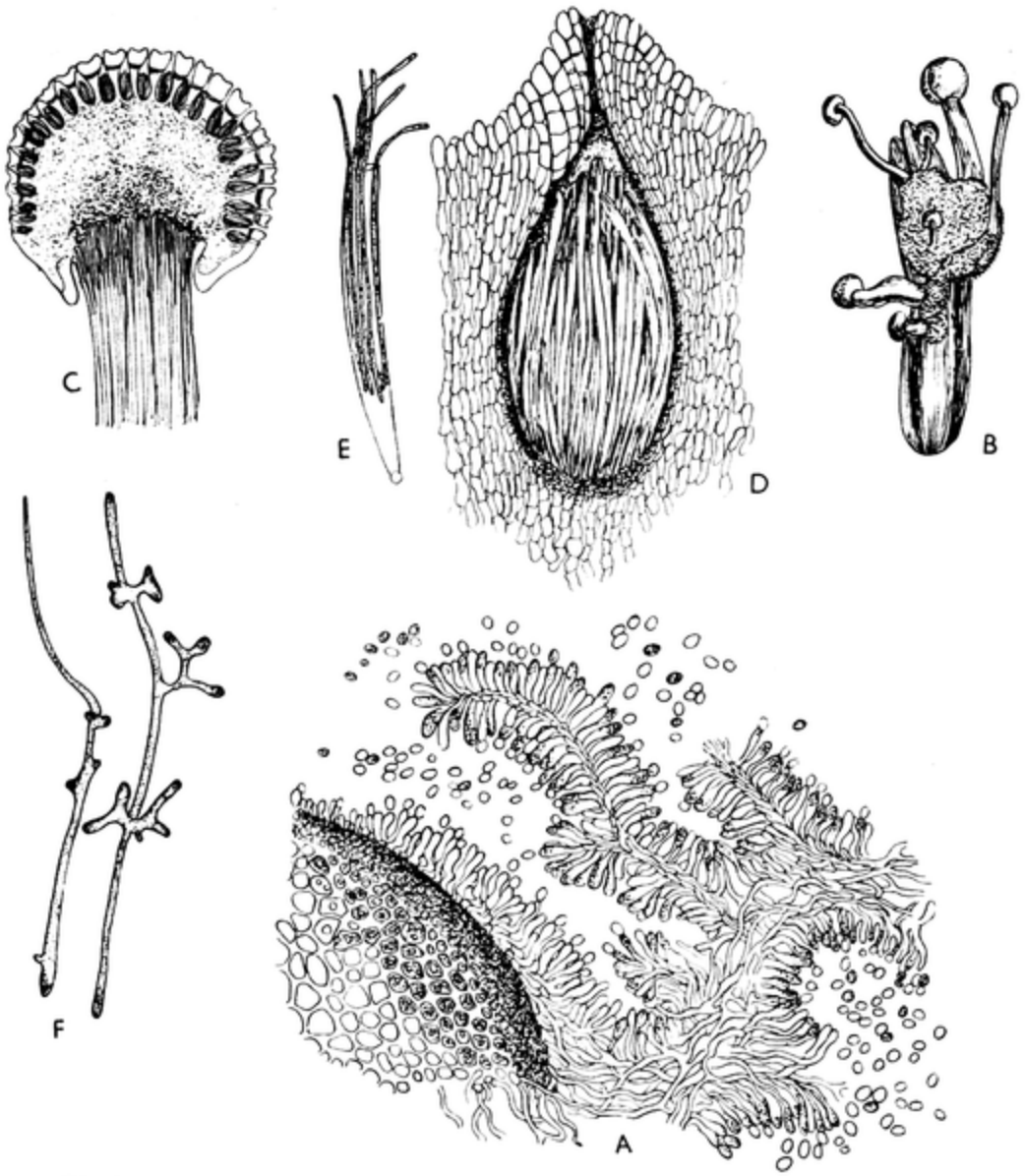


FIG. 223.—*Claviceps purpurea*. *A*, the conidial, ' honeydew ' (*Sphacelia*) stage on the ovary. *B*, the germinating sclerotium showing seven stromata. *C*, longitudinal section of a perithecial stroma showing numerous perithecial cavities (see also Fig. 42 A). *D*, a perithecium. *E*, ascus discharging ascospores. *F*, two germinating ascospores (from Sachs' *Lehrbuch*)

open for pollination, and it is probable that one reason, at least, to account for the comparatively greater susceptibility of rye than of wheat to infection is the fact that the flowers of rye remain open for a much longer period than those of wheat which remain open for a few minutes only and are thus protected by early closing of the glumes against infection [20]. The ascospores germinating on the moist extruded stigmas produce very long germ-tubes which proceed quickly to the base of the ovary without, however, penetrating this region as yet, but later creep up to the apex of the ovary to form the contorted mycelial system composing the sphacelial, conidial stage. The production of conidia takes place very quickly, in about two days after infection with ascospores, provided atmospheric humidity is not less than 74 per cent. saturation, with a prevailing temperature at the com-

mencement of flowering not above 13° or 15° C. [22]. Secondary infections may be effected by the conveyance of the conidia-laden honey-dew by insect carriers or by contact of diseased with healthy inflorescences or by splashing rain and, most probably, it is the conidial infections that are accountable for most of the damage to rye crops especially in humid low-lying localities where insects are prone to flourish.

The conversion of the young ovary into a fungoid sclerotium takes place gradually from below upwards, the superficial hyphae penetrating the ovary wall at various points, mostly near the base. The cavity between wall and ovule also becomes filled with dense mycelium. The ovule itself and the upper part of the ovary-cavity are relatively free from fungus until they finally become obliterated by pressure of the encroaching mycelium [9]. With the gradual protrusion of the young sclerotium beyond the floral glumes towards the light, its superficial mycelium changes from white to carrot-red and finally to a dark-violet, almost black colour, the extreme tip retaining for a long time a wisp of the whitish or tawny twisted sphacelial hyphae, but this too, is finally shed [12].

Claviceps purpurea is amenable to culture on various kinds of media, e.g. rye, malt, and cherry extracts, but only the flocculent colonies of conidia are produced and they develop best over a range of temperature from 20° to 25° C. [5]. On a 2 per cent. agar medium containing 0·1 per cent. monopotassium phosphate, 0·025 per cent. magnesium sulphate, 1·0 per cent. cane sugar, 0·1 per cent. asparagin, the conidial colonies produced peculiar knotted groups of hyphae resembling somewhat the internal structure of the sclerotia but they developed no further and bore no superficial resemblance to the natural ergots. The conidial stage thrives under slightly alkaline conditions but has the power of changing the reaction to acid under which, presumably, further development of conidia is checked [9]; but more recently it has been recorded that cultures begun from ascospores require an acid reaction of the medium for the development of conidia and that an alkaline condition is essential for the production of sclerotia [16a].

For the culture of ergots on a large scale, for medicinal purposes, as already indicated, crops of rye are cultivated, inoculated at flowering time, and the sclerotia gathered by hand. Nodal inoculations also yield sclerotia [20a]. In Budapest it is reported that fields of winter rye are systematically inoculated by special machinery which not only grips the young ears but at the same time sprays them over with an ascospore suspension of the fungus. After some 6 to 12 days the infected ears are seen to be permeated with conidial honey-dew. This is collected and diluted, 1 litre of honey-dew being sufficient to make 60 litres of inoculum containing about 5,000 conidia per cubic mm. Extensive spraying infections are then carried out with the diluted fluid and in order that insects may also help in dissemination of the spore-laden fluid, the crops are usually grown in dense formation, preferably in low-lying humid localities favourable to insect life. In about 10 days before the rye crop is mature, the sclerotia are picked by hand, and it is computed that some 90 to 190 kg. of ergots may be gathered from a hectare of the crop [4, 5].

Claviceps purpurea embraces a number of specialised races. The race attacking rye infects also wheat, barley, *Festuca pratensis*, *Bromus sterilis*, and a few species of *Poa*. Another race collected from sweet-vernal grass (*Anthoxanthum odoratum*)

also attacks rye and various grasses but not barley. The wood brome (*Brachypodium sylvaticum*) and *Glyceria fluitans* have each their exclusive single race. The race on the perennial rye-grass (*Lolium perenne*) also infects *Bromus erectus*, but not rye [17-20].

As the principal cereals subject to attack by the fungus are rye and durum-wheats, the disease can be kept under control by a system of rotation with the commoner wheats, or oats and barley which are rarely attacked. The prevalence of sclerotia on such a wide range of pasture and wild grasses makes it imperative to eradicate as far as possible all such hosts, especially if occurring in wet sheltered borders of rye fields. As the disease affects the inflorescence only, hay should be cut while still green, before ergots develop [16]. The ergots, however, are not long-lived, and deep-ploughing, together with a rotation of 2 or 3 years before planting rye again, is usually long enough to starve these bodies in the soil. Since the sclerotia, in any case, lose their viability after one year, sowing seed 2 or 3 years old, even if contaminated, is reported to be a sufficient precaution against recurrence of the disease [8]. Whole and broken sclerotia may occur in threshed grain and may be returned in a viable condition to the soil at sowing time; it is recorded that they occur fairly commonly in samples of grass-seed, e.g. in perennial and Italian rye grasses, timothy, bent, crested dogstail, and Yorkshire fog; and to a lesser extent in cocksfoot and red and meadow fescues [16]. Ergots may be separated from the grain by immersion in a brine solution of 40 lb. of salt in 25 gallons of water; the sclerotia rise to the surface and the grain is then washed with water immediately [6].

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Chapter XI

DISEASES OF PASTURE AND FORAGE CROPS

Crown Wart of Lucerne, *Urophlyctis alfalfae* (Lagerh.) Magn.

Crown wart of lucerne, preferably called marbled gall (to distinguish it from true crown gall of other plants caused by bacteria), attacks in Britain only the one host lucerne, *Medicago sativa*, but in the United States *M. falcata* is also reported about equally susceptible with *M. sativa*, and it appears that all other leguminous plants, weed or cultivated, are everywhere quite immune from this disease [7].

It was first observed in 1892 in Ecuador [6], and in England in 1906 near Herne Bay, Kent [11, 12, 13]; in the United States [17], where the disease is localised in a few places, it was first found in 1909 and is abundant only west of Sierra Nevada and the Cascade Mountains [4]. Crown wart is relatively of little importance in Britain, but its sporadic occurrence in France [2], Germany [5], Italy, Switzerland, and comparatively recently in Belgium [9], Portugal [3], Utah [1], Canada [2a], and California [16] seems to indicate that it is more common than reported.

Affected plants in the spring show characteristic whitish warts or galls around the base of the stem at or below soil-level (Fig. 224), so that unless the soil is cleared away the extent of the damage is not always realised. A gall begins as a swelling of one or more of the numerous adventitious buds which arise in succession from the woody crown of the plant, especially of those less protected and developed below the level of the soil. All parts of the buds, including the scales, leaves, and stipules, may become involved in gall formation, but in other cases where buds are only partially infected they may produce weak shoots in spite of gall formation. The general effect at the base of the plant is the development of a large dirty-white excrescence (often as much as 6 inches in diameter, and extending from 1 to 3 inches into the soil) having a more or less rounded contour from which partially infected buds may continue growth, but only to form weak shoots. Unaffected buds close to the galls at first produce normal leafy shoots which, however, soon begin to show signs of wilting and yellowing, especially in hot weather, and these symptoms are helpful in picking out infected plants in the field [7]. After long exposure the exposed galls may turn green and form a firm crust around the base of the stem [4].

When a gall is cut across it shows a number of dark-brown diseased areas surrounded by white tissue, the gall thus presenting a marbled appearance (Fig. 224 B). These brown patches contain the spores of the causal organism, and while most of the galls decay and liberate their spores, a few more deeply situated galls become covered with corky layers and survive the winter. Individual galls continue to develop spores over a long period, and while the spores are capable of

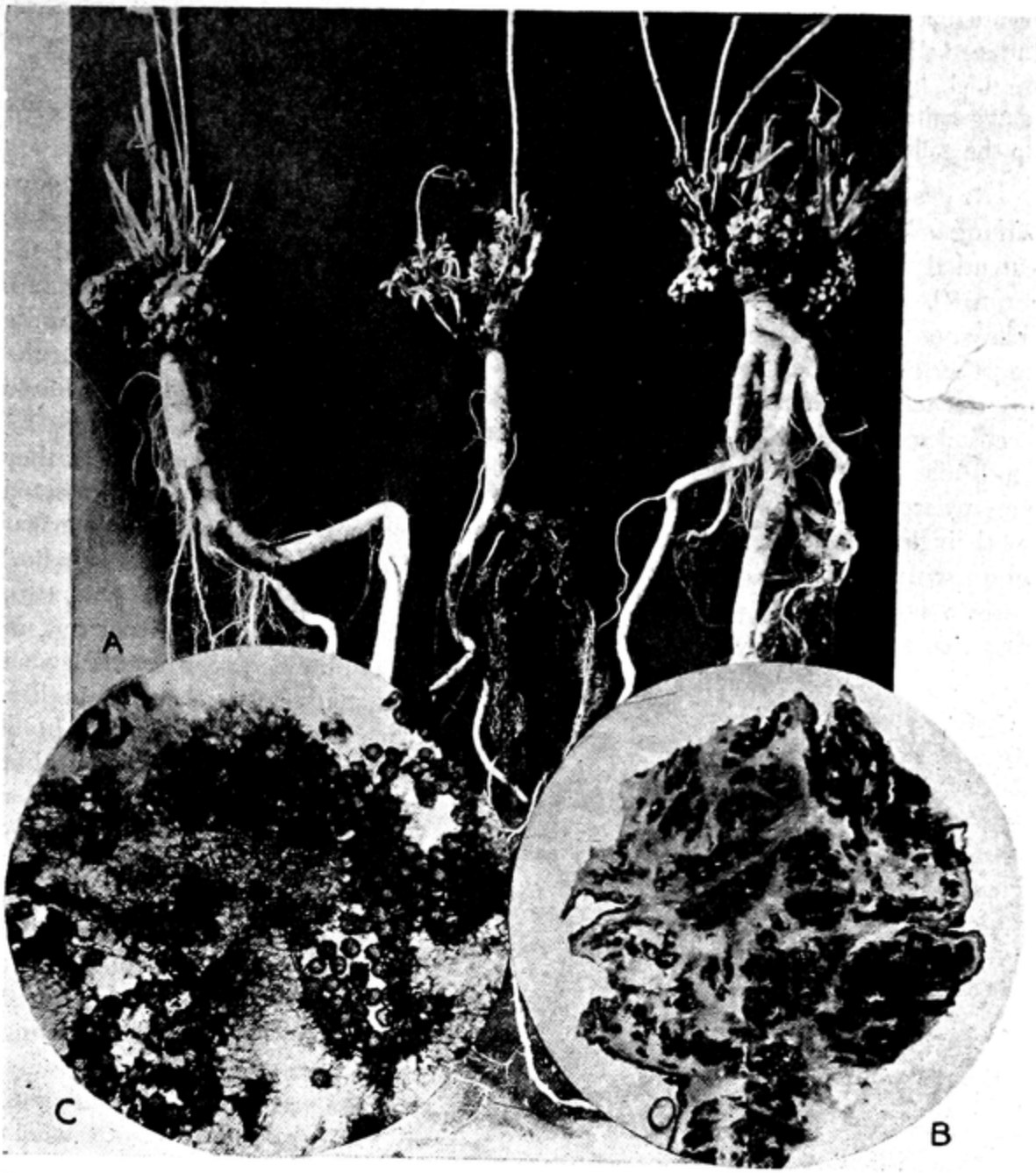


FIG. 224.—Crown wart of lucerne (*Urophlyctis alfalfae*). *A*, centre, normal plant; right and left, infected plants showing warts at the crowns. *B*, section of wart showing the marbled effect. *C*, section of a wart showing the chambers filled with spores (photos by Salmon & Ware, *Wye Reports*)

survival over the drought of summer, in rainy periods they are set free into the soil in great numbers to initiate fresh infections [4].

This disease is caused by the fungus *Urophlyctis alfalfae* [6, 8, 10] of the Chytridiales, a family belonging to the primitive group Archimycetes, of which another member *Synchytrium endobioticum* is described in this book as the parasite causing wart disease of potato, which this disease resembles in many respects (p. 499). The browned areas in the galls contain a number of golden-brown spores which function as resting sporangia and

eventually give rise to zoospores (Fig. 224 C). The spores are somewhat spherical, flattened slightly at one pole, from 30 to 40 by 45 μ in diameter, having a brittle wall about 2 μ thick, lined within by a thin colorless wall (Fig. 225 A). They are not easily germinable under artificial conditions but the action of mould fungi and bacteria in helping to break up the galls appears also to assist in the germination of the spores.(7)

At germination (Fig. 225 B) the spores become converted into sporangia containing a large number of biciliate zoospores which may either be passed into an extruded vesicle before dispersal (15) or, without forming an external zoosporangium (7), may escape into the soil through one or more cracks in the spore wall. The zoospores remain active for several hours and may (17) or may not (7) unite together in pairs before infecting the host ; but nothing is clearly known about the occurrence of a sexual fusion in the life-history of this parasite, nor has the initial method of infection by zoospores been actually observed.

Buds of lucerne become infected during early stages of development as they emerge from the crown. Those developed below soil-level, imperfectly protected by their bud scales, offer easy access to the motile zoospores in wet soil, and penetration usually takes place into tender leaf rudiments if these are not covered by bud scales ; infection may also reach the tissues of the growing point itself. One, two, or three zoospores may sometimes be found within the same epidermal cell, in which they appear as somewhat elongated pear-shaped bodies. The narrow end of each body becomes drawn out into a thin beak or hypha, and the broader end, from which further development takes place, enlarges somewhat while retaining the single nucleus of the zoospore and most of the cytoplasm. This dilated part of the body, which may be referred to as the 'bulb' or 'collecting cell', puts forth, in a terminal position, a narrow, branching hypha of limited growth which appears to fulfil the functions of an absorptive haustorium, collecting nourishment from the host for the development of the bulb. The bulb soon comes to possess some 10 to 15 nuclei derived by division of the single nucleus of the zoospore. Within the top half of the bulb, 2 to 4 convex, intersecting walls arise which, with the wall of the bulb itself, thus form 2 to 4 cells, each furnished with a single

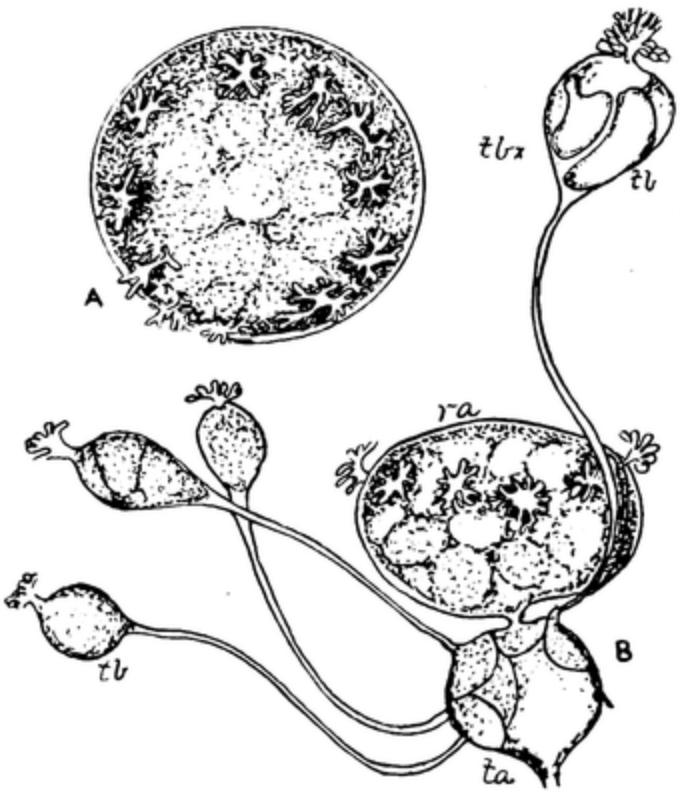


FIG. 225.—*Urophlyctis alfalfae*. *A*, nearly mature resting spore showing a zone of eleven haustoria. *B*, primary 'bulb' *ta* showing four new 'bulbs' *tb* of successive orders and *ra*, new resting spore ; *tbx*, peripheral segments of new bulb *tb*, at top (× approx. 847) (after Jones & Drechsler, *J. Agric. Res.*)

nucleus contributed from the group of nuclei in the bulb, the remainder of which is, therefore, still multinucleate. It is this remaining multinucleate portion of the bulb that is important, for it gives rise indirectly to the spore or future resting sporangium. At a point on the surface of this portion a small vesicle is developed and into it migrate all the contents of the multinucleate central part of the bulb. Eventually, about a hundred nuclei are formed in the vesicle as it matures. The wall of the emergent vesicle now thickens to become that of the spore but not before it has produced, at some three or more points on its equator, short hyphal processes which branch out into short haustoria of the same nature as the haustorium formed at the apex of the original bulb. These branched haustorial processes no doubt assist in the absorption of food material for the developing spore, and when the latter is mature they are discarded, leaving behind very evident scars where they were attached. To return to the developing bulb, each of the 2 to 4 peripheral cells at the rounded end of the bulb (and which took no part in spore formation) gives off a narrow hypha which diverges from the rest so that by penetration of different host cells in the neighbourhood new points of internal infection are established by these hyphae. Each of these exploring, penetrating hyphae, by expanding within the entered cell into an ' apical bulb ' (' collecting cell '), repeats the same history as above described, and the same sequence may be repeated several times. It is the function of these hyphae, therefore, to spread infection within the host. Eventually, however, within a prescribed area in the gall, the process of spore formation is checked. This occurs when, consequent upon the digestion of the host cells within that area, a cavity is formed which makes no further expansion once its delimiting cells develop thickened walls on the sides facing the cavity. Though the host cells in the path of the invading parasite disappear by absorption in advance of the exploring hyphae, once the wall of the cavity is thickened the fungus makes no further inroad into the cells beyond, and meristematic activity stops in the tissues surrounding the older parts of the cavity. It would appear, therefore, that each brown-spotted area of the marbled gall is traceable to a separate infection.

Whilst this development is taking place, the galled tissue, according to the degree of infection, has all the time maintained more or less the shape of the normal bud. The gall develops a vascular system of its own by conversion of parenchyma into tracheids which establish direct communication with the vascular system of the host. The galls are therefore swollen buds, or parts of buds which, through infection, have diverted nourishment to their own abnormal growth, to the great detriment of the normal development of the host.

Water-logged soils and faulty drainage are favourable to the spread of crown wart once the spores are liberated into the soil. During periods of drought the trouble is immediately checked, and the best method for its control is attention to drainage [2, 14].

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Powdery Mildew of Clover, *Erysiphe polygoni* DC.

Powdery mildew caused by *Erysiphe polygoni* is very common on a wide range of wild and cultivated plants which include swede, turnip, pea, clover, and other leguminous plants. It occurs extensively in Britain and North America, especially in the eastern parts of the United States, eastern Canada, and in India (4, 8, 9, 10, 17). The mildew is often responsible for considerable reduction in the crops generally, particularly on those of late peas. On most varieties of clovers it is reported in some localities to reduce the weight and quality of the herbage, while in other places its effects are hardly noticeable (9). Under field conditions in Britain, the aftermath of a clover ley usually suffers more from mildew than the main hay crop (14).

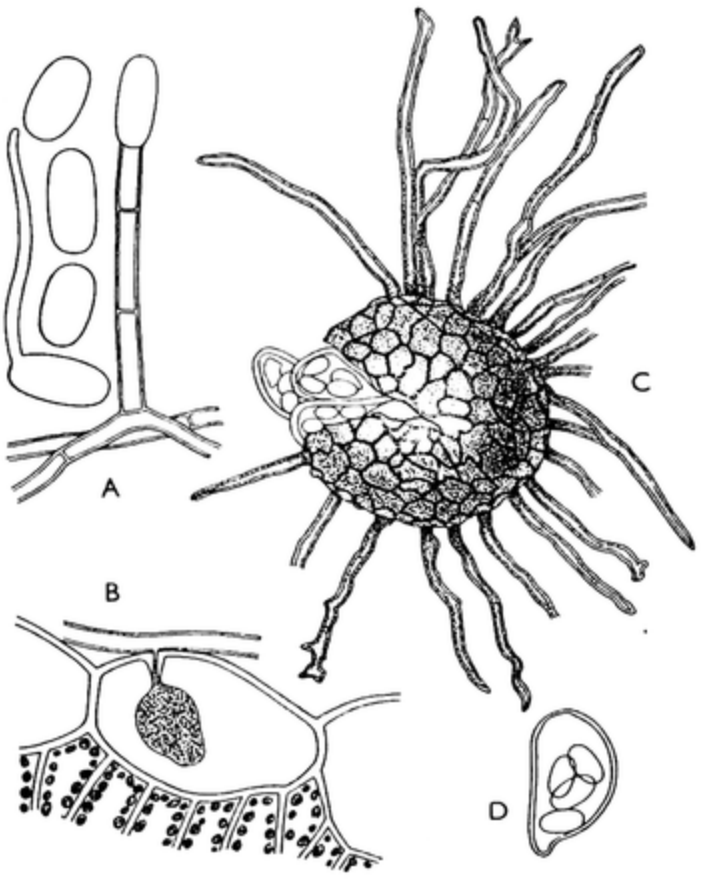


FIG. 226.—*Erysiphe polygoni*. *A*, conidial stage (from *Anthriscus sylvestris*) (× 270). *B*, haustorium (from *Polygonum aviculare*) (× 450). *C*, cleistocarp (from pea) (× 180). *D*, ascus with ascospores (× 270) (after Salmon)

On any of its numerous hosts first signs of the mildew consist of small patches of a thin, fine meshwork of white mycelium, arising at various places on the upper surface of the leaves. These white patches soon join together to form larger areas, and from the superficial mycelium arise upright conidiophores and conidia (Fig. 226 A) in such profusion that the foliage looks as if dusted with flour, and an entire crop may appear white from a distance. Leaves of crimson and red clover often carry heavy growth of the mildew for several

weeks before they become chlorotic and die; leaves and pods of the pea may be covered in late summer with the white mycelium of the mildew [15].

When the production of conidia is about over, giving place in some cases to the development of cleistocarps, the leaf surface becomes patchy yellow, and in parts dark purple or even black, until the entire lâmina is killed. Clover plants attacked during early growth remain small and dwarfed. The stunting effect is believed to be due indirectly to excessive transpiration brought about by the demands of the mycelial webs on the leaves, and it is known that leaves so covered transpire much more freely than healthy leaves [9].

E. polygoni embraces a large number of physiologic races. Specialised races attack pea, swede, turnip, and various other hosts; on red clover at least three physiologic races exist [6, 9, 12, 15, 18].

In many localities only the conidial fructifications are known, cleistocarps being comparatively rare. The elliptic, hyaline conidia (Fig. 226 A) are cut off at the ends of vertical conidiophores one at a time, continuing in succession from below [5, 13]. They are dispersed by wind, and appear to be the chief means of spreading the disease; they are rarely absent from clover fields, though often confined to traces on the lower leaves [14]. The cleistocarpic fructifications (Fig. 226 C) are not common, but often occur on the pea; they are produced only sporadically on clover, and it is doubtful if they play any important part in the spread of the mildew in Britain. They are globose, 65 to 180 μ in diameter; the appendages are brown or colourless, variable in number and length, distinct, or more or less densely interwoven with the superficial mycelium. The asci vary in number from 2 to 8 according to the host on which they are found (Fig. 226 D); they are ovate in shape, and measure from 46 to 72 by 30 to 45 μ; the ascospores vary from 3 to 8, rarely 2, per ascus, and measure from 19 to 25 by 9 to 14 μ [13]. The dearth of cleistocarps is probably due to infrequent infection with strains of the fungus which are sexually compatible, and it is likely that the organism is heterothallic [1]. The cleistocarps appear to be set free from the host when wetted with dew or rain, and after conveyance to a new host become firmly attached thereto by their appendages [20].

While infection in some places may start from conidia which have survived the winter, in other places it is thought that the mycelium itself may be enabled to live throughout the winter, especially on hardy hosts such as the Brassicas, or on the numerous perennial weeds which serve as hosts for the production of conidia to spread fresh infections during summer and autumn [15]. It is recorded from Bombay [17] that the disease on peas may quite possibly be carried internally in the seed, but there is no other evidence that the mildew may be seed-borne in any of its numerous hosts.

The history of mildew infection may be studied by depositing conidia on the upper surface of excised leaves of clover floating on a 10 per cent. solution of sucrose, at a temperature of 20° C. The conidium is seen to put forth a germ-tube which forms an appressorium before direct (not stomatal) penetration of the epidermis takes place. The infection peg from the appressorium is very narrow, and first bores its way through a swelling (' lignituber ') in the inner layer of the epidermal wall (Fig. 227), before its tip expands into a small, round haustorium, which, moreover, appears to be furnished with a sheath [11]; sometimes penetration may go deeper than the epidermis, and haustoria may actually be found in some of

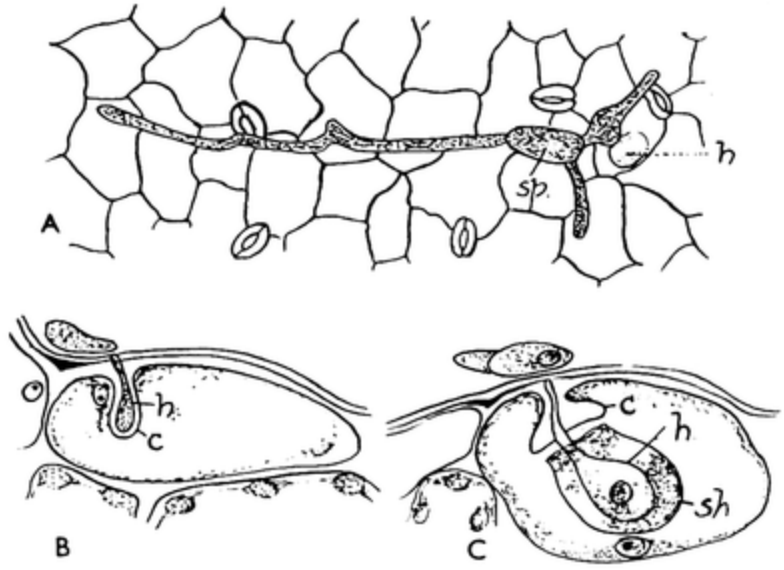


FIG. 227.—Mildew of clover (*Erysiphe polygoni*). *A*, spore *sp* germinating on leaf of clover, showing three germ-tubes and a haustorium *h*; note direct cuticular penetration. *B*, *C*, sections of penetrated epidermal cells showing haustorium surrounded by ingrowth of the host cell wall *c* which, in *C*, forms a collar around the stalk of the haustorium *h*; note the prominent sheath *sh* of the haustorium (after Smith, *J. Agric. Res.*)

the mesophyll cells [11]. Incidentally, it is suggested that the presence of such an internal mycelium may provide for the survival of the fungus over the winter, if leaves are sufficiently protected [9]. After the germinating conidium has secured anchorage by virtue of its first haustorium (about 9 hours after inoculation) it puts forth another germ-tube from the opposite end, and the second tube, by dint of repeated branching, spreads out over the leaf surface to form a small patch of mycelium. This consists of uninucleate cells from which numerous tiny swellings like the appressoria from the germ-tubes develop, and numerous penetrations, followed by the formation of haustoria, occur during the extension of the mycelium over the leaf. In 5 or 6 days after inoculation the mycelium is sufficiently advanced to begin sporulation. Production and dispersal of conidia appears to be much more active during the hours of daylight, in particular from 8 to 12 noon, than in darkness. It is of interest to note that the normal closure of clover leaves at night seems appreciably to reduce the chances of infection [18].

Any correlation between the incidence and severity of mildew on clover and the relative humidity of the atmosphere is difficult to understand, as infections may start and continue during seasons of light rainfall and be equally severe during periods of comparative drought [19]. It is probable that different races of the parasite react differently to changes of the environment, as well as in their attacks on different strains of clovers. The mildew is said to be encouraged by the incidence of cool nights and warm days during middle and late summer [9].

Little appears to be known about the occurrence of this mildew in relation to its attacks on susceptible hosts grown on different types of soils, or on soils of different reaction. On plants of the cowpea in Virginia the disease had a preference for alkaline rather than acid soil conditions [3].

Powdery mildew is difficult to control in the field. In Bombay [17] sulphur dusting of peas proved effective against attack, and this treatment has also given good results on clover in Wales [14] and America [12]. In Canada, some strains of red clover, and some European varieties as well, have shown considerable resistance to powdery mildew [10].

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Black Blotch of Clover, *Cymadothea trifolii* Wolf

' Black blotch ', or ' sooty blotch ' disease, is common on various kinds of clover in Britain, Europe, and America. While the susceptible hosts in Britain include crimson, red, and white clovers, the disease is reported in New York State to be prevalent mostly on alsike and white clovers, red clover being rarely affected [2] ; in North Carolina no fewer than 25 species of *Trifolium* are recorded to be susceptible to this disease [7].

The disease is confined to the foliage, and early symptoms are usually evident towards the end of May. It spreads throughout the summer, and reaches its height in the autumn ; from January onwards the symptoms tend to disappear in the open but may be seen on plants under glass practically all the year round [3]. The disease seldom causes widespread damage in the field, and though epidemics may occur under glass, causing much defoliation, the plants are rarely killed [5]. Early symptoms consist of a few small granular spots, dark olive-brown, almost black in colour, on the under side of the leaves (Fig. 228). The spots may increase in size up to 1 or 2 mm. in diameter, and while mostly confined to the base or margins of the leaflets, they sometimes cover the entire under surface of the lamina. In advanced infection, the leaflets turn brown and wither but may still remain attached to the plant and assume a more or less upright attitude, a characteristic feature which distinguishes



FIG. 228.—Black blotch of clover (*Cymadothea trifolii*). The conidial (*Polythrincium*) stage on clover (photo by Wolf)

them from healthy leaflets, which are laterally extended (3). Sometimes the spots break out on the upper side of the leaf as well, especially on white clover, and often become angular in outline if their growth is checked by the veins (6).

The disease is caused by the fungus *Cymadothea trifolii* (=*Dothidella trifolii*), an Ascomycete of the group Dothideales. There are three types of spores, namely conidia, pycnospores (which are probably spermatia), and ascospores. The conidial stage (*Polythrincium trifolii*) develops from a stromatic mycelium established below the epidermis, and conidia are formed in great profusion from dense masses of conidiophores which aggregate below the cuticle (Fig. 229 A, B). They are liberated to the surface in powdery masses, and continue to be formed in sympodial fashion so that the conidio-

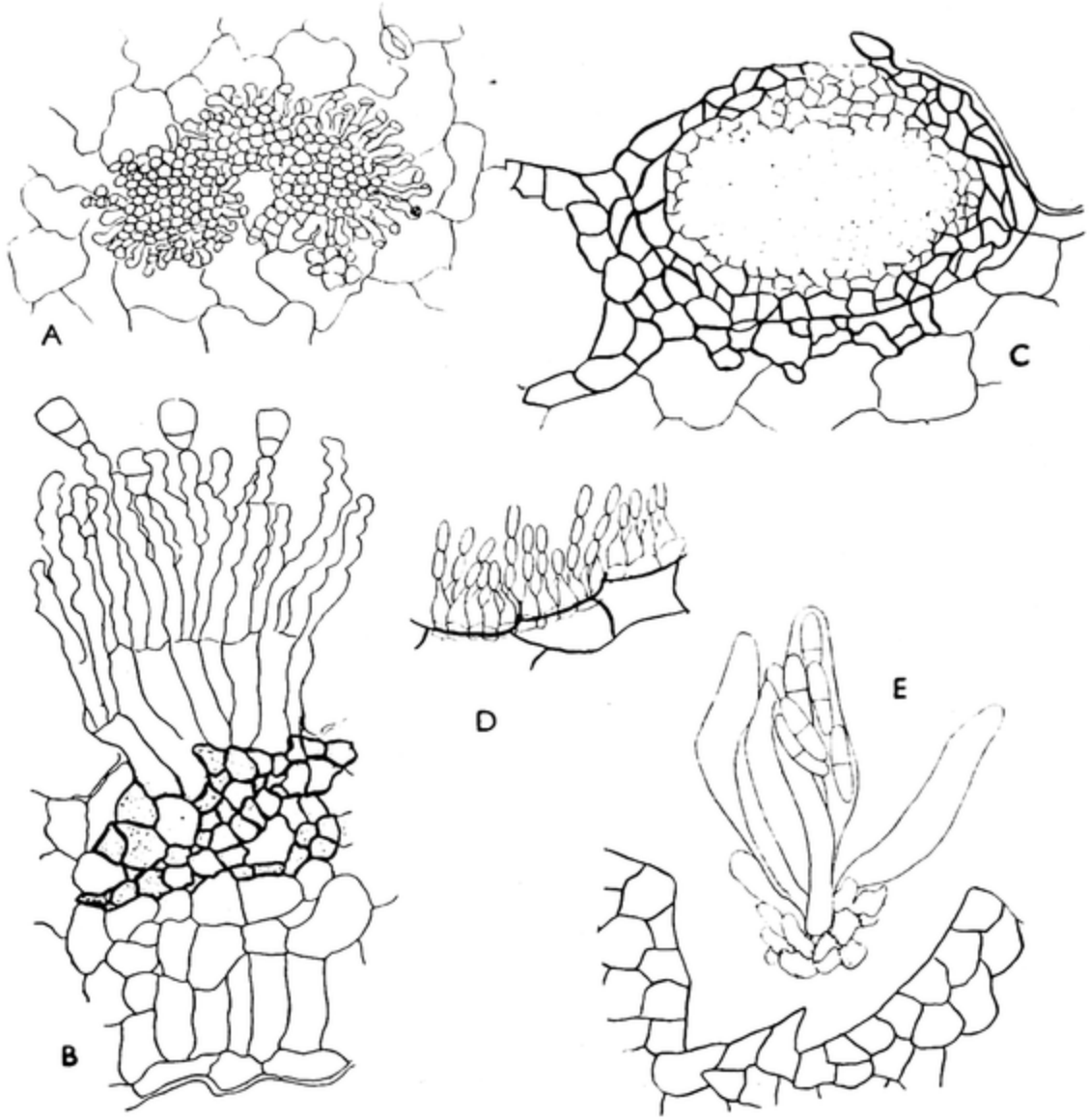


FIG. 229.—*Cymadothea trifolii*. *A*, surface of leaf showing a stroma producing young conidiophores. *B*, section of clover leaf showing conidial stroma with conidiophores and conidia. *C*, pycnidium showing disintegration of its pseudoparenchymatous tissue. *D*, hymenial surface of pycnidium. *E*, asci and paraphyses, crushed from a perithecium (all, ×325) (after Bayliss-Elliott, *Trans. Brit. Myc. Soc.*)

phores assume a characteristic crooked or wavy outline. The dark-coloured conidia are obovate, bicellular, 20 to 22 by 11 to 15 μ; their germination in water is feeble, and all attempts to grow them artificially have failed (1, 2, 3, 7). The production of conidia is generally over towards the end of summer, and thereafter pycnidia or spermagonia (Fig. 229 C, D) and perithecia are developed. The former appear first, either on places previously occupied by the conidia or in clusters close around them. They arise in the mesophyll at points directly below the stomata which are later utilised as ostioles for discharge of the spermatia (3). The tiny, oval spermatia, 3 to 5 by 1·5 to 2 μ, are embedded in slime which exudes to the surface in pale, mucilaginous droplets. Some have observed the spermatia to grow and even to cause leaf infections (1), but others have failed to ascribe any function to them other than that of possible fertilisation in connection with perithecial development (2). Perithecia are initiated within the leaves simultaneously with spermagonia, and although fertilisation by spermatia of ascogonial initials furnished with receptive hyphae has not been detected, perithecial primordia make no further progress unless contact with spermatia has been made (7). The perithecia complete their development on moribund or over-wintered leaves, and the ascospores are discharged in the spring. The ripe perithecia, sunken in the leaves, are not very definitely delimited from the surrounding host tissues, but each is furnished with a blunt, ostiolar beak lined with periphyses. The clavate asci are not numerous; they contain 8 bicellular, fusiform ascospores, 24 to 26 by 7 to 8 μ; there are no paraphyses (Fig. 229 E) (1).

The fungus survives the winter in its perithecial form in decayed leaves, and ascospore infections take place in the spring (3). Primary infections are believed to start in this way from ascospores, but in some localities conidia have been found in a viable condition all the year round and may also be responsible for primary infections (7).

Although numerous species of *Trifolium* susceptible to black blotch were found in North Carolina (7), no evidence was obtained of any morphological differences in the conidia collected from any of these hosts grown together in the same crop (7). In Russia, however, ascospores collected on *T. repens* infected only their own host, and to a lesser extent *T. hybridum*, while those collected from *T. medium* infected neither of the above species, except, sometimes, under greenhouse conditions; these two races of the fungus differed in ascospore dimensions and pathogenicity (4).

As the perithecia are reported to survive on the soil or in clover debris for at least five years, long rotations appear to be necessary before this trouble can be eradicated from the soil.

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Clover Rot, *Sclerotinia trifoliorum* Erikss.

'Rot', or 'stem rot', is one of the most serious troubles which affect leguminous herbage plants. It is considered to be one of the chief factors contributory

to ' clover sickness ', a form of soil deterioration which follows when clover has been grown at too short intervals in the rotation. It is also known that infestation of the soil by the eelworm *Anguillulina dipsaci* and probably the incidence of other undetermined factors are also concerned in this unproductive condition of the soil [17].

As far back as 1898, clover rot, associated with the fungus *Sclerotinia trifoliorum*, was known in Britain to be highly destructive to clover crops in many parts of the country [5]. In America it was first discovered in 1890, in Delaware [6], and in 1914–15 was reported to have caused heavy damage to clover crops in Kentucky [12]. The disease is now widely distributed in Britain, the United States, Canada, Germany, Denmark, and Sweden [6, 11, 12, 13, 23, 29]. Whilst the trouble in this country is common mostly on broad red clover, it is known that late-flowering crimson, alsike, and white clovers are also sometimes lightly affected; trefoil, lucerne, and sainfoin are also susceptible [26]; and the field bean (*Vicia faba*) has recently been found in various localities in Britain to carry a distinct strain of the fungus causing clover rot [14]. Peas and certain weeds of cultivation are reported to be susceptible hosts on the Continent, but not in Britain [2]. In Sweden and Germany it is recorded on winter peas, bush-vetch, and on certain non-leguminous plants, namely, common erodium, dandelion, white campion, field geranium, and forget-me-not [18, 23].

Clover rot makes its first appearance on young plants in the autumn, usually about November, in scattered patches in the field, in the form of a whitish mould covering the stems and leaves, and from which it may later extend for short distances over the surface of the soil. The white mycelium soon becomes yellow and then a brown discoloration sets in which gradually spreads over the leaves until they wilt and collapse to the ground. While only one or any number of the stems of a stool may be attacked and even rotted with disease, the roots may remain free for a considerable time, and during a long spell of dry weather affected plants may throw off the infection by developing new buds which produce perfectly healthy shoots. In severe infections, however, entire plants may be destroyed, and under wet, muggy conditions whole patches of ground may be laid bare [2]. In the final stages of disease the stools are covered at the base with a dark-coloured mycelium, and embedded in the decayed tissues of the stems, and sometimes of the roots, numerous black sclerotia (Fig. 15 B) are found, the presence of the latter confirming the fungus as the causal organism, as distinctive from the eelworm pest [3]. With the development of more and more sclerotia there is a complete rotting of the tissues, and the plants disappear, leaving, here and there, bare patches of soil, in which, however, the resistant sclerotia remain viable for years.

While eelworm trouble and other undetermined factors are, no doubt, productive of much ' sickness ' in certain types of soils, it is generally agreed that *Sclerotinia trifoliorum* [10] plays an important part in clover rot disease. This organism must not be confused with another type of *Sclerotinia* which was first found on seeds of white clover (*T. repens*) imported to England [1], and described by various authors [1, 23, 27]. This seed-borne type is believed to be a different species (it has much smaller sclerotia and apothecia (Fig. 40 B)), and as it may form its sclerotia actually on the seeds [1], there is little doubt that the disease

caused by it is carried by the seed. But there is no weight of evidence that clover rot due to *S. trifoliorum* is ever started in this way from seed infection, though spores resembling those of *S. trifoliorum* have been found on a few samples of red clover seed [17a]. It is quite possible, however, that sclerotia of *S. trifoliorum* are often harvested along with clover herbage and become an admixture with the seed, and may thus, by the sowing of unclean seed, be returned to the soil, in which case they undoubtedly start infections, but they do not develop on the seed of red clover [8, 25].

The whitish mycelium of the clover rot fungus covering the plant may sometimes bear clusters of small, spherical cells or microconidia 2 to 4 μ in diameter; they are abstricted from flask-shaped cells on the mycelium and may also be produced on the germ-tubes produced by the ascospores, as well as on mycelium in artificial cultures [14, 29]. These small conidia are apparently functionless as infective agents, and it has been suggested that they may act as fertilising male cells during the development of the apothecia [9]. It has been established, however, that apothecia may also be developed from monosporic lines of the organism [14, 17a]; in other cases mycelial anastomoses have been observed, so that union between strains which may be sexually compatible may also occur prior to the formation of apothecia [4a, 11a]. Several authors report the existence of physiologic races of *S. trifoliorum*, and host specialisation also occurs [4, 4a, 11a, 14, 17a, 20]. (Cf. Fig. 58.)

The fungus may be cultivated on a wide variety of media, e.g. bread soaked in plum juice [24], or clover leaf decoction; the general appearance at temperatures of 14° to 16° C. is that of a whitish mycelium, the hyphae measuring from 5 to 12 μ wide, with much branching and anastomosing [28]; sclerotia are commonly produced from small masses of mycelium which soon change into olive-green and finally black; sclerotia are irregular in shape and vary from the size of a pea to that of the smallest of clover seed, on some media (Fig. 15 B) [26, 28]. Sclerotia in the field occur chiefly in the spring, on the crowns and upper parts of the root-stock, embedded in the moribund tissues of epidermis and cortex; on the stems of mature plants they may sometimes be found at a height of 20 to 40 cm. above ground, and may thus quite easily be collected at harvest-time and become mixed with the seed during threshing operations [23]. On the decay of the host plant the sclerotia fall to the ground or remain in the upper layers of the soil or may lie as deep as 4 inches below the surface [2]. During the summer they lie dormant and may persist in a viable condition in the soil for 4 years or more [22].

During periods of moist cool weather in the autumn sclerotia germinate to produce apothecia, and during October and November apothecia are not uncommon on old clover leys left unploughed, the sclerotia still remaining attached to dead roots or stems killed



FIG. 230.—Clover rot (*Sclerotinia trifoliorum*). The apothecia on the ground; inset, a stalked apothecium arising from a sclerotium (see also Fig. 15 B) (photo by Sampson & Western, *Diseases of British Grasses and Herbage Legumes*)

the previous winter. Apothecia may arise singly, or in number, up to 10 or more, from one sclerotium ; they are disc-shaped (Fig. 230) and elevated on stalks of varying height, but do not become fully expanded into typical saucer-shaped fructifications unless exposed to the light [4 a, 23]. An expanded apothecium, yellow brown in colour, varies from 3 to 10 mm. in diameter ; the hymenium consists of numerous 8-spored, club-shaped asci, with paraphyses ; the ascospores are hyaline, unicellular, elliptical, and, according to various authors, vary in dimensions thus : 15·23 by 8·17 μ [14] ; 14 to 18 by 8 μ [10] ; 12 to 14 by 8 μ [29]. Ascospore discharge and wind dispersal of the spores take place during mild autumn weather.

New infections of clover crops are believed to originate from ascospores, but under natural conditions in the open it is not at all clear that they are effective unless the host plant is injured or already in a weakened condition [7, 14, 27]. Though primary infections with ascospores are probably accomplished in the autumn by penetration of the outer leaves, the symptoms of disease in so far as they are manifest on the crown are not seen until several months later, when the leaves affected in the autumn have been killed. In the spring the tissues of the crown are much depleted of food reserves and the young growth is, therefore, in a condition which renders the tissues liable to attack. After the fungus has killed the leaves it progresses into the petioles and thence into the crown, which, once infected, allows the fungus to travel up into other shoots on the stool and, perhaps, down into the roots as well. As the tissues disintegrate the fungus breaks out at the surface of the decaying parts of the crown, or roots, and with the development of the sclerotia in these regions the life-cycle is complete. While the sclerotia themselves have not been observed to produce vegetative mycelium in the soil, yet such mycelium may exist saprophytically on clover debris for a good part of the year, but to what extent it can survive in the soil is not known. However, in Denmark, in 1942, mycelium from clover debris was observed to creep over damp soil, and there is little doubt that in the absence of apothecia and ascospores, spread by mycelium can sometimes take place [11 a]. It is recorded that both ascospores and mycelium, when kept comparatively dry, still remained viable after seven months, but there is no evidence to show how long they are capable of withstanding competition under ordinary conditions with other micro-organisms in the soil [23].

The disease is especially favoured by mild winter conditions, and is appreciably checked during periods of dry or frosty weather [2, 15, 19, 27]. The minimum, optimum, and maximum temperatures for the growth of the fungus are 0°, 15° to 20°, and 30° C. respectively. While both acid and alkaline conditions of the soil can be tolerated, growth is best under an acid reaction of *p*H 5·5 [23, 28]. There is no definite correlation between soil-texture and incidence of disease [15, 16], but some have observed the trouble to be more prevalent on permeable and chalk soils than on heavy land [21].

The most important method for the control of clover rot lies in the lengthening of the rotation over a period that will ensure the decay of the long-lived sclerotia and clover debris in the soil, and an interval of from eight to twelve years has proved not to be too long before red clover can be restored with safety. Growth of this plant is not improved by any methods that open up the texture of the soil, and since a loose soil is also conducive to the welfare of the parasite, distinct improve-

ment can be obtained by rolling and consolidating the ground. Further, since the disease is prone to appear when growth is dense and encouraging to high humidity, luxuriant growth should be kept down by sheep-grazing, especially during the autumn months, a practice which also helps in trampling down and consolidating the ground. Excessive use of nitrogenous manures should be avoided, and lime, potash, and phosphates applied to induce strong growth [3]. The disease is apt to be more prevalent when the crop is sown among oats rather than rye, and it is the experience of some farmers that clover rot is especially liable to break out when clover is sown among wheat which has followed beans [3, 26]. In the rotation interval sainfoin, trefoil, alsike, or white clover, owing to their infrequent susceptibility, may be grown alone or with Italian rye-grass, and peas may also be grown [3].

As the sclerotia in the soil are primarily responsible for initial infections, deep ploughing is recommended, as sclerotia do not produce apothecia at depths of 3 inches or more in the soil [29]. If there is any suspicion of the presence of sclerotia among the seed a method devised for their elimination is to mix the seed sample with iron powder, which by virtue of adhering only to the sclerotia may be extracted by an electro-magnet; or the mixture of seed and sclerotia may be shaken up in a 23 to 24 per cent. solution of potassium chloride, in which both sclerotia and defective seeds come to the surface and can be skimmed off, while the good seeds sink; the latter should then be washed and dried quickly [23].

A considerable reduction in the incidence of clover rot has been attained by constant selection and adoption of varieties of clovers which have proved to be best suited to a particular environment; foreign varieties appear to be more susceptible than home-produced kinds. Breeding experiments on resistance to clover rot are hampered by many difficulties, such as the appearance of new physiologic races, the varying degrees of pathogenicity exhibited by certain races of the parasite in different localities, the sporadic nature of epidemics, and the difficulty of submitting the material to a sufficiently severe form of infection [11a, 19a, 26, 27].

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Leaf Spot of Clover, *Pseudopeziza trifolii* (Fr.) Fuckel.

Leaf spot disease is very common wherever clover is cultivated, and in Britain early varieties of red clover appear to suffer more than the kinds which mature later [7]. It also attacks lucerne, and is often locally abundant on white clover, alsike, trefoil, and crimson clover [4, 5, 6]. In America, where it is considered to cause more serious economic losses on red clover than in Britain, it has a wide geographical range, especially in the Northern States and in Canada ; the disease is also recorded from Russia, Italy, Belgium, Germany, and South Africa [1, 2, 4].

The spots on the leaves are variable in size and colour and may range from tiny specks up to 2 to 3 mm. in diameter. They may appear at one or both leaf surfaces, and quite commonly at exactly opposite points on the leaf, as spots of a dark-reddish, purple, drab-olive, or black colour (Fig. 231). A characteristic feature is the irregular fringe-like margin of the spotted area seen when the leaf is examined by transmitted light, but this feature may be absent if the weather has been dry for any length of time. The spots are practically confined to the leaves, but sometimes small, elongated dark streaks may also be seen on the leaf petioles and less frequently on the stems. On the spots, after a rainy period or in moist weather, tiny amber-coloured discs raised slightly above the level of the leaf epidermis appear, which are the apothecial fructifications of the fungus causing this disease.

The organism causing leaf-spot disease of clover was first described in 1816 as *Ascobolus trifolii*, but with the creation of the genus *Pseudopeziza* in 1869 the fungus was called *P. trifolii* (Fr.) Fuckel. So far as is known, no pycnidial or conidial stage is developed ; the small, jelly-like erumpent apothecia contain a dense hymenium of asci and paraphyses (Fig. 41) ; the ascospores are unicellular, ovoid, 10 to 15 by 4 to 6 μ in diameter.

The disease is favoured by wet weather in the autumn, and is severe under cool, humid conditions when growth is luxuriant. The fungus over-winters on affected leaves which have managed to escape complete decay, and from a renewal of spore production from the apothecia on them, fresh discharges of ascospores bring about primary infections, and newly infected leaves in turn produce their apothecia and spores [6].

Early infections within the leaves are laid down here and there within the sub-stomatal cavities, and take the form of small coils of mycelium in which the hyphae, travelling both intra- and intercellularly, consist of uninucleate cells, but those of the ascogonial coils developed below the stomata are multinucleate, and

it is in these situations that the apothecia arise. From apothecial initials arising at one or other side of the leaf, one or more ascogonial branches may travel directly across the mesophyll towards sub-stomatal cavities on the opposite side to give rise again to apothecia, a process which may account for the frequent occurrence of groups of apothecia on both sides of a leaf, at the same spot. During their development the young apothecia become delimited from the surrounding and disintegrated leaf tissues by the formation of much black pigment which gives the spots their colour, and towards maturity the fungal cells of the sub-hymenium increase considerably in bulk so that the apothecia are raised up, the tips of the asci and paraphyses impinging closely against the leaf epidermis, which is finally broken through for spore discharge (3).



FIG. 231.—Leaf spot of white clover (*Pseudopeziza trifolii*). The symptoms on a leaf of white clover (photo by Sampson & Western, *Diseases of British Grasses and Herbage Legumes*) (see Fig. 41)

Little is known about any practical methods for controlling leaf spot disease. It appears that the varieties Vale of Clwyd, American Medium, and Bohemian are less susceptible to it than Italian, French, Swiss, and Silesian varieties. There is no evidence that the disease is carried by the seed (6).

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Violet Root Rot of Clover, *Helicobasidium purpureum* Pat.

'Violet root rot' disease, so called from the colour of the fungal mycelium creeping over the basal parts of the shoots and roots, has long been known on a wide range of hosts. These include many of economic importance, e.g. clover, lucerne, trefoil, black medick, mangold, beet, potato, celery, asparagus, shallot, sea-kale, carrot, strawberry; ornamental plants, e.g. poppy, phlox, crocus; some trees, e.g. sitka spruce, sycamore; and several weeds, e.g. nettle, dog's mercury, cornmint, meadow grass, speedwell, knotgrass, and others (4, 5, 6, 9, 10, 12).

The disease is widely prevalent in many parts of Europe and America. The first record of its occurrence on clover in Britain was made in 1922, on dead stubble of a previous oat crop in which red clover had been sown (11). Clover plants suffering from the disease are stunted in growth, the outermost leaves turn a sickly, yellow colour, wilt and die; in the young shoots at the centre of the crown



FIG. 232.—Violet root rot of clover (*Helicobasidium purpureum*). Red clover showing the fructifications of *H. purpureum* on petioles and stems, at ground-level, and infection cushions of *Rhizoctonia crocorum* on the tap root (photo by Buddin & Wakefield, *Trans. Brit. Myc. Soc.*)

many petioles and stipules are of a bright red colour, while the leaf blades remain pale green or yellow. A closer examination shows that many of the affected plants are already covered at the base with the characteristic coloured mycelium of the fungus causing this disease (Fig. 232). But the trouble is essentially a root rot, and if affected plants are dug up, the purple mycelium, tinged with white at its advancing margin, may be seen just above and below soil-level, closely investing the roots, and even covering the soil, stones, and plant debris in the near vicinity. On the surface of the root the fungus gives rise to small aggregations of mycelium known as ' infection cushions ', and to minute black sclerotia by means of which the fungus survives for long periods in the soil.

In severe infections of clover, the root system becomes dark-coloured, soft, and rotten, and with the collapse of the shoots the latter soon become covered over with mycelium, and the plants are killed. In cases of lighter infection, however, plants may often recover if, by stimulation of growth, new adventitious roots are developed from the crown to replace those lost through disease.

The fungus of violet root rot has long been familiar merely as the coloured, sterile mycelium called *Rhizoctonia violacea*, later changed to *R. crocorum* ; the perfect stage, a Basidiomycete, first described under the name *Hypochnus purpureus*, was placed, in 1885, in a new genus, *Helicobasidium*, on account of the characteristic way the basidia are bent like a crosier, the new combination being styled *H. purpureum* (Fig. 233). The basidial fructifications bear little resemblance to any type of sporophore in the higher fungi, and consist merely of ill-defined, spreading mats of felted hyphae, purplish like the vegetative mycelium and paler around the margin where growth and extensions occur, the whole body, however, changing with age and finally, after drying out, acquiring a drab cinnamon colour. The hymenium consists of a diffused layer of curved basidia which are septated into 2 to 4 cells, from each of which a single sterigma bearing a basidiospore arises, but usually only 2 or 3 sterigmata are developed ; the latter are 10 to 15 up to 35 μ long by 3·5 to 4·0 μ wide at the base, and the hyaline, ovate, usually somewhat reniform basidiospores vary from 10 to 12 (to 15) by 6 to 7 μ ; the spores at maturity are usually binucleate

(Fig. 233 A, B, C). These fructifications are of brief duration, but may often be seen from the end of March till the close of May if warm, moist conditions prevail during this period [2].

Growth in artificial culture may be started from mycelium, infection cushions, mass inoculation by basidiospores, or from the minute black sclerotia, on a variety of media. The slow-growing mycelium, at first colourless, but soon changing to violet, consists of slender, septated hyphae, 4 to 6 μ wide, which branch and interweave to form a matted mycelium. There are several strains of the organism, and some of these produce, but only in culture (Fig. 233 D), tufts of unbranched conidiophores 25 to 35 by 4·5 to 5·5 μ, bearing conidia which are usually globose, from 10 to 16 μ in diameter, but sometimes elliptical or ovate, from 10 to 18 by 9 to 15 μ; the conidia are binucleate (Fig. 233 F) [2]. Growth is favoured at room temperatures up to 25° C., but beyond this degree progress

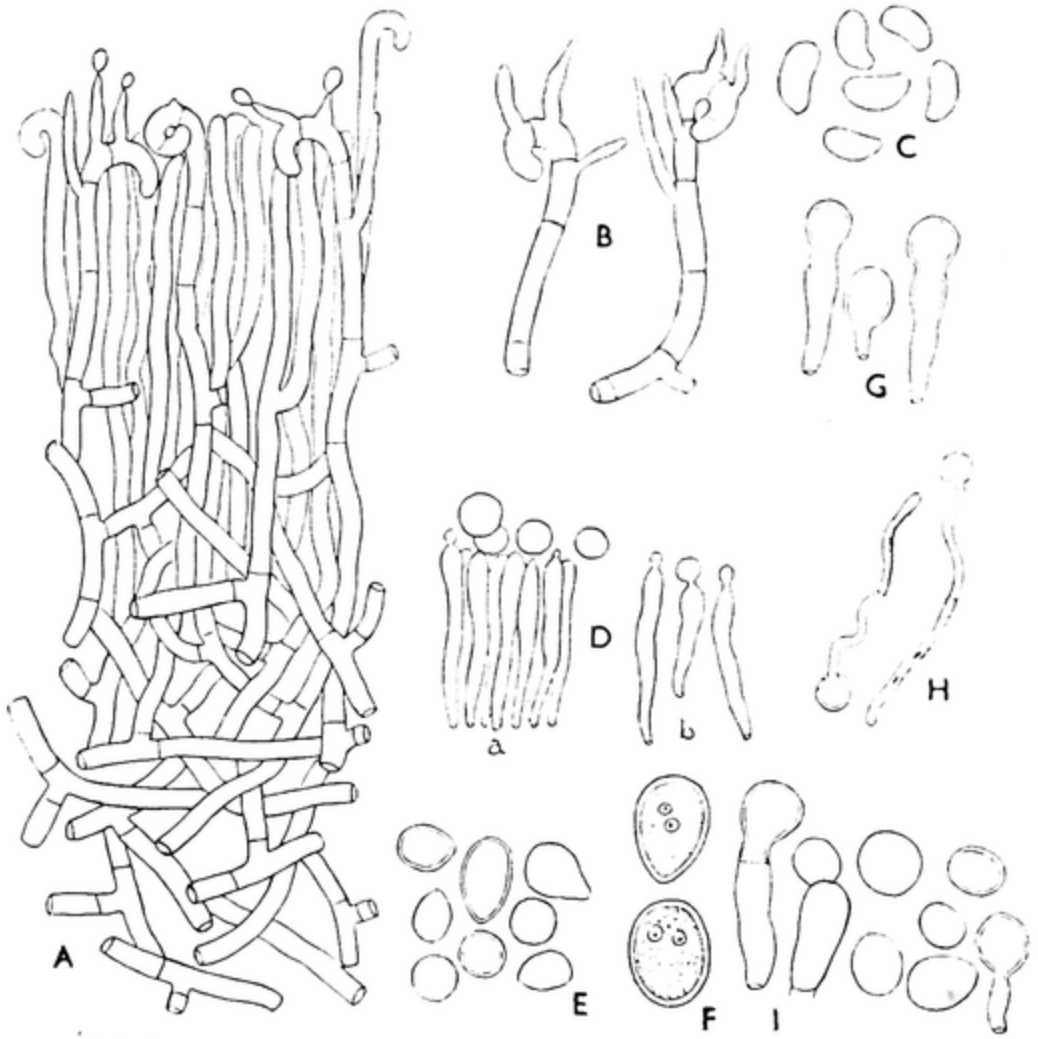


FIG. 233.—*Helicobasidium purpureum*. *A*, vertical section of sporophore showing hymenium and part of sub-hymenial tissue (× 450). *B*, two basidia. *C*, basidiospores (× 500). *D*, a conidial form (*Tuberculina*), as developed in pure culture (a strain from red clover); *a*, portion of conidial layer of pustule; *b*, isolated conidiophores (× 500). *E*, conidia from pure culture (*Urtica* strain) (× 500). *F*, conidia showing two nuclei. *G*, conidia formed in old cultures, not in definite pustules (*Urtica* strain) (× 500). *H*, germination of '*Tuberculina*' conidia (× about 350). *I*, *Rhizoctonia crocorum*, conidia developed in culture (of mangold strain) (× 500) (after Buddin & Wakefield, *Trans. Brit. Myc. Soc.*)

is soon checked because of early staling of the medium [1]. In old cultures, bodies resembling sclerotia may be seen as the medium dries out ; the perfect *Helicobasidium*-stage has not been produced in artificial cultures. Very little appears to be known about the existence of conidia under natural conditions, and of the possible rôle of conidia and basidiospores in natural infections. Artificial inoculations have been successfully performed with certain strains of *Rhizoctonia*, with pieces of the infective cushions, and in a few instances with *Helicobasidium* spore cultures [2].

Survival of the fungus is effected by the sclerotia, and probably by resting mycelium, left behind in the soil after the death of the host tissues. By virtue of the infection cushions developed on the surface of the roots, the fungus is apparently capable of penetrating the cork layers of the root [11], in the same way as it attacks the potato, through the suberised skin of the tuber [8]. In another investigation of the mode of penetration of the potato tuber by the same fungus, it is stated that the investing hyphae enter the tender epidermal cells of young buds on the tuber, or young apices of the growing sprouts, and that root infection occurs by hyphal penetration between epidermal cells [6].

As above stated, the fungus is extremely slow of growth in culture and the same applies to its development within the host. Young plants grown from seeds of red clover sown in sterilised soil in July, after being inoculated by applying a pure culture of the fungus to the roots on 3rd September, showed many of the plants by 11th April typically attacked by violet root rot, and infection cushions were observed both on the tap roots and on some of the lateral roots [1].

A high degree of humidity is necessary for the progress of this disease in the field, but a sandy, well-aerated soil is more favourable than a heavy soil, though a certain degree of moisture must always be present to maintain the delicate mycelium in growth on soil and plant debris. The organism survives from season to season in the soil as sclerotia, and probably persists also for variable periods practically all the year round on one or other of its numerous weed hosts, but whether as sclerotia, resting mycelium, or spores is not known. Some strains of the organism, from different hosts, vary considerably in virulence, while others appear to be non-pathogenic or only feebly parasitic on clovers, but their existence on common weeds of cultivation must always be considered a menace to clover crops [3].

Owing to the wide occurrence of the fungus of violet root rot on so many hosts, and the persistence of the sclerotia over undetermined periods in the soil, its eradication is obviously very difficult. Fortunately, the disease is not serious in Britain, and long rotations with non-susceptible crops will help to starve the organism out of the soil.

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Clover Scorch, *Kabatiella caulivora* (Kirchn.) Karak.

'Scorch' disease attacks mainly red clover, though alsike, white, and shaftal clovers may be artificially infected [5]. It is responsible for a considerable loss of leafy shoots and, by attacking the stalks of the flower-heads, interferes greatly with seed production. The disease is fairly common in Britain, especially in the wetter, seed-growing districts in the west. It occurs in many parts of Europe [1, 2, 4, 7], in Canada, and the northern areas of the United States where it is apparently more harmful than in other parts [3, 6].

The disease causes a blackening and breaking of the stems and withering of the leaves, and affected clover fields as a whole appear as if scorched; injured leaves soon wilt, become dry and brown but may still remain attached to the plant (Fig. 234). Fresh shoots which are developed prematurely to replace those lost through disease are also in turn attacked and destroyed. Sometimes when a second cut of the crop has become established it may suffer even more severely than the first [6]. Farm crops of clover are sometimes infected in the first seedling year but in general the disease attacks the plants in the first harvest year [5].

First symptoms of scorch on the stems consist of small, elongated, ellipsoid, light-brown spots with a rather wide, dark-brown or black margin, and may later vary in length from $\frac{1}{8}$ to 3 inches, or more. They are sunken and shallow at first, but may soon become quite deep so as to penetrate into the central cavity of the stem, and as these lesions become dry, the sides tend to roll inwards and the slits open wider. Frequently only a part of the stem may thus be destroyed on one side, but sometimes the entire stem may be completely girdled by a more shallow depression, with the result that the stem collapses to the ground and all leaves and shoots above the lesion are killed. Similar lesions develop on the petioles, and as these are generally of the girdling type the leaflets hang limply and wither for lack of water; occasionally such girdling lesions may be seen on the short stalks of individual leaflets (Fig. 235) [6]. Spots on the leaves are dark-coloured and are clearly defined on both surfaces. Lesions which occur on the stalks of the flower-heads interfere greatly with the development of the flowers and seed production [8].



FIG. 234.—Clover scorch (*Kabatiella caulivora*). Red clover affected with scorch (photo by Sampson, *Trans. Brit. Myc. Soc.*)

Scorch is caused by *Kabatiella caulivora* (Melanconiales). Sporulation from small white pustules is profuse on

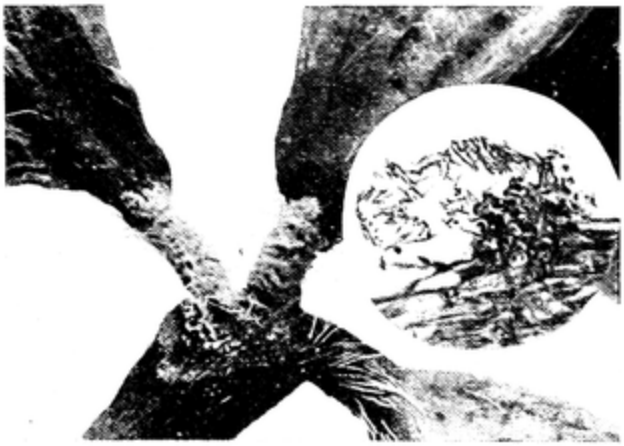


FIG. 235.—Clover scorch. Stalks of the three leaflets of a leaf of red clover; two are attacked by *K. caulivora*; the third is free from disease (photo by Ware). Inset, an acervulus of the fungus on red clover (photo by Sampson, *Trans. Brit. Myc. Soc.*)

all lesions. Unicellular, oblong, slightly curved conidia are borne in clusters of 3 or more at the ends of broad, blunt conidiophores. The conidia are hyaline and measure from 8 to 24 by 2·5 to 4·5 μ (average, 14·6 by 3·5 μ) [4]. In cultures, smaller-sized spores within pycnidia have been observed, but neither these nor a perfect stage in the life-history of this organism has so far appeared under natural conditions [4, 7]. Growth in culture produces colonies of a slimy consistency, the cardinal temperatures for growth of the mycelium and germination of the spores being 4°, 20°, and 28° C. [7]

The fungus remains alive in infected leaves in the field during the winter, and is believed also to be capable of surviving in the soil [7]. While there is no evidence that the disease can be carried on the seed, it has been shown experimentally that conidia may survive on dry seed for at least eighteen months and are capable of infecting the cotyledons and first leaves of the seedlings [4]. But infected leaf stalks bearing spores have been collected in the open as late as December [4], and the fungus was found to tolerate temperatures considerably below freezing point [7]. The spores from the over-wintered leaf stalks infect neighbouring plants and young seedlings. Inoculations of young shoots by spraying them with a spore suspension showed that the fungus travelled for some distance between cuticle and epidermis before passing in between the deeper cells which soon developed browned walls and blackened contents. The mycelium is scanty until the host tissues are destroyed, when coarse strands are formed, which eventually give rise almost directly to the widely dilated conidiophores already mentioned, so that no stroma is developed, the broad conidiophores running more or less parallel with the epidermis before finally breaking through to the surface [7]; no setae are formed [4].

The disease is favoured by humid conditions such as prevail when the crop is too densely grown. In certain areas it breaks out periodically after years of absence and appears to be stimulated by wide fluctuations of temperature, with moderate but frequent rainfall [1].

Different strains of red clover vary in their degree of resistance to scorch. In general, Italian and French, and English Broad Red clovers suffer severely, while Early Vale of Clwyd, English Late, Montgomery, and Cornish Marl are not so heavily attacked, and the most promising method of control lies in a selection of new strains, showing still greater resistance [5]. More distant planting, mowing, and rotations, or mixing with resistant or immune types such as alsike and white clover, will help to reduce the amount of disease [7].

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Choke Disease of Grasses, *Epichloe typhina* (Fr.) Fr.

This disease, which affects the development of the panicle or inflorescence, is very common on many kinds of hedgerow and pasture grasses in Britain. It is not found outside the *Gramineae* and seldom occurs on cereals, but in 1921 it was reported in the Soviet Union of Russia on wheat and rye as well as on pasture grasses [4]; it is not common in America. While it has been reported in some years and localities to cause considerable damage to certain kinds of pastures [2], it does not usually reduce the amount of herbage, and appears to do little harm to pastures generally. Its economic importance lies chiefly in that, by destroying the panicles before seed formation in some types of grasses, and in others by establishing infection of the seed itself, it affects adversely crops cultivated for seed production [5, 6].

The disease stifles the development of the panicle, 'choking' it while still enclosed in the leaf sheath, and generally preventing its emergence. But its effect is not the same on all grasses. In some types investigated, namely cocksfoot (*Dactylis glomerata*) and red fescue (*Festuca rubra*), the disease in cocksfoot entirely inhibits the emergence of infected panicles from the sheath, so that no seed is formed (Fig. 236 B), but in the case of fescue, in which infected panicles are not prevented from emerging, all grades of infection of the inflorescence may be found (Fig. 236 C), from stages where the panicles have, by rapid growth, entirely outpaced infection travelling up the stem, the panicles remaining fertile, to the other extreme where infection has all the time kept pace with host development, infection being found in all parts of the flower, seed included. Infection in fescue, and probably in many other grasses susceptible to 'choke', is therefore systemic, culminating in the production of infected seed which transmits the disease [3, 7]. But in all cases infection resides within the host, the fungus perennating in the rhizome and vegetative organs, in which it may remain latent for variable periods before reproduction. In perennial grasses, like cocksfoot, the disease appears to spread exclusively by vegetative propagation of the plant, while in others, like fescue, transmission by infected seed appears to be the rule. An instance is recorded of a single plant of cocksfoot supplying, when broken up, 287 propagants, all of which, with one exception, produced the disease in the following season [3].

'Choke' is caused by the Pyrenomycete *Epichloe typhina*. Conidial and perithecial fructifications are formed, in sequence, on the same stroma on the surface of the leaf sheath; and although the fungus is perennial within the host, the reproductive phase lasts only for about three months, and only during this period can the disease be detected by the casual observer in the field (Fig. 236).

Infected plants of cocksfoot, from May to July, are seen to be well distributed

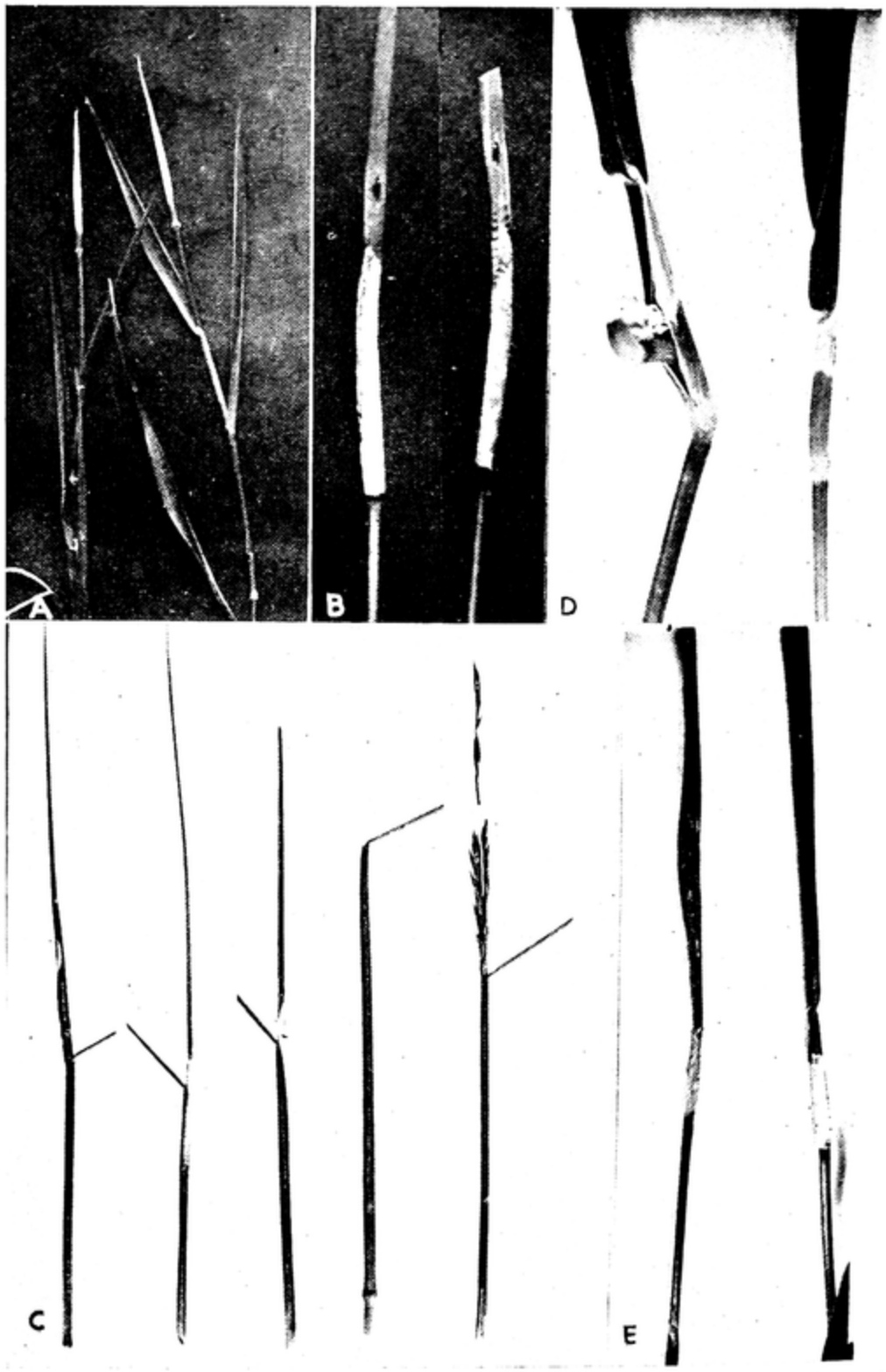


FIG. 236.—Choke (*Epichloe typhina*). *A*, the conidial stage on *Agropyron repens*. *B*, the perithecial stage on *Dactylis glomerata*. *C*, on *Festuca rubra*; note partial development of the inflorescence on the specimen on extreme right. *D*, on *Holcus mollis*; note the extrusion of an aborted inflorescence, a common feature of the infection in this grass. *E*, on *Agrostis canina* (*C*, photo by Sampson; *D*, *E*, by Walton)

throughout the field. The infection is not usually visible on this host during its first harvest year, and even during the second year of growth some panicles are not prevented from producing viable seeds, but by the third year the disease has got such a hold on the plant that infected panicles are entirely suppressed. It is clear, therefore, that infection remains latent within the host for several years, no external symptoms of disease appearing, until the plant presumably has reached a certain state of maturity [6]. It is very unusual for cocksfoot to extrude an infected inflorescence, and when on rare occasions infected plants of this host produce panicles in the same manner as healthy plants, the inflorescences are (with very few exceptions) normal and healthy, producing sound seed. The first external sign of infection consists of a delicate white mycelium covering more or less completely the entire leaf-sheath of a young tiller, and if the shoot is severed in this region the mycelium is seen to clog all the parts like a packing of fine wool within which the axis of the suppressed panicle has been trapped (Fig. 237). The young panicle failing to emerge degenerates within its sheath. In red fescue, however, and more rarely in other species, the panicle is usually extruded only to be covered with white mycelium, the same as on the leaf sheath. The external mycelium in all cases forms a compact, white, smooth, and waxy stroma on which conidia are developed first (Fig. 237) and perithecia after (Fig. 238). The first appearance of the conidia on the leaf sheath usually coincides with the exsertion of panicles on healthy plants.

The conidia, arising on fine, undifferentiated hyphae, are small, elliptic, and hyaline, from 4 to 5 by 3 μ in diameter. With the disappearance of the conidia the same stroma increases in thickness, turns yellow and then orange, and develops a large number of sunken, densely aggregated perithecia, each furnished with a papillate ostiole. Each perithecium is closely filled with long, somewhat curved, club-shaped asci ; each ascus is 8-spored, with filiform, multiseptate ascospores about 2 μ in diameter, almost as long as the ascus (Figs. 42 C, 237, 238).

The fungus grows well on a variety of media [3, 4] and goes through the whole cycle, producing conidia and perithecia, but the latter fail to produce ascospores. Conidia collected from *Glyceria nervata* in Michigan in the Autumn of 1928 were viable for 10 to 14 days and apparently functioned as soon as they were exposed, from the sheath ; they were observed to germinate in 15 to 18 hours, at 18° to 21° C. but light retarded their growth [1]. The production of perithecia on the natural host is highly variable and appears to be influenced in part by the type of host and in part probably by particular strains of the fungus, for there is evidence that the disease is not biologically identical on all its hosts [3]. Thus, it is characteristic of the fungus on cocksfoot to produce perithecia in abundance but on red fescue these fructifications are comparatively rare ; and we have seen that while red fescue spreads the disease by planting of infected seed, transmission in cocksfoot is mainly from infected propagants, seed infection in this plant being exceedingly rare [3, 6].

Though conidia and ascospores germinate easily in artificial culture, it is remarkable that neither kind of spore appears to be capable of reproducing the complete life-cycle of the parasite on any of its host plants. Since the conidia are

produced in great profusion at the time of flowering, it might be thought that direct infection of the flowers might possibly take place at that time, but this, seemingly, does not occur. Moreover, *Epichloe typhina* matures its ascospores so early (in cocksfoot they are ready for discharge at end of June, about a month after the first appearance of conidia) that these spores, too, might be expected to infect the flowers ; but there is no definite evidence that the panicles are directly

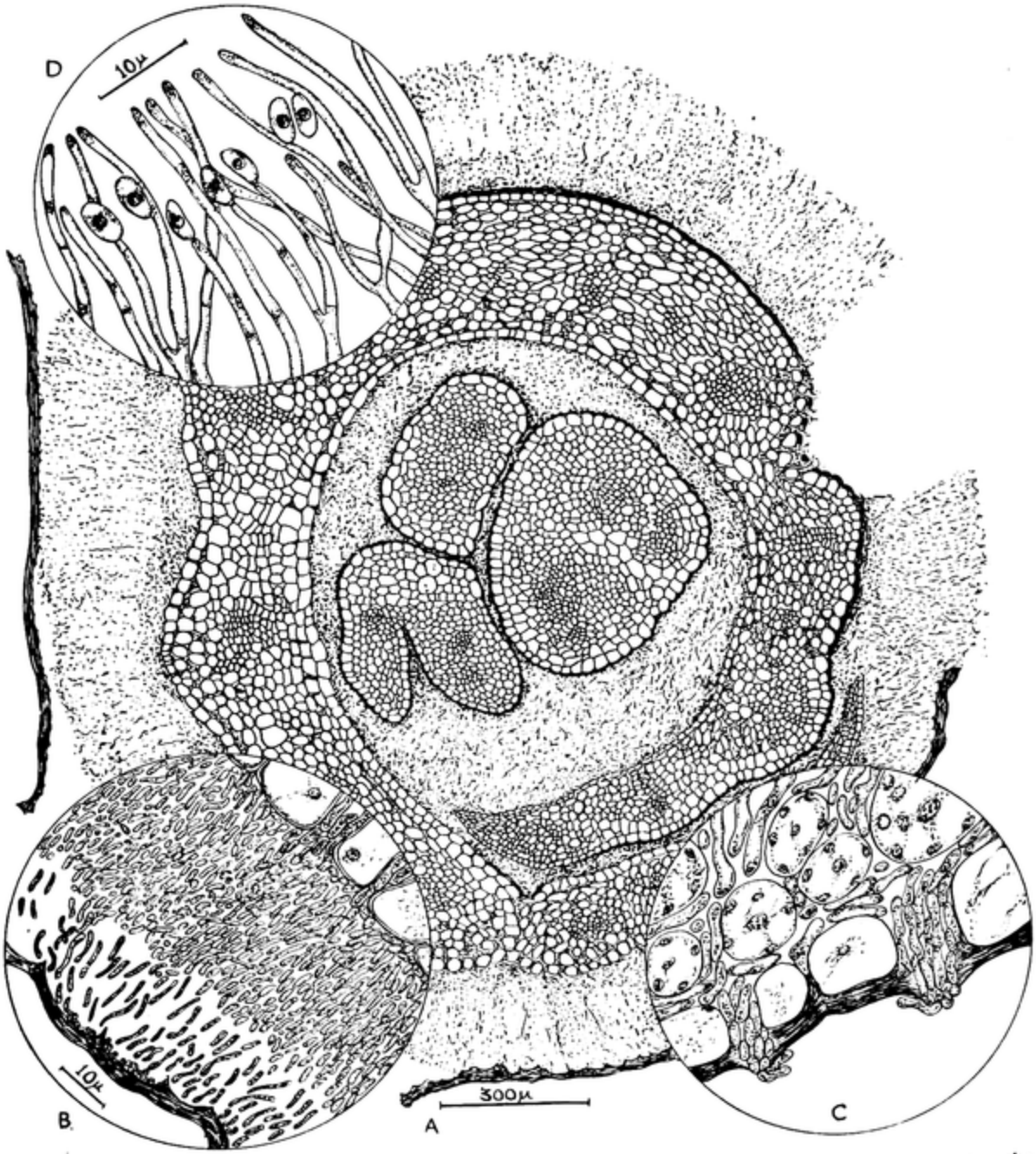


FIG. 237.—*Epichloe typhina*. *A*, transverse section of timothy-grass, *Phleum pratense*, showing the conidial stage on the exterior of leaf sheath, the fungal stroma lifting the cuticle (diag.). *B*, early formation of the branched conidiophores from the stroma. *C*, the mycelium breaking out between the epidermal cells and through the cuticle. *D*, the conidiophores and conidia.

infected in this way, and healthy plants growing together with infected ones have not been seen to contract the disease [3]. Grasses in general are, of course, exceedingly variable with regard to the period of flowering and duration of anthesis, and the opportunity for infection at time of pollination, though brief, may yet be long enough to set up primary infections in the field. Though the perithecial stromata of *Epichloe* appear to be ill-adapted for over-wintering of the fungus (the asco-

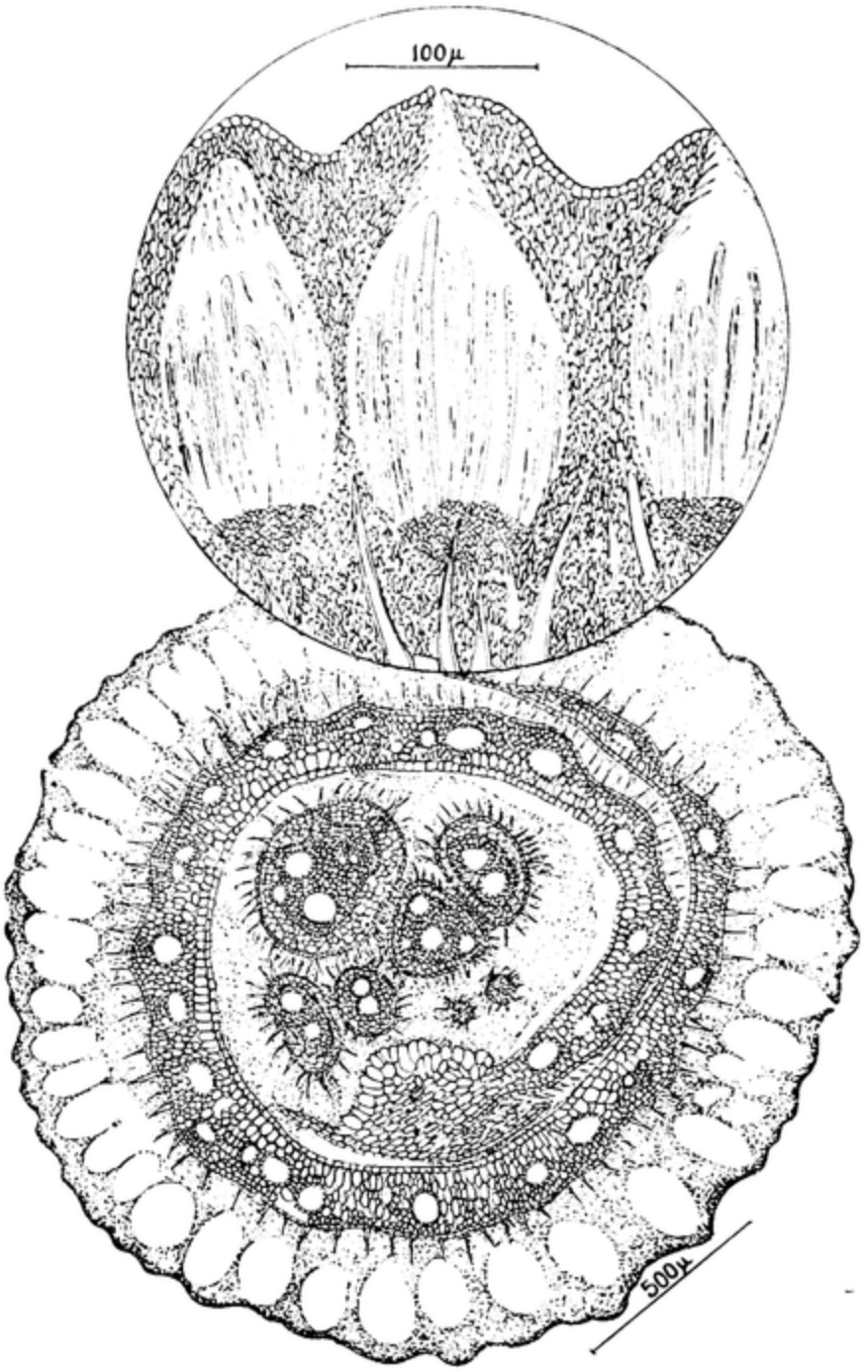


FIG. 238.—*Epichloe typhina*. Transverse section of cocksfoot, *Dactylis glomerata*, showing the perithecial stage; top, details of the perithecia, showing long narrow asci with needle-shaped ascospores (see also Fig. 42 C)

spores, in fact, being found not to be viable the following spring), it is suggested that since they mature during the summer they might sometimes infect the tillering buds at that time. If this is the case, mycelium probably remains dormant in these buds to produce the primary infections in the following spring [4]. Further information about the mode of initial infections with *E. typhina* is awaited with interest.

The fungus is present in all parts of the vegetative host except the roots, and in all parts of the flowers. It occurs at all seasons in tiller buds, leaf blades, leaf sheaths, root-stocks, and may be found in creeping stems and bulbils of susceptible grasses possessing these diverse habits of growth (Fig. 96) [3]. The mycelium within the plant is not easy to detect as it consists of uniformly narrow hyphae (1 to 2 μ in diameter) which run their course for considerable distances in the host without branching, and cross-septa are infrequent. It is mainly intercellular in all tissues and, when it comes to the surface for stroma-building, it wedges apart epidermal cells, and may also be found at that time inside parenchymatous cells and xylem vessels.

In such a type as red fescue, where the fungus finally enters the seed (Figs. 96 C, 239), the mycelium can be found in the pith of the inflorescence axis, thence passing into all parts of the flowers, penetrating the rachilla, glumes and pales, filaments and anthers, and all parts of the ovary, even the branches of the style [3]. But all degrees of flower-infection are met with, thus stamens may be so badly diseased that no pollen is formed, or infection may only be present in the anther-wall leaving the pollen normal, and in other cases bits of mycelium and pollen mother-cells in active division may reside together in the same pollen-sac. The ovaries, too, may be similarly affected in greater or lesser degree. In the ovary the mycelium occurs mostly between nucellus and integuments; in mature grains it is chiefly outside the aleurone layer, around the embryo, and may also penetrate the endosperm (Fig. 96 C).

The above histological details have been established in connection with the systemic infection of red fescue. From planting of infected seed, infection is

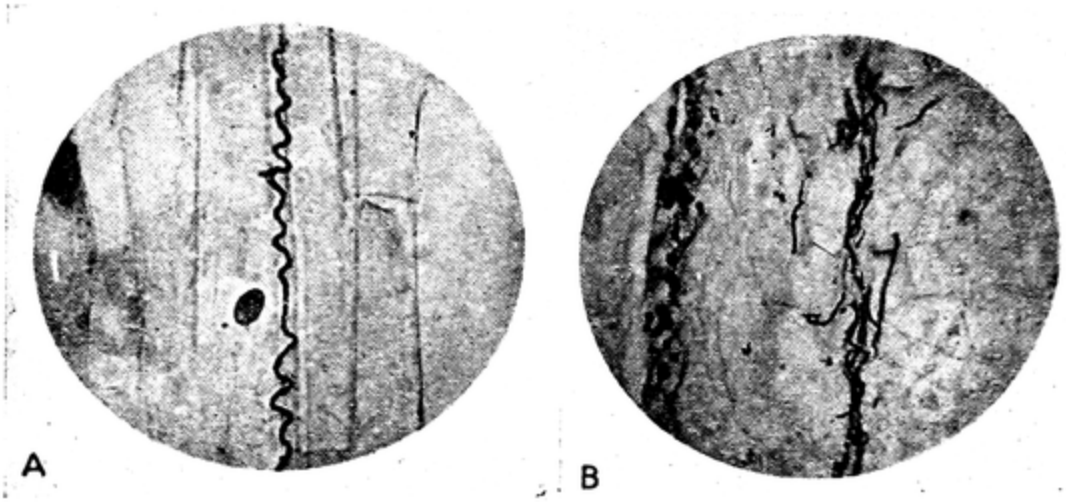


FIG. 239.—*Epichloe typhina*. *A*, mycelium in the coleoptile of a seedling of red fescue. *B*, the mycelium in the seed of same (photos by Sampson; *A*, *Trans. Brit. Myc. Soc.*)

believed to start during germination, from intraseminal mycelium lying near the embryo, and invasion is very complete even in plants which may show no signs of the fungus externally. In this host, therefore, the female parent is naturally the one to transmit the disease, but since the pollen may also become contaminated with the fungus whilst in the anther, it is not impossible that infection may be carried to the ovary at pollination, but this unusual procedure in plant infection has not been established here (3).

Little is known so far about the control of choke disease of grasses, but the production of healthy seed appears to be the first need.

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Diseases of Turf, *Calonectria graminicola* Wollenw.; *Corticium fuciforme* (Berk.) Wakef.; *Sclerotinia homoecarpa* F. T. Bennett

Grass seedlings, and various grasses which compose turf under intensive management on golf courses, lawns, sports greens, etc., are often attacked by various fungi. The commonest of these are *Calonectria graminicola*, an Ascomycete of the group Hypocreales, better known in its conidial stage as *Fusarium nivale*, in its relation to the disease on grasses called ' fusarium patch disease ' or ' snow mould '; *Corticium fuciforme*, a Basidiomycete, causing ' corticium disease ' or ' red thread '; and *Sclerotinia homoecarpa*, an Ascomycete (which rarely fruits in the open) causing ' dollar spot ' disease. There are numerous other fungi, and probably many factors unconnected with fungal organisms, which are also responsible for the deterioration of ' managed ' turf (10a, 13, 16, 18).

Snow Mould, *Calonectria graminicola* Wollenw.

Snow mould of turf, so called from the white cottony masses of mycelium which cover leaves and stems of grasses attacked by *Fusarium nivale*, occurs on all types of ' managed ' turf, but is rare on common turf consisting of indigenous grasses. In Britain this disease may be seen from May onwards, numerous infections occurring about September, but is not confined to this period (13, 14). The same fungus also causes a pre-emergence blight, a root rot, and less commonly, head blight of cereals. It is well known in Europe (1, 10, 15, 16), in the northern parts of the United States, and in Canada (7, 8).

The disease appears in small circular patches which vary from the size of a penny to areas of a foot or more in diameter, showing brown, dead or dying grass in the centre, and extending in a yellowish border around. In old patches, young shoots may pierce the centre of the browner area and extend by growth so that

sometimes annular patches of the disease are found. The annual meadow grass (*Poa annua*) appears to be the worst attacked, followed by creeping-bent (*Agrostis stolonifera*) and the fescues, *Festuca rubra* and *F. ovina*.

Under damp conditions the mycelium of *F. nivale* may be seen developing on the leaves and spreading from blade to blade, forming patches of a faint pink colour. The salmon or salmon-orange conidia arise in sporodochia, and finally become rufous in colour; they are sickle-shaped, 1- to 5-septate, but mostly 3-septate, 22·5 to 25·5 by 3·25 to 4·1 μ [1], averaging 23·6 by 3·5 μ; there are no chlamydospores [2]. Perithecia of *Calonectria*, found on cereals, are sunken, free or gregarious, spherical, dark brown, 160 to 300 μ in diameter; asci numerous and spindle-shaped are 50 to 60 μ in length, with long paraphyses; ascospores biseriate, fusoid, 1- to 3-septate measure from 12·5 to 16 by 2·8 to 3·5 μ [1].

The fungus appears to survive the winter as dark-brown mycelium embedded in grass residues, and is capable of withstanding very low temperatures, as low as −20° C. [2]. The optimum temperature for growth of the fungus in Britain is between 20° and 21° C., with little growth at 32·5°. In temperature relations as well as in size of conidia, strains of this fungus on the Continent are clearly different from the British strain and appear to tolerate much lower temperatures, a feature which seems to justify the name 'snow mould' applied to this disease by Continental authors who describe it on cereals covered over with snow. In artificial culture the *p*H limit for growth lies between 2·5 and 13·0, the optimum being between 6·5 and 6·9; the spores cease to grow below *p*H 5·2.

It is generally observed that snow mould frequently follows upon intensive treatment of the turf with nitrogenous fertilisers which encourage soft growth, and manures rich in soluble phosphates and organic nitrogen applied in the autumn also favour the disease. It is recorded that bowling-greens on sea-marsh turf in Cumberland and Lancashire — where the turf exists under naturally alkaline conditions and low nitrogenous supply — are adversely affected when leaching of lime and treatment with unbalanced dressings are resorted to. Such changed conditions render the greens very liable to attacks of snow mould [3].

The development of aerial mycelium and spores is greatly favoured by a moist atmosphere, and closely matted grasses which retain moisture are very prone to develop the disease. In Britain, cultivated areas in the south are more affected than in the north, one reason perhaps being the need for heavier watering of the greens in hot weather owing to the higher prevailing temperatures in the south. Greens surrounded by trees are often severely attacked and old-established turf where soil action is poor is more liable to develop snow mould than newly laid grass [9].

To check the disease, if the turf is very matted and fibrous, deep forking and use of the spiked roller are advisable. Long growth should not be left in the autumn, and instead of forcing with manures the greens should be kept rather on the poor side in regard to management. Treatment with fungicides such as Bordeaux mixture (with malachite green), corrosive sublimate, or calomel is also recommended. It is advisable to spray the whole green, preferably during dull weather or in the evening, after the customary watering, using preferably a knap-

sack sprayer; malachite green, added in a dilution of 1 in 20,000 parts, with Bordeaux mixture reduces the disease and at the same time increases the vigour of the grass [3, 9]. A distinct difference in susceptibility to snow mould has been found among various strains of *Agrostis stolonifera*, but little appears to be known about any other turf grasses except that in some localities *Lolium perenne* has showed marked resistance [15].

Corticium Disease or Red Thread, *Corticium fuciforme* (Berk.) Wakef.

'Corticium' or 'red thread' disease is also a common trouble of lawns and sports greens. In Britain it may be seen usually from September to October, but may occur earlier or later according to the season, and may persist if the winter is mild. Sometimes the disease lasts only for a few days to a few weeks, causing damage only to the leaf tips and resulting in a mere thinning-out of the turf without doing any permanent harm, but at other times it is quite as serious as snow mould, persisting in patches which become brown, and later, bleached white as if killed by drought.

Greens and lawns in which red fescue is dominant suffer severely from this disease, but other grasses, *Agrostis tenuis*, *Poa annua*, *Lolium perenne*, as well as *Holcus mollis*, *Bromus mollis*, and *Agropyron repens*, are also affected.

Corticium fuciforme, the causal fungus, grows on the leaves in a very characteristic manner, producing from a fine mycelium, pink coral-like threads or spicules projecting more or less at right angles to the leaf, each "like a minute alga, about ½ inch long when dry" [6]. These spikes, formed near the tips of the dying leaves, are gelatinous at first but later become dry and brittle and are easily detached. They consist of parallel, septated hyphae tinged pink, showing frequent anastomoses and clamp connections. Owing to their gelatinous nature they are frequently found to bind the blades and stems of grasses together and may so extend by vegetative growth as to spread the disease to other plants by contact.

The fructifications of *C. fuciforme* consist of delicate gelatinous incrustations, here and there, on leaves and stalks of the host, forming a hymenium of slightly pink, clavate basidia with 2 to 4 stout, curved sterigmata, bearing pip-shaped (one-side depressed), hyaline, apiculate basidiospores which measure from 11 to 12·5 by 5 to 6 μ [17].

The fungus exists in the turf in a more or less dormant condition ready to resume growth and to fructify when weather conditions are favourable. During periods of warm weather accompanied by heavy dews at night abundant mycelium is formed, but the fungus can also tolerate temperatures at, or just above freezing point, growth increasing, however, with rising temperature to a mean of about 70° F. and ceasing at about 85° F., so that, while the fungus is checked by summer heat and winter cold it survives practically throughout the whole year [5, 12]. The hardened 'threads' or 'spicules' are highly resistant to desiccation and were found to be still viable after being kept dry for over two years [11].

The disease is reported to be frequent and severe in the southern parts of England on chalky or sandy soils, but elsewhere in Britain it seems to occur on all types of soils [4].

Corticium disease does not appear to be influenced by any manurial treatment on the lines indicated above in connection with snow mould. Vigorous growth of the grass should be encouraged and the same spraying treatment with Bordeaux mixture and malachite green, as mentioned above, should be carried out before the disease appears [5]. The fine-leaved sheep's fescue appears to be more resistant to this disease than the creeping red fescue [11].

Dollar Spot, *Sclerotinia homoeocarpa* F. T. Bennett

This disease of turf is also fairly common throughout Britain, especially during periods of mild weather in the autumn. Circular brown spots about 2 inches in diameter may develop separately or fuse together to form irregular patches which eventually turn white. The fungus *Sclerotinia homoeocarpa* causing 'dollar spot' exists in a number of strains only, some of which produce the characteristic apothecia, which, however, are rarely found in nature. The mycelium consists of reddish-brown masses and the sclerotia are of variable size and texture. The apothecial hymenium consists of numerous asci containing hyaline, oblong-elliptical ascospores which measure from 16 to 17 by 5·2 to 6·5 μ [5].

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Chapter XII

DISEASES OF POTATOES AND ROOT CROPS

Black Leg of Potato, *Bacterium phytophthorum* (Appel) Burgwitz

THIS bacterial disease causes a soft decay of the tubers and lower parts of the stem of the potato. It has been variously called ' black leg ' [8], ' basal stem rot ' [24], and ' black stalk rot ' [22]. Though not usually serious or epidemic in the field, it may cause considerable losses in storage clamps. It occurs in most parts of Europe, the United States and Canada. In Britain, it is reported to be more troublesome in the northern than in the southern parts of the country [1].

The disease usually breaks out at random in the crop, at any stage in the germination of the seed tuber, and plants may become affected up to a late period during the growing season. Affected plants may be detected by an unnatural stiff, stunted appearance of the haulms which are not so pliant as the normal stems, and the branches tend to grow upright instead of spreading out in the usual manner. Moreover, the topmost leaves often shine with a bright metallic lustre and have the margins curling inwards [17]. The plants soon turn a sickly, pale-green or yellow colour, wilt and die. At and below soil-level affected haulms are jet black in colour, but all the shoots of a plant are not necessarily diseased, though they often are, and in many cases apparently healthy stems and infected ones may grow from the same tuber. If the soil around the blackened shoots is cleared away, the black discoloration is seen to be continuous right down to the old seed tuber. Sometimes the disease may progress so rapidly that the young sprouts on the tuber are destroyed before they emerge out of the soil, or the seed tuber may not germinate at all, so that gaps are left in the drills. Usually, however, black-leg disease begins to appear when the plants are about 6 to 8 inches high, but other plants may be almost full size before symptoms are visible. In the former case, when the shoots are attacked at such an early age, the plants may perish before any new tubers begin to develop, and when they are attacked later, any new tubers that may have formed usually contract the disease through the ' heel ' end where they are attached to the stolons. The heel end is, therefore, the first part of the new tuber to contract the disease and a brown discoloration or sometimes a grey metallic lustre of the skin may early be seen over this part of the affected tuber.

In a wet season, black-leg disease is rapidly progressive and entire stools of tubers may rot in the soil, but this does not usually take place unless the soil is continuously wet, and only under water-logged conditions and heavy contamination with infection does the trouble travel from tuber to tuber in the soil. The disease does not, therefore, become epidemic, and in comparatively light, dry soils, makes little headway. The greater ravages of black leg take place in storage, especially in wet, badly ventilated pits or clamps. Tubers to all appearances



FIG. 240.—Black leg of potato (*Bacterium phytophthorum*). The disease in the tuber (photo by Foister & Noble)

sound when harvested may, especially if infected late in the season, harbour the disease at the heel end and, if such potatoes are placed in damp storage, decay sets in and the tubers become converted into a soft, watery mass from which bacteria ooze out to contaminate and infect sound tubers. Infection may take place either through wounds or abrasions in the skin, or fungal lesions, and perhaps through injuries caused by insects, or through lenticels.

It is by no means certain how the disease is carried over from one season to the next. Seed tubers, unsuspected of harbouring the bacteria at time of planting, are believed to be one way of starting infection in the crop, for there is evidence that the causal bacteria are capable of surviving throughout the winter protected under the skin of the tuber. Apparently healthy tubers have been found to produce as much as 10 per cent. or more infection, while tubers from diseased plants when planted in clean soil seldom produced the trouble in the following season [18]. But there is increasing evidence that the organism is able to live in the soil over winter, being highly resistant to extremes of temperature and desiccation [13, 15, 16]. Others state, however, that direct infection from contaminated soil occurs only in very heavily infested water-logged soils, the organisms being conveyed by drainage water from plant to plant. Ordinarily, however, the disease does not extend throughout the crop in this way [19, 20].

The bacterial organism causing black-leg disease of potato is *Bacterium phytophthorum* [2], but around its identity much controversy has, from time to time arisen [6, 7, 8, 9, 11, 19, 21, 22, 24, 25]. Numerous other bacteria are apparently also capable of causing soft rot of potato without, however, producing the identical symptoms of black leg set up by this particular organism. It is a small, rod-shaped germ measuring from 1·3 to 1·8 by 0·9 μ, and is motile by peritrichic flagella, non-sporing, Gram-negative, aerobic and facultative anaerobic, gelatine liquefying and nitrate reducing; it produces round, greyish colonies on agar.

Infection of healthy tubers in contaminated soil may take place, as already stated, through wounds. During the germination of a seed tuber already infected, the bacteria, living mostly in the intercellular spaces of the superficial tissues of the tuber, multiply and pass into one or more of the growing sprouts, and so find their way into the stems, where they collect for the most part in the intercellular spaces of the cortex. The enzymes liberated by the bacteria into the host tissues travel up the stem considerably in advance of the organisms themselves, and the effect of the secretions, especially on the vascular bundles, is to cause a brown discoloration in the walls of the lignified elements; this effect may be seen when the stem is cut across, the staining being more or less confined to the vascular cylinder. Beside their solvent action on the middle lamella, particularly of the cells of the

pith and cortex, causing a disintegration of these tissues and setting up a soft rot, the bacterial by-products are also responsible for causing a brown discoloration and morbid condition in the leaves, causing them to wilt and die [10]. There is little doubt that the infection is systemic, for the browning can be traced right up to the growing apex of the shoot, and the discoloration extends all the way down the stem, forming longitudinal streaks corresponding in number and position to the location of the vascular bundles within. With greater exposure to the air, as by cutting open an affected stem or tuber, the infected tissues turn from brown to black, owing probably to the oxidation of the bacterial secretions (Fig. 240). A peculiar feature of diseased stems, which renders them more rigid than the stems of healthy haulms, is the greater development of fibrous tissue in the vicinity of the vascular cylinder and the increased amount of sclerotic cells in the cortex. There is also a greater deposit of protein crystals in the foliage leaves than in those of the healthy plant [3].

The organism attacks the host over a wide range of temperature, the optimum being about 26° C., but it can also withstand low temperatures, and, as stated above, is known to survive in the soil during an entire winter and to resist desiccation in artificially dried soil for eight months [13]. The fact that the parasite is capable of thriving in water-logged soil shows that it can adapt itself to anaerobic conditions, but these same conditions react unfavourably on the tubers, for in the absence of free oxygen, wounded tubers are unable to form cork to protect their wounds from infection, and this is one of the reasons why cut tubers planted in wet soil fall such easy prey to the disease [13, 14, 15].

For the control of black leg disease, no seed should be saved from plants, however lightly affected, for the least amount of infection in a tuber may be sufficient to start the trouble at planting. Tubers for seed should preferably not be cut unless duly exposed to the air so as to hasten suberisation. As a precaution against the conveyance of the bacteria in soil adherent to the tubers, it is recommended to steep the tubers for 1½ hours in a solution of mercuric chloride (2 oz. per 25 gallons of water), or in formalin (1 pint in 30 gallons of water) for 2 hours. It must be borne in mind that good storage begins at digging time, and all reasonable care should be taken to avoid injuring the tubers in lifting or handling [15].

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Common Scab of Potato, *Actinomyces scabies* (Thaxt.) Güssow

Common scab attacks the tubers of all kinds of potatoes, late as well as early varieties. Although the effect is largely superficial, tubers are rendered unsightly and unacceptable for ' seed '. There is often considerable reduction in the yield, and the keeping qualities of the potato are impaired in storage.

The disease is widely distributed, and has been extensively studied in Britain [37-40], Europe, and America [30-33 c]. In Britain, six, more or less distinctive, types of the scab are recognised : *superficial*, a mere russeting or abrading of the skin; *ordinary scab*, the commonest form, showing concentric, wrinkled layers of the skin around a central depression ; *pitted scab*, deep depressions bordered by torn skin,

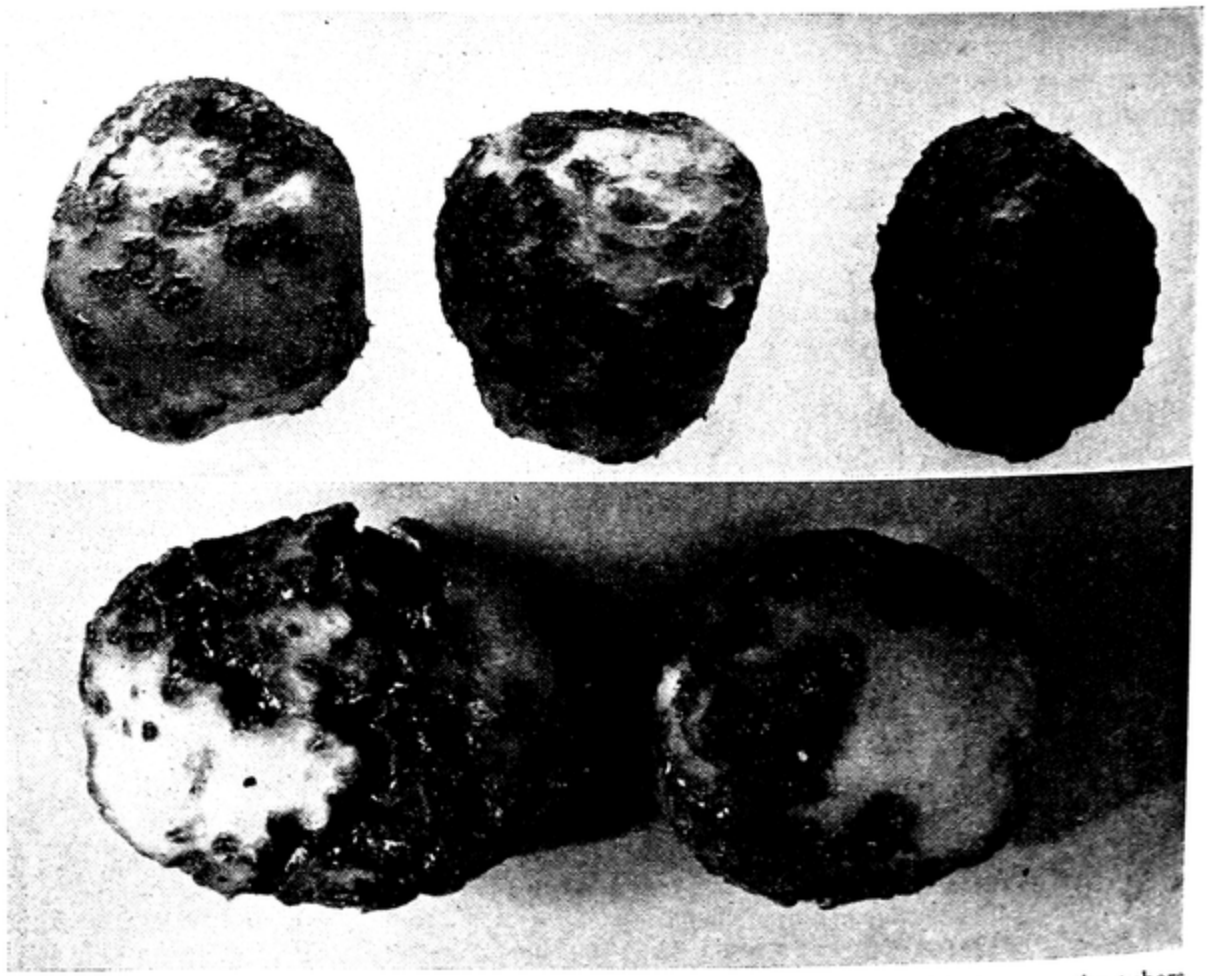


FIG. 241.—Common scab of potato (*Actinomyces scabies*). Various types of lesions on the tubers (photos of top three tubers by McKay ; two tubers below by Millard)

a severe and unsightly form, frequently infested with bacteria and various soil microfauna [13, 21], and in which the fissures or craters may become joined together during development; *stud scab*, round swellings, with vertical sides; *tumulus scab*, like the stud kind, but with sloping sides; and *pimple scab*, of small, pimple-like growths (Fig. 241). In addition to any of these features, small nodules may be found on the roots and stolons, and browned areas may also occur on rootlets [26]. In the United States, only three types of scab are usually observed, *common*, *deep*, and *russet*, and as two or all three of these may be found on the same tuber, the differences are believed to be due to variable degrees of pathogenicity of the organism rather than to the mode of development of the lesion [1]. According to some observers, a particular type of scab appears to depend on the place of origin and the variety of 'seed', features which are believed to modify the natural resistance of the tuber to the scab [41, 53]. It is further stated that different varieties of potatoes may be attacked by different forms or races of Actinomyces, and not that each form or race of the organism can cause a particular type of scab irrespective of the potato variety [48, 49]. A certain degree of soil reaction is also said to account for a particular type of scab, thus, below *p*H 5·4, Green Mountain and Katahdin tubers showed exclusively the superficial type of scab [48].

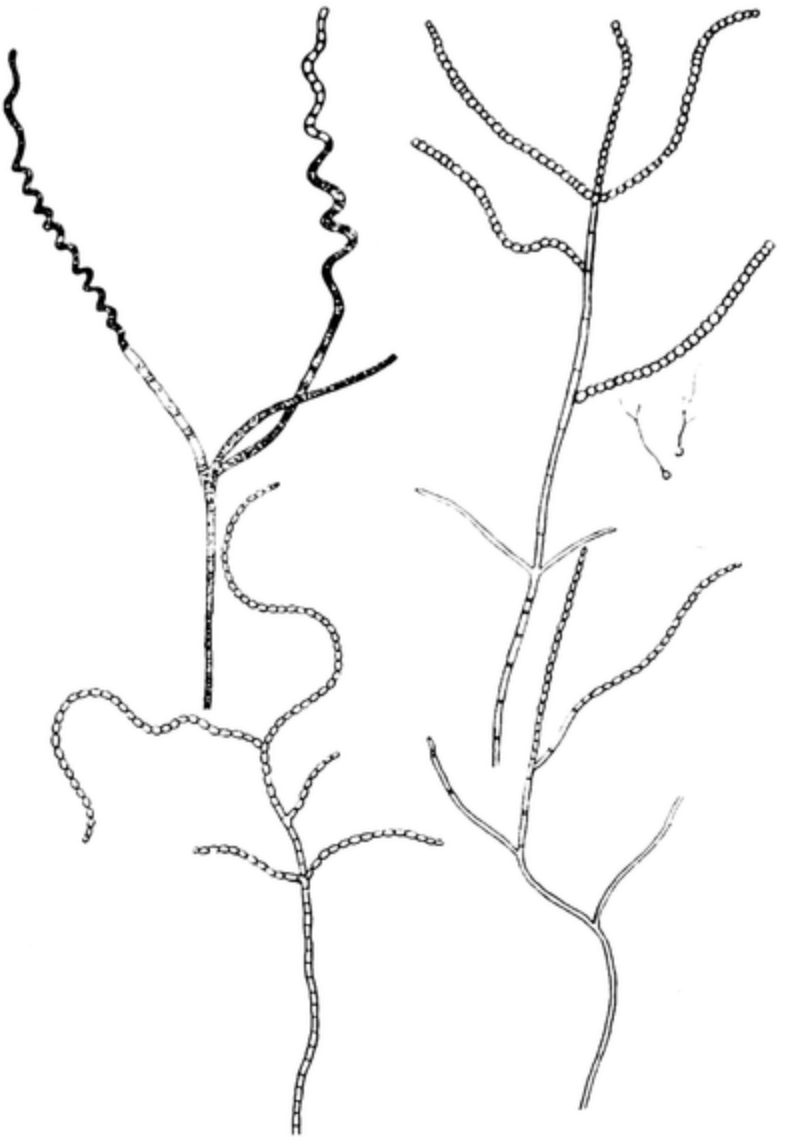


FIG. 242.—Different species of *Actinomyces*; note the sporing terminal portions (after Millard, *Ann. App. Biol.*)

Common scab is caused by *Actinomyces scabies*, a member of the Actinomycetes, an ill-defined group showing characters common both to bacteria and fungi. Scraped off from the surface of a fresh lesion the organisms appear as a white or greyish film which soon dries on exposure to air [2]. It consists of a mass of simple or branched filaments which are non-motile, very narrow, from 0·5 to 1 μ in diameter, and septated at irregular intervals; little is known about the nature of the cell contents (Fig. 242). Whether

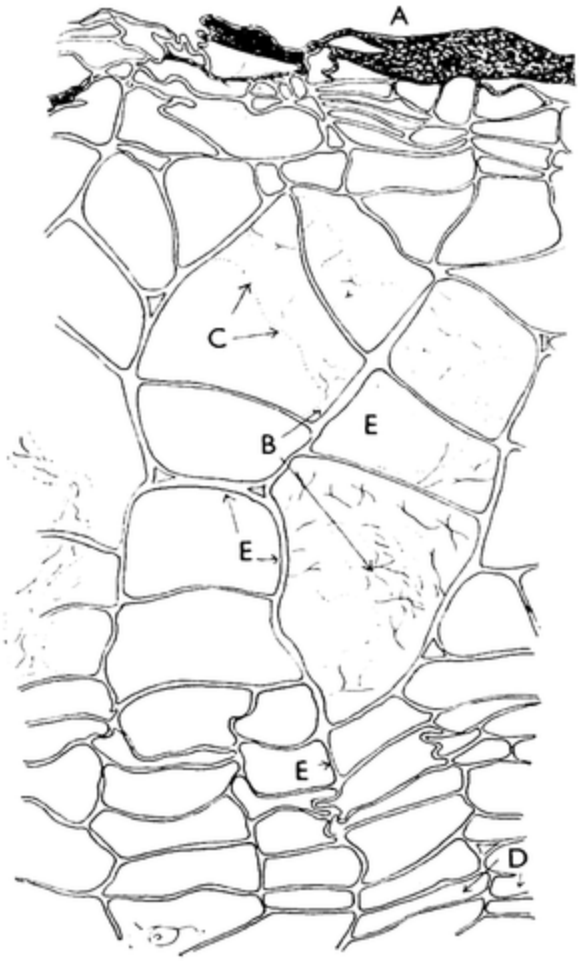


FIG. 243.—*Actinomyces scabies*. Cross-section of a portion of the wound cork lining the margin of a moderate-sized pitted scab. *A*, collapsed cells. *B*, *Actinomyces* threads in basal wall, *C*, of a cell. *C*, basal wall. *D*, wound cork meristem. *E*, thickened cell walls (×720) (after Powell Jones, *Ann. App. Biol.*)

simple or branched, there is a characteristic spiral twist at one end of a filament or branch, and sporulation is confined to this region. The spores, abstricted in succession at the apex, look more like segments of the filaments than organised spores [33], but at other times the segments cut off resemble conidia or resting spores, and probably these differences are to be attributed to different strains of the organism; no endospores are formed. The organisms are not easily stainable [24, 52]. In culture the colonies are usually very small, white or creamy, but very variable in colour, irregularly star-shaped, or filmy-diffused; on a dextrose-nitrate medium good growth was obtained from 8° to 38° C., the optimum being about 22° C. [46]. The organism is strongly aerobic. The degree of relative humidity for best growth on nutrient agar is about 33 per cent. saturation, and a limiting acid reaction for spore germination lies near *p*H 5·3, and a *p*H 8·5 is favourable for rapid germination of spores and general development. The spores are resistant to freezing for long periods without injury. All Actinomycetes are tolerant of desiccation, as indicated by their abundance on dry straw, hay, and arid soil.

The parasite, whether in the form of filaments or spores, attacks young tubers at very early stages of development, and infection continues as long as the tubers are growing [18]. Indeed, the parasite appears to have a preference for plants with a high degree of vitality [20a]. Penetration is by way of stomata or young lenticels and therefore occurs at the apical, growing end of the tuber, but at other areas too if previously wounded [10, 26, 33a]. Early signs of scab appear as small round spots, hardly more than 1 mm. in diameter and very difficult to distinguish from the lenticels, except that the lesions turn brown as they enlarge. The presence of the organisms in a young lenticel appears to stimulate the cells of the lenticel-meristem to more rapid division than occurs below an uninfected lenticel, so that most of the cells in the upper half of the lenticel become elongated in a radial direction, from the tangential meristem. These radially elongated cells are, unlike the normal round and loose cells of the uninfected lenticel, packed close together without intercellular spaces and, it is important to note, they are not suberised. All, or most of them, soon become occupied by the filaments of the parasite (Figs. 243, 244), and the infected area increases in size by virtue of tangential and radial

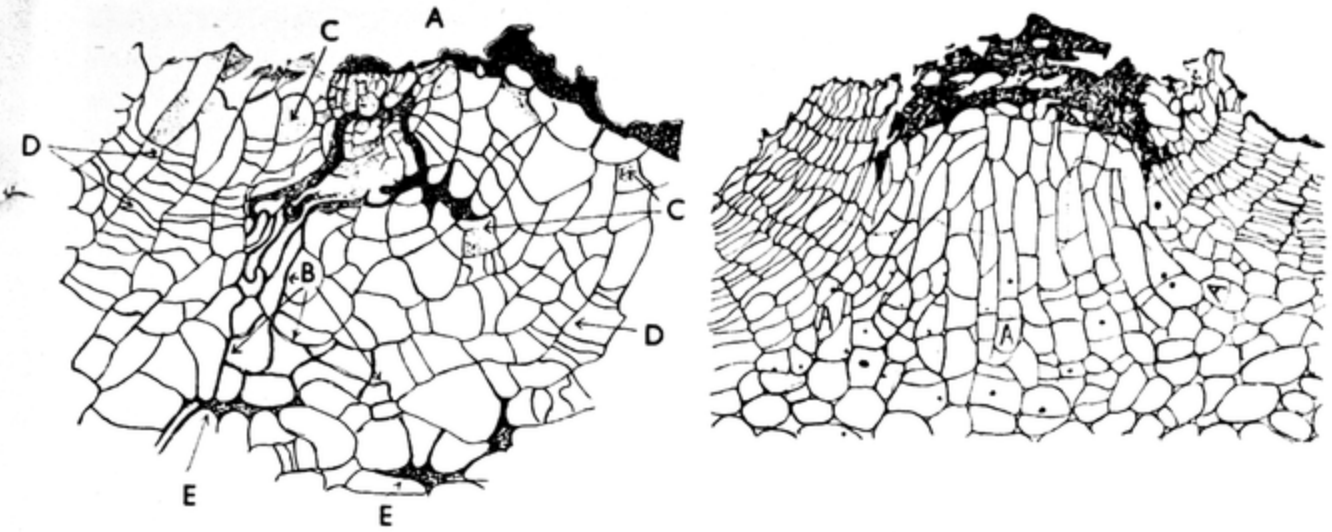


FIG. 244.—*Actinomyces scabies*. Left, cross-section of a portion of a moderate-sized pitted scab showing the route by which the organism can penetrate beneath a suberised barrier. *A*, dark-stained collapsed tissue. *B*, brown-stained walls along which the fungus probably travels. *C*, shaded cells containing *Actinomyces* threads. *D*, cells with suberised walls. *E*, internal infected areas (× 333). Right, cross-section of a very young scab (pitted type) (× 66). Note at *A-A-A*, lenticel meristem. All cells external to the dotted line contain the fungal threads (after Powell Jones, *Ann. App. Biol.*)

divisions of the lenticel-meristem so that the number of elongated cells is increased. (Fig. 244, right). As more and more of the latter are formed, they become infected and, with the collapse of the older infected cells, the lesion at the lenticel is considerably widened. The development of the abnormally elongated cells appears to be a reaction of the host to infection, being in fact an attempt at the formation of a cork barrier to delimit the lesion, but it is apparent that the development of wound cork in association with scab disease does not follow the same sequence of events as in ordinary wound healing in the potato tuber [26]. Presumably, owing to the presence of the parasite, or to the infiltration of its by-products [18] in interfering with, or otherwise delaying normal suberisation of the cork layers (Fig. 244, left), penetration of the imperfectly suberised cells is allowed to proceed and, consequent upon deeper infection, further meristems arise, one below the other, in repeated attempts at the erection of a cork barrier to check invasion. Eventually, however, as the tuber gets older, when possibly the influence of a principle which appears to inhibit suberisation becomes neutralised, complete suberisation of the last cork layers is established, and further inroads are checked. Progress of infection is, however, never very deep, except in some varieties [33a], and while a single barrier is often sufficient to check most types of scab, it is unusual to find more than three attempts at the formation of periderm before the innermost barrier is completely suberised and the lesion finally delimited. Owing to the development of these successive barriers, more and more infected tissue at the surface of the scab is thrown off, and if the tuber is still in active growth, no doubt further barriers would develop one below the other in the extending scab [26]. There is no evidence that the degree of suberisation of the lenticel meristem, in any particular variety of tuber, bears any relation to varietal resistance to the disease [29].

Recent investigations in Vermont seem to indicate that the scab parasite may be more widely distributed throughout the tissues of the host than hitherto suspected. Parasitism appears to amount to systemic infection, the organism being found in the roots, aerial stems, leaves, and flowers [33b].

Several factors are concerned in the incidence of common scab. They are chiefly soil texture, moisture capacity and temperature of the soil, hydrogen-ion concentration, nature of fertilisers, and the antagonism of other micro-organisms of the soil towards the parasite. Dry, gravelly soils with an alkaline reaction are almost invariably favourable to the development of scab, while an acid reaction greatly reduces the amount of disease [35]. Strong aerobism of the parasite demands a freely aerated soil, with a certain degree of moisture [23]; the disease is inhibited in soils having high moisture-content, and develops more actively on the return of drier conditions [19, 45]; in clay soils the disease is almost entirely absent during a wet season [11, 33, 42]. Recent experiments in America have shown, however, that in the absence of effective antagonism from associated saprophytes, scab may be severe in soils of high moisture content as well as in drier soils [47a]. Common scab is more prevalent in regions where soil temperatures are comparatively high during the growing season, and is reduced as the soil becomes cooler. Experiments at Wisconsin, using the same strain of potato, showed the amount of disease to increase with rise of temperature, the percentage of scabbed tubers being 6·25, 13·23, and 30·55, at temperatures of 19°, 21°, and 25° C. respectively [27]. Owing to alkalinity of the soil being favourable to the disease, fertilisers and other materials which tend to produce an alkaline condition should be used with discretion on potato soils. The action of lime or chalk in tending to neutralise acidity would appear to encourage the disease. On neutral soils lime has been observed to have little or no effect on the development of scab, but on distinctly acid soils it tends to aggravate the trouble unless the soil contains a large reserve of vegetable organic matter (Fig. 245). When soils for potatoes require liming for correction, the process should be done at another stage in the rotation; after three or four years potatoes may again be grown, since the decomposed lime will contribute no further risk to the promotion of the disease [42]. Emphasis has recently been made on the importance of the ratio of calcium to potassium in relation to the incidence of scab. The addition of calcium has been found to increase the flow of potassium to the shoots, and freedom from scab, with higher increment of yield, was obtained when the two fertilisers were applied in approximately equal quantities, and it is suggested that the long observed efficacy of an acid condition of the soil is due to the movement of certain cationic nutrients in the plant [15]. Alkaline fertilisers such as nitrate of soda, calcium cyanamide, basic slag, etc., are not recommended, as they are liable to induce scab, while sulphate of ammonia and superphosphate hinder its development [3, 8, 17, 20, 25, 42].

The Actinomycetes are eminently saprophytic until their natural food in the soil becomes exhausted, and there is little doubt that there are several species of Actinomyces which, under stress of hunger, can become 'educated' to a parasitic life in the soil, if a suitable host such as the potato is available to them [31, 32, 33, 48]. In Britain [37-40] and elsewhere, researches on the control of potato scab, based on

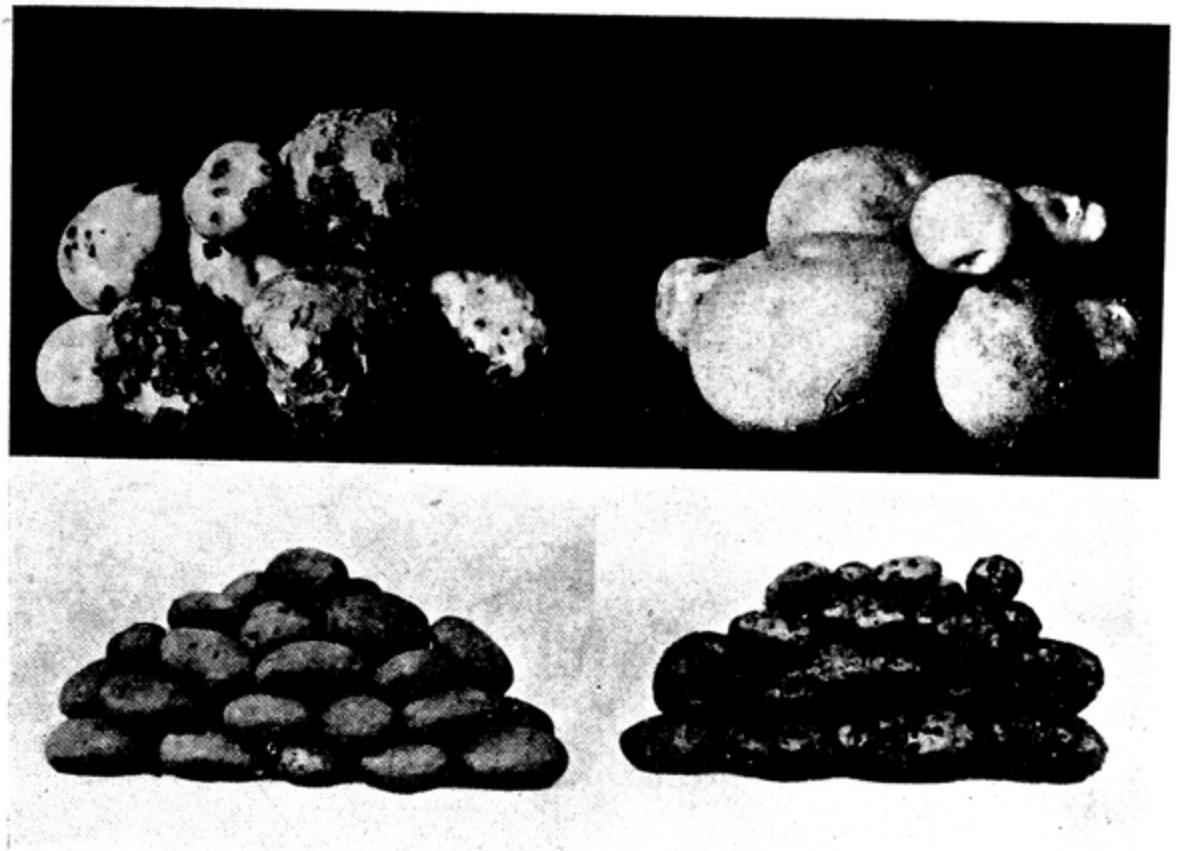


FIG. 245.—Common scab of potato. Top, effect of injudicious liming; left, heavily treated; right, untreated. Below, control of scab by green manuring; left, green manured; right, untreated (photos by Millard, *Ann. App. Biol.*)

a 'preferential food' hypothesis, show that a very appreciable amount of control can be obtained by green manuring (Fig. 245), a process which consists in the ploughing in of a green crop, such as rye, mustard, vetches, etc., or of the application of green material like grass cuttings, spent hops, etc., to the furrows before planting; in the presence of green manure it is maintained that saprophytic species of Actinomyces become dominant over the parasitic forms, and under such circumstances the potato crop may be lifted entirely free from scab. In the control of the disease by crop rotation a period of seven years is not considered too long for interposing fallow, oats, swedes, summer barley, and oats [3, 7, 44]; the inclusion of clover is not advisable owing to the necessity of lime as a top dressing, the resulting alkalinity being favourable to the reappearance of the disease [4, 5, 43]. Since it has been established that *A. scabies* can exist on decayed remains of the potato other than the tubers, all such remains should be removed and destroyed or used to manure other crops [33 c].

As the organism of scab is a soil dweller, control of the disease by 'seed' treatment appears to give no general satisfaction. While some have found no benefit from any of the standard seed-tuber treatments [47], others state that discrepancies which have been observed in the effects of mercury compounds are to be attributed not only to the wide variation in pathogenicity between different races of Actinomyces, but to variable degrees of tolerance of these races towards mercury compounds [28]. However, good results have followed the use of yellow

oxide of mercury and calomel, the yield showing an increase when these substances were mixed into the fertiliser [1a, 2, 36]. The use of sulphur has not proved to be uniformly effective in the control of scab [16]. Dipping the seed tubers for five to ten minutes in acidulated mercuric chloride, or in hot or cold formaldehyde [22, 34, 50], or an ' instantaneous dip ' in an organo-mercuric compound [7], have all proved beneficial.

Complete immunity from scab is not claimed for any commercial variety of potato [3, 10], but, from experiments conducted in Germany and America, the occurrence of scab-resistant seedlings and their breeding behaviour in a limited number of progeny tests lends encouragement to the belief that varieties of potatoes possessing high resistance to common scab may soon be available [10, 14, 17, 53]. In Indiana loss of yield of Katahdin, Irish Cobbler, and Sebago varieties was greatly reduced by planting as early as possible in the season [44a].

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Powdery Scab of Potato, *Spongospora subterranea* (Wallr.) Lagerh.

This disease of the potato attacks all parts of the plant underground, the stem, stolons, tubers, and roots, but has little adverse effect on any except the tubers, on which unsightly brown blisters, emitting dry masses of powdery spores, ultimately appear. It does not occur on any other crop, but the roots of tomatoes and of a few wild plants of the same family, yield to artificial infection with the organism causing this disease.

Powdery scab of the potato was first described in 1841 in Germany [22]. It appeared in England some five years later [7], but was not reported in America until 1913, when it was recorded in Canada and Maine [6], and although it has been seen in various isolated regions, powdery scab is not widely distributed in the United States. There are various reports of its occurrence in South America, Australia, New Zealand, Tasmania, Kenya, and Cyprus.

The disease first appears during the development of the tubers, near the growing, apical end, in the form of small, circular, light-brown spots, usually less than $\frac{1}{4}$ mm. in diameter, and hardly distinguishable from lenticels, except for a circular translucent halo, 1 to 2 mm. wide, surrounding the infected spot [14]. The spots may arise close together, but with the gradual expansion of the tuber by growth, older lesions become spaced out over the greater part of the tuber (Fig. 246). The spots soon become raised to form small pimples or blisters which, as long as the skin remains unbroken, are quite smooth. When the skin breaks it forms a frill around the margin so that each blister becomes a shallow cavity filled with a dry, powdery mass of spores which are dispersed into the air at the least touch. This primary stage of the disease forms but small cavities on the surface, and there is very little destruction of the fleshy part of the tuber. The size of the pustules appears to vary with the humidity and aeration of the soil [13]. Sometimes only the primary stage is present [12]. If the soil is continuously wet during the growing season, a more extensive and severe secondary stage of the disease develops from the same lesions, and much deformity of tuber growth takes place. This secondary phase may be described as one of canker formation, and extensive areas of the tuber may be covered with corroded patches bearing little resemblance to the

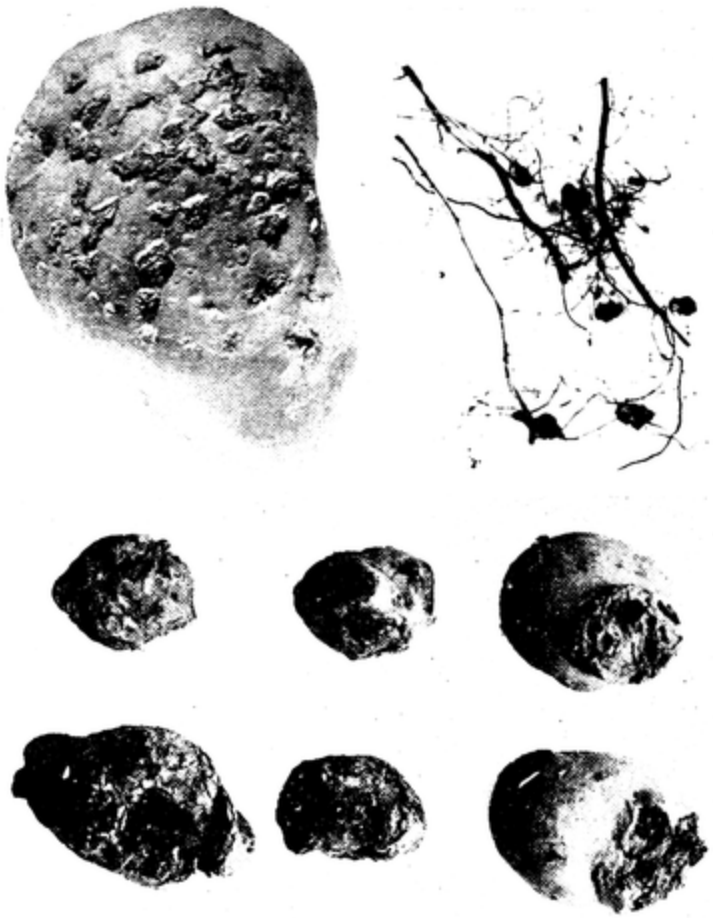


FIG. 246.—Corky, or powdery scab of potato (*Spongospora subterranea*). The large tuber shows the characteristic pustules on the tuber (photo by Foister & Noble). Below, six tubers showing the severe ' canker ' form (photo by McKay). Top, infective nodules on the roots

original blisters of the primary stage (Fig. 246). Under drier conditions of the soil, however, or after the tubers are lifted, even the severe canker phase may be checked, but if the soil remains wet towards the autumn, or if the potatoes are stored under too moist conditions, still a third form of the disease may develop in the shape of warty outgrowths bearing a striking resemblance to the tumours of the true ' wart disease ' of potatoes (*Synchytrium endobioticum*) described below. But unlike the latter, the tumours of powdery scab have a smooth contour, not the rough, rugose surface of the true warts, and they eventually collapse, leaving behind raised brown or chocolate coloured scars [7], resembling those of the canker stage. In the case of early varieties of potatoes, lightly attacked, it is often very difficult to detect any pustules of disease, because they may not break out until the tubers are in storage.

The disease on the roots causes the formation of small warty growths, not unlike the bacterial nodules on the roots of leguminous plants (Fig. 246). These nodular growths do not seem to interfere much with the normal functions of the roots, but as they are still liable to form the spores of the organism (Fig. 247 C), they undoubtedly increase the amount of infection, when they finally decay to discharge their contents into the soil. The occurrence of the scab on other underground parts of the stem and stolons is comparatively rare.

Powdery scab of potato is caused by *Spongospora subterranea*, a member of the slime fungi, Plasmodiophorales [5, 11, 15, 22]. Unlike *Plasmodiophora brassicae*, another well-known member of this group, causing ' club root ' of crucifers, the spores of which are dispersed into the soil singly when the host decays, those of *S. subterranea* are liberated in coherent masses or ' spore balls '. The latter are more or less spherical in shape and measure about 50 μ in diameter. The individual spores are very small and remain adherent by their cell walls, but with gaps here and there so that a spore ball has the appearance of a tiny sponge (Fig. 247). When a diseased tuber decays in the soil the

spore balls are liberated whole but may sometimes become disintegrated into individual spores.

In germination, the contents of each individual spore are released through a small aperture in the wall, as a minute, hyaline, uninucleate body called a myxamoeba [18]. The further history of the myxamoebae in relation to penetration and infection of the tuber is by no means clear. Penetrations into the root hairs of both potato and tomato seedlings by myxamoebae were observed soon after planting in soil inoculated with the spore balls of the organism; after a fortnight's incubation at 65° F., there were found within the root hairs not only myxamoebae, but also one or more bodies which had apparently developed from myxamoebae by budding. These bodies turned out to be sporangia, for they were found to contain a number of zoopores or swarm spores. The zoospores did not penetrate farther into the host but were discharged from the root hairs into the soil through tiny holes in the root hair wall at points in contact with the wall of the zoosporangium [16]. The formation and liberation of these zoospores, so early after infection, probably constitutes a gametophytic phase in the life-history of the organism, the zoospores being, therefore, gametes, or facultative gametes (cf. the 'prosorus' stage in the life-history of *Synchytrium endobioticum* causing 'wart disease' of potato). But uninucleate myxamoebae have also been observed in sections of diseased tissue and in the dividing cells in the lesions, on both sides of the divisional membranes [8, 21 a].

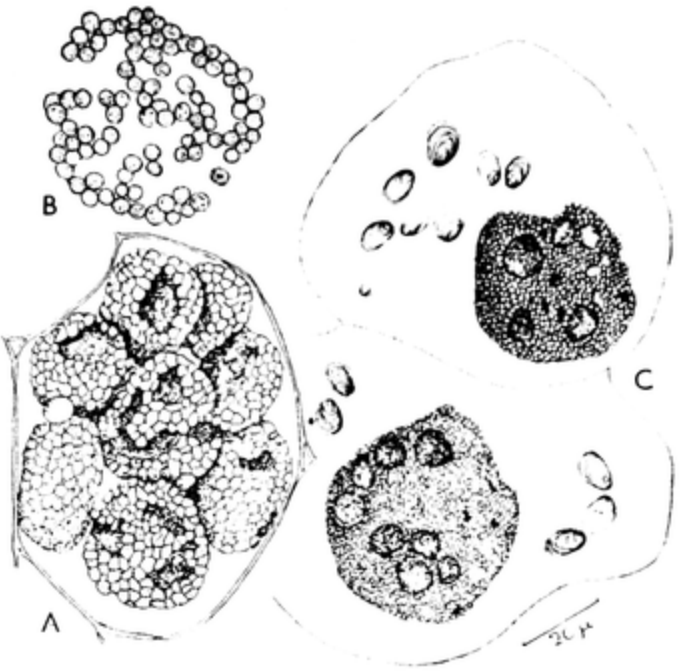


FIG. 247.—*Spongospora subterranea*. *A*, host cell with eight mature spore-balls and a starch grain. *B*, section through a mature spore-ball showing the rounded spores and the cleavages between them which give the spongy appearance when seen in surface view (after Osborn, *Ann. Bot.*). *C*, Spore-balls and starch from the nodules on the roots (see Fig. 246)

Whether infections of the tuber take place from myxamoebae or zoospores singly, or in mass, or by gametic zoospores (gametes) after pairing, is not clear. Under dry conditions in the soil myxamoebae and zoospores become encysted, and in that form are capable of resisting desiccation over a long period; and the spore balls themselves may survive in the soil over winter to germinate in the spring. The probability is that infection of young tubers takes place in the following way. In the presence of abundant moisture the spores, or encysted forms, germinate to emit myxamoebae and, consequent upon the mass discharge of these bodies into the soil, coalescence of a large number of them takes place to form one or more plasmodia, at one or more selected points of entry on the moist surface of the young tuber. That is, the first preliminary to infection appears to be the formation of one or more plasmodia external to the host. Accepting this interpretation of the mode of entry of the parasite, we may describe the action of the plasmodial parasite on the tuber in two stages: (*a*) the primary, resulting in the formation of the early 'pimple' stage, and (*b*) the secondary or 'canker' stage, in the same lesion as the primary.

Primary Stage

It is not clear how the plasmodia enter the tuber, but they have been found to occupy young lenticels and small wounds, and plasmodial masses have also been seen in the intercellular spaces close to lenticular openings (9, 10, 23). The translucent areas with tiny brown spots in the middle, indicating the early symptoms of the disease, as above mentioned, are the parts of the tuber where plasmodia have become established, and the ' halo stage ' around a lesion shows how far the plasmodium has spread beneath that area of the epidermis. If plasmodia do not enter by such natural openings as stomata or young lenticels, it is probable that, in close contact with the epidermis, a plasmodium exercises a softening action on the cellulose wall (presumably such points of entry have not yet become suberised), after which it appears to be capable of penetrating and passing between the epidermal cells possibly by solvent action on the middle lamella (14). The same effect is also claimed for the spread of the plasmodium between the host cells, immediately below the lenticels (23). The plasmodium, now within the host, spreads out more and more under the epidermis, in a lateral direction, and there is usually comparatively little inward penetration, except to the extent of a few layers of the host cells. From this situation, under the epidermis, the plasmodium sends out strands of itself, the ' infecting pseudopodia ', and, presumably by solvent action on the middle lamella, these finer branches of plasmodium push their way between the host cells, in the manner of an intercellular mycelium. Small portions of these plasmodial strands which impinge on the cell-wall may also dissolve their way through the wall, and portions of plasmodium are said to stream through tiny holes into the host cell, and thereafter the retained portion is pinched off at the point of entry. The intruding plasmodia are described as actually penetrating the host cytoplasm, as if the two masses were blending together but still retaining their independence. The plasmodium is sometimes globular or, at other times, of an irregular shape within the host cell, finely granular, and furnished with a large number of small nuclei evenly distributed throughout its substance.

Cells invaded by plasmodia are stimulated to growth, first, by becoming considerably elongated in a direction at right angles to the surface of the tuber, and if, as is usually the case, a number of invaded cells occur side by side, a portion of the epidermis becomes lifted from pressure of the radially elongating cells below. Each of the latter now proceeds to divide transversely, to form 5 or 6 cells, all of which have received a portion of plasmodium during division of the mother-cell, and the daughter plasmodia continue to grow and increase their number of nuclei by free nuclear divisions, in the same manner as the parent plasmodium. The active division of the elongated cells consequent upon infection, stimulates also the non-infected cells immediately below them to divide so as to bring about cork formation, and whether such activation is due to secretions set up by the parasite, or is merely a wound reaction to mechanical attack, is difficult to decide (23). Spread of the disease into the inner tissues of the tuber may thus be early checked by cork formation, but very frequently, before the first cork cambium has become thoroughly established, its cells are immediately invaded by further penetration of plasmodia from the cells above them, and this fresh invasion appears again to

stimulate the formation of another cork meristem below, so that it is not uncommon to find two or more attempts at cork formation beneath a lesion before the tuber finally succeeds in making a suberised cork barrier against further inroad into its tissues. The sequence of infection, so far, is strikingly similar to that described in common scab, above.

The nuclei in young plasmodia are constantly adding to their number by division, but at some stage in development there is, presumably, widespread pairing and fusion of these nuclei throughout the plasmodia, and such fusions are probably followed by reduction divisions. When the plasmodia have ceased to grow, they segment into as many portions as there are nuclei, to form a large number of spores which, as we have seen, do not separate from each other but remain together though each spore is delimited by its own cell wall. A number of spore balls may appear in the same host cell, a condition which arises mainly through the breakdown of cell walls intervening between cells containing single spore balls. With the expansion of the infected group of cells, and the lifting and rupture of the skin over the lesion, the spore balls become exposed at the surface of the tuber as yellow-brown powdery masses for dispersal [20].

Secondary Stage

In wet soil in the autumn, or under damp storage conditions, the primary lesions are followed by the much more destructive canker stage. After the discharge of the greater part of the spore balls from the primary lesions, a considerable quantity of the spores still remain, however, at the base, and lurking in particular beneath the frayed edge of the lesions. These retained spores or spore balls germinate *in situ*. From the large number of myxamoebae liberated from them on to the floor of the cavity and around its margin, numerous young plasmodia arise by fusion of these bodies, and it is likely that bigger plasmodia are formed by coalescence of small ones [14]. The cells of the tuber lining the cavity and especially those around the edge are now attacked with great vigour by the plasmodia. These cells are not only penetrated by the plasmodia, but large masses of host cells may be engulfed within a plasmodium, as if in process of ingestion, with undigested starch grains and portions of disintegrated cell wall remaining in its substance. It is clear that the canker phase is much more destructive than the primary stage, and while in the latter the host cells invaded appear to suffer no apparent injury for a considerable time, in this secondary stage of attack the host cytoplasm is killed outright by the plasmodia. Much deeper cavities are formed at the canker stage than was the case in the primary lesions, and cork barriers, imperfectly developed beneath the lesions, appear in some instances to be quite inadequate to check further inroads into the tuber. In general, however, the greater progress of the plasmodia is in a lateral direction, so that there is actually little hindrance from the presence of a cork barrier below the canker, and by the constant rupture of the skin during the lateral spread of the disease corroded areas ultimately cover extensive areas of the tubers. This severe wounding of tubers during the spread of canker inevitably results in the admission of secondary parasites and saprophytes, the most common of these being the rot-forming *Phoma tuberosa* [19], the final result being the development of a deeper and harder

kind of dry rot than the canker itself, and the tubers are often rendered worthless.

The organism of powdery scab is long persistent in the soil and a period of five years, or more, is recommended for a rotation of crops before potatoes can be restored to the same ground. It is not known whether the spore balls can exist in the soil for indefinite periods, but it appears that the encysted myxamoebae are better suited to tide over adverse and longer periods in the soil than the spores themselves [19, 21]. In the absence of the susceptible host it is probable that the plasmodia ean exist saprophytically in the soil, but nothing is known about the relations between these bodies and other micro-organisms in the soil [5].

Powdery scab of potato assumes serious proportions only under conditions of prolonged rainfall, followed by cool, damp weather during the growing season; it is of little consequence in localities enjoying a dry climate [19]. The hydrogen-ion concentration of the soil is reported not to have any marked influence on the incidence of this disease [23]. In a series of experiments, correlating the effects of temperature, soil reaction, and water content of the soil, it was found that with a moisture-holding capacity of 90 to 100 per cent., over a range of *p*H 6·3 to 6·7, the percentage infection, after an incubation of 19 to 25 days, depended mainly upon the prevalence of a comparatively low temperature; thus the amount of infection was 70 and 63·7 per cent., when daily temperatures were 15·5 and 19·5° C. respectively, and only 4 and 2·9 per cent. when the temperatures were 18·7 and 24·2° C. respectively; and during these determinations the *p*H of the soil exerted no influence [17]. But others report that acid conditions, in a water-logged soil, are the most favourable conditions for the disease [3].

For the control of powdery scab, much can be done by improving the drainage of the soil. As the organism can exist in the manure or compost heap, infected tubers should not thus be disposed of, and the application of pig manure should be avoided, as the spores can survive digestion of raw infected material fed to stock [10]. The organism can also be carried in soil and on farm implements. The application of sulphur to the soil greatly reduces the amount of disease; it may be applied at the rate of about 6 cwt. per acre, or incorporated with fertilisers [4, 19]. The seed tubers may also be dressed with the finely powdered sulphur, after moistening. Tubers slightly infected may be treated before sprouting by steeping for three hours in a formaldehyde solution of 1 pint to 30 gallons of water.

No variety of potato is yet known to be immune from powdery scab.

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Wart Disease of Potato, *Synchytrium endobioticum* (Schilb.) Perc.

This disease at one time threatened the extinction of many of the older and popular varieties of potatoes, but the extremely virulent character of the affection is now counterbalanced by the successful production of new and immune varieties. It was probably in existence in Britain long before Potter [33] published, in 1902, a short account of the disease, and indeed there is some evidence [32] that the trouble was present in England some years before Schilberszky [43] made, in 1896, what was believed to be the first discovery of the disease, in upper Hungary (Czechoslovakia). It is now widely prevalent in many foreign countries, especially in Europe, and it exists also in the United States, Canada, South Africa, and has lately been recorded in Peru, South America — the home of the potato — but it is not believed to be endemic in that country [2, 17, 25].

The potato is the only plant attacked by wart disease under natural conditions, but several other members of the *Solanaceae* may be successfully infected artificially, though they do not suffer to the same extent as the potato. The best known of these susceptible hosts are the three nightshades, woody, black, and winged; henbane, and tomato, but apparently only certain varieties of tomatoes [10, 13, 30, 44, 48].

The disease is essentially an affection of the tubers during the growing season. There are hardly any signs of disease to be seen on the green shoots of a plant affected with warted tubers and the haulms may actually be a little taller and bear bigger leaves which, moreover, are of a deeper green colour, than those of healthy plants. But sometimes, according perhaps to the variety of potato, there may be seen around the base of the haulm, at soil-level, a dirty-grey or greenish-yellow mass resembling a discoloured cauliflower and, if the potato is one of a coloured variety, the excrescence is also similarly coloured. Not infrequently some of the smaller or rudimentary leaves at the base of the haulm, at and just below soil-level, become replaced by fleshy coralloid outgrowths (the so-called 'radial galls') [23]. But the typical symptoms of wart disease occur on the tubers underground. On some or all of them the characteristic brown or brownish-black warts, like lumps of cauliflower, of variable size and shape, may be seen to grow, chiefly out of the 'eyes' of the tubers, but some badly affected specimens may be warted all over and are more like dirty lumps of cauliflower than potato tubers (Fig. 248). Not all the tubers of a stool are necessarily affected, but some tubers, to all appearances sound, may, if examined very closely at the eyes, show tiny warts, and these are often so small as to defy detection except with a powerful lens. Such apparently healthy tubers, when placed in pit or clamp, or in storage, develop the disease more and more, and pass it on to sound tubers in contact with them if the

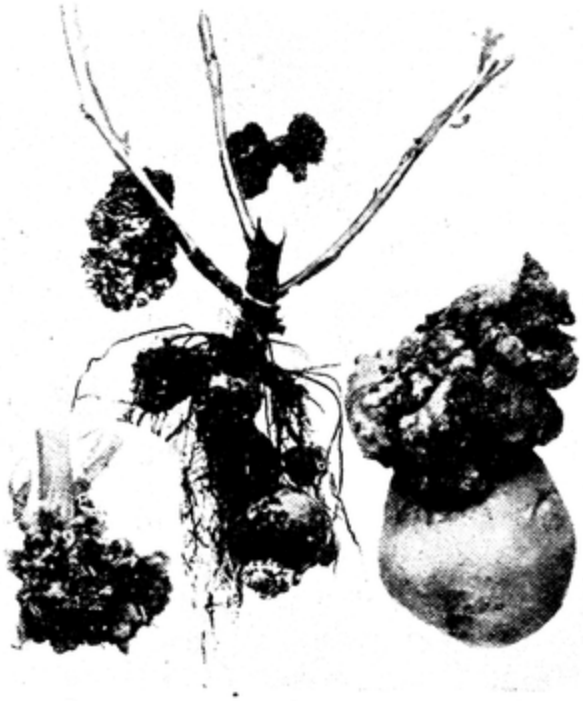


FIG. 248.—Wart disease of potato (*Synchytrium endobioticum*). *A*, a severely diseased plant; note the warts at the 'eyes' on lowermost tubers, and the conversion of other tubers into cauliflower-like lumps; the uppermost tubers are on aerial shoots ('radial') galls (photo Adv. Lft. 274, Minis. Agric.). *B*, a warted tuber at the base of a stem (photo by Foister & Noble). *C*, formation of a massive wart from apparent coalescence of smaller ones (photo by McKay)

conditions are moist. The disease also attacks the underground stolons but the roots remain free. Under dry conditions wart development is checked but the parasite remains viable in the warts for a long time and, if infected tubers are planted, the organism is returned to the soil to start fresh infections again in the spring.

Wart disease is caused by *Synchytrium endobioticum* (previously called *Chrysophlyctis endobiotica*), an organism belonging to the most primitive group of fungi, the Archimycetes. The life-history is very simple and, throughout the whole cycle, there is no development of mycelium at all. The organism starts its parasitic life as a minute, actively motile, uniciliated zoospore and, in the warted tissues, crops and crops of zoospores are formed during the summer for liberation into the soil, and these in turn bring about fresh infections on the tubers. Later, towards autumn usually, there is a resting stage to tide the organism over the winter and, when this stage revives in the spring, zoospores are again released into the soil to start infections anew.

Thus there are two stages in the life-history of the wart organism, namely, the *Prosorus* (Fig. 249 A, B, C), consisting of summer sporangia, and the *Resting Sporangia* (Fig. 249 D), constituting the over-wintering resting phase [8, 24].

(a) *The Prosorus*

During its development on the plant a susceptible tuber is open to attack at a very early stage, at any part of its surface as long as the skin has not become suberised, but as this natural process of cork formation sets in comparatively early in the history of the tuber, the parasite can only attack the more delicate tissues at the apical end, especially those of the young buds and bud-scales at the eyes [21].

To start the life-cycle (Fig. 250), we may follow the history of one of the very numerous zoospores which escape into the soil when a resting sporangium germinates in the spring. In the presence of free moisture this small, naked, uninucleate body settles down, say, within the depression around an 'eye', where after coming to rest it rounds itself off in contact with the epidermis of a bud scale, and, still naked, bores its way through, by making an exceedingly narrow channel, into an epidermal cell (Fig. 109). There is no further inward migra-

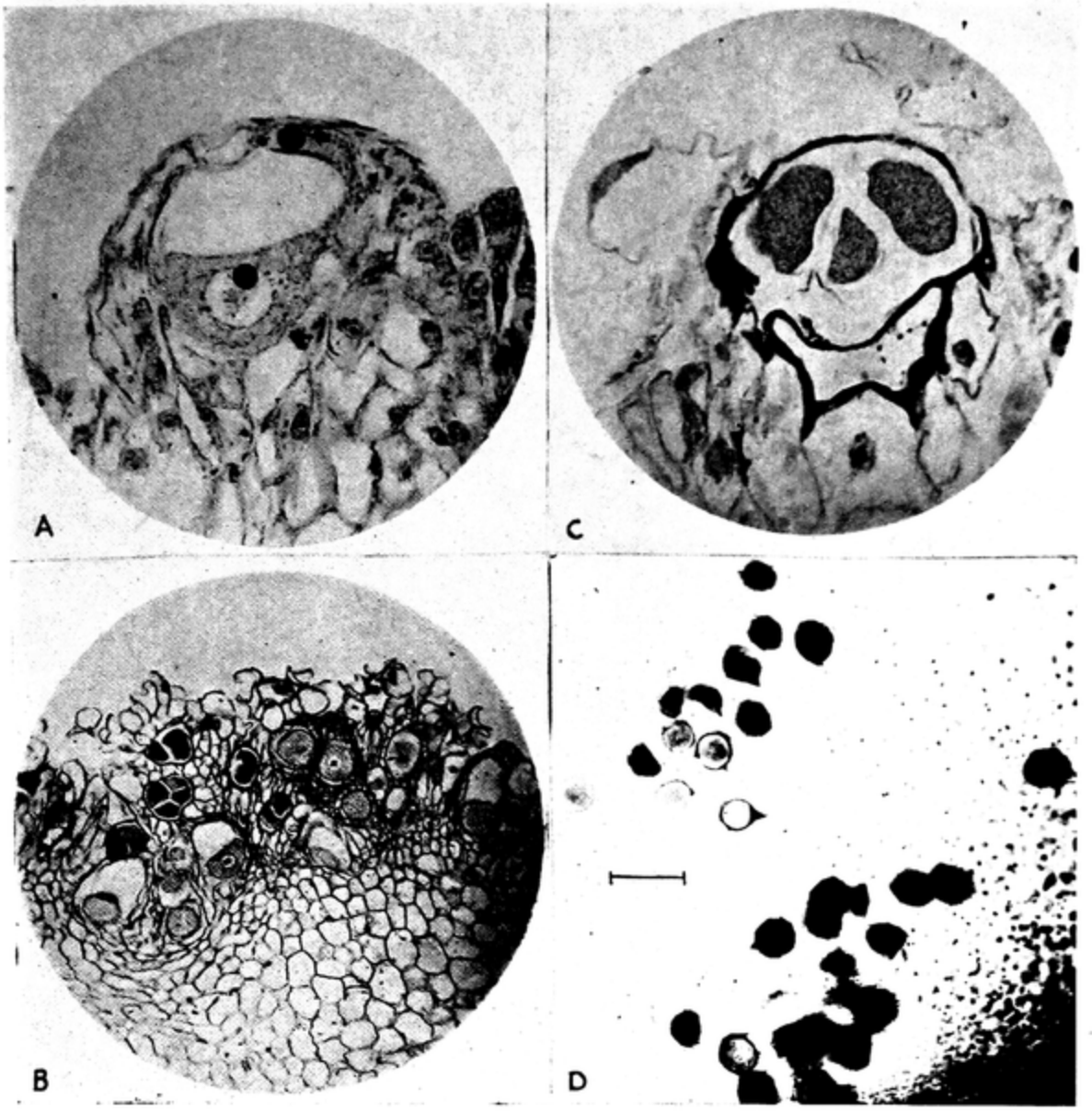


FIG. 249.—*Synchytrium endobioticum*. *A*, young prosorus. *B*, section of tuber tissue showing development of prosori, sori, and sporangia (at left, centre). *C*, a ripe sorus with three sporangia in view, empty shell of the prosorus below (× 450) (photos by Glynne, copyright of Rothamsted Exp. Station ; *Ann. App. Biol.*). *D*, section of tuber tissue showing a group of resting sporangia (inset line = 100 μ)

tion by the infecting body and the whole development is carried through in the epidermal cell originally invaded. The entry of the parasite into a particular cell, however, causes hypertrophy of those epidermal cells which are in immediate contact with that cell, so that there is formed a raised ' rosette ' of cells (Fig. 250, *11*), at the centre of which, therefore, the infected cell remains sunken as it itself takes no part in this outward growth. Meanwhile the infecting body enlarges considerably, filling almost the entire cell space, and, having developed a firm cell wall, is now called a ' prosorus '. This body, still uninucleate, soon puts forth a large vesicle which receives the contents of the prosorus, and from the repeated division of the nucleus a large number of nuclei are laid down in the

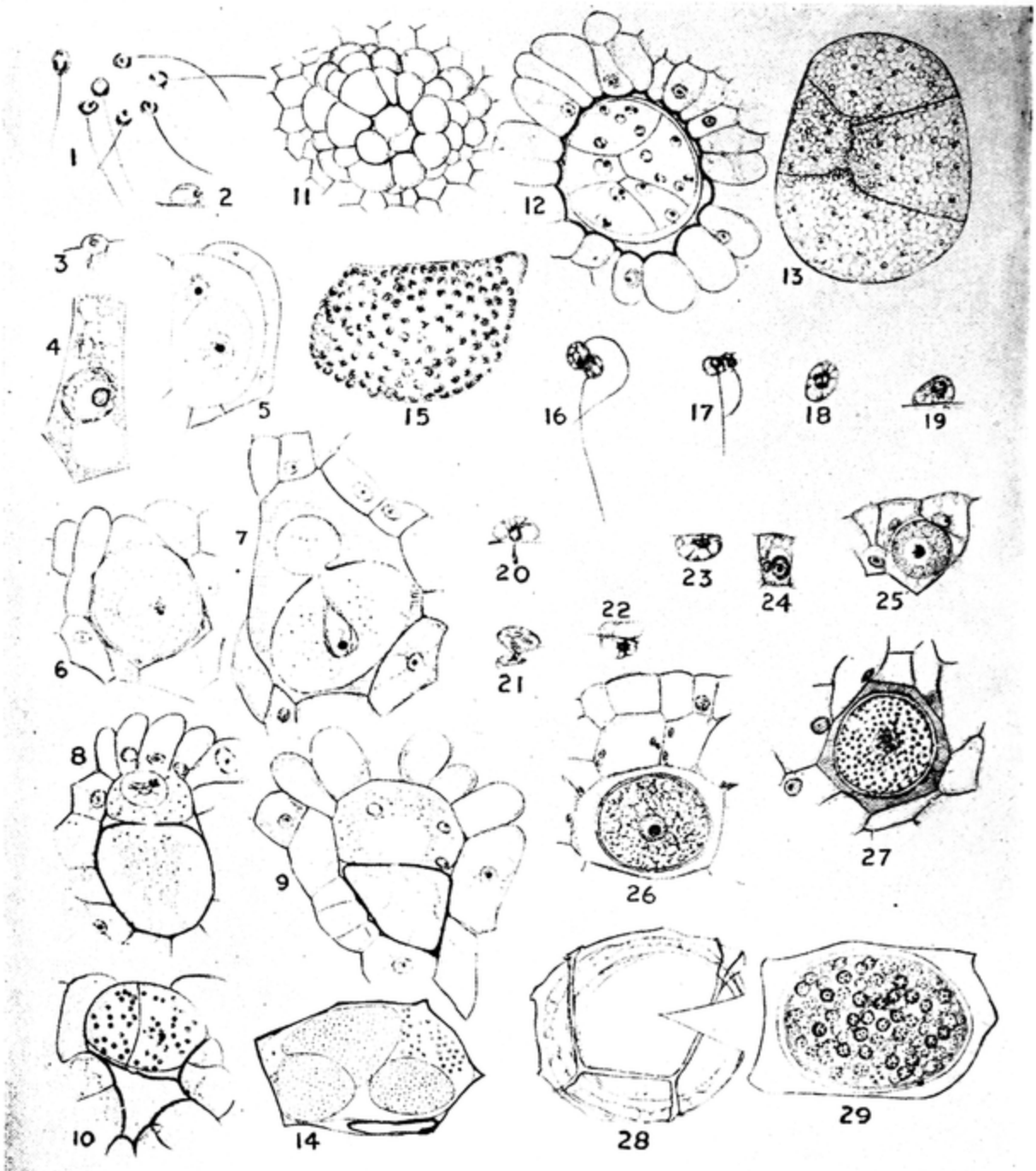


FIG. 250.—*Synchytrium endobioticum*. 1, living zoospores. 2, zoospore in contact with host cell. 3, stage of entry. 4, very-young prosorus passing downwards towards the nucleus of host cell (× 1650). 5, disorganisation of host nucleus is beginning; adjoining host cell at top is one of a 'rosette' group. 6, prosorus has developed a firm membrane. 7, passage of prosoral contents into a vesicle; the nucleus beginning to move in. 8, the nucleus reconstituted. 9, two nuclear divisions have occurred. 10, two sporangia of a sorus, and empty prosoral membrane below. 11, surface view of a rosette, with three sporangia in view. 12, a sorus showing five sporangia. 13, early stage of segmentation into sporangia. 14, three mature sporangia, and liberation of zoospores, original shell of prosorus at the base. 15, a sporangium swelling prior to discharge of zoospores, a hyaline projection formed. 16, the fusion of gametes. 17, later, membranes disappearing. 18, later stage, nuclei pressed together. 19, entry of the zygote. 20, early stage of zygote entry, the nucleus close to the surface of contact. 21, later stage in entry of nucleus and cytoplasm. 22, the nucleus now within. 23, entry of cytoplasm almost complete. 24, a very young resting sporangium which has passed down the host cell as far as

vesicle or sorus as it is now called. The sorus ultimately divides into a number of compartments or sporangia in which are finally developed a large number of uniciliate zoospores. With the breakdown of the sporangial walls and rupture of the epidermis the zoospores are liberated into the soil. Repeated crops of prosori, sporangia, and zoospores may develop from fresh infections during the summer, and these are initiated not only from zoospores released from the germinating resting sporangia, as above described, but also from the generations of zoospores formed during the summer at the prosorus stage. With such a rapid method of reproduction taking place at a time when the tubers are making active growth, fresh infections are constantly taking place, and as every infection involves the formation and proliferation of ' rosette ' cells which may themselves become infected, there is so much hypertrophy around infected cells that the tuber soon becomes more or less covered with the warted excrescences which are so characteristic of this disease.

(b) *The Resting Sporangium*

The zoospores liberated from the summer sporangia may function, as above mentioned, as asexual infecting cells, or they may *fuse in pairs*, acting as *sexual cells* or gametes. The fusing gametes produce diploid bodies or *zygotes* which again are capable of infecting the host in much the same way as the single zoospores, but the mode of development of the zygote after entry is entirely different from that of the asexual zoospore forming a prosorus [8, 28].

The arrival of the zygote within an epidermal cell (Fig. 250, *23*, *24*) is again a stimulus to cell division, but this time it is the invaded cell itself, not those surrounding it as in the instance of the prosorus, that is induced to divide. The one or two divisions which take place in it are parallel to the surface of the tuber and, since the new cells arise above the zygote, the latter appears in a section of a young lesion as if it had penetrated deeper into the host than the epidermis (Fig. 250, *26*). The zygote enlarges considerably, develops a very thick (three-layered) wall, and remains throughout as a unicellular body to form eventually the over-wintering resting sporangium. Resting sporangia vary considerably in size but, on an average, measure about 50 μ in diameter (Fig. 250, *27*, *28*). Towards the end of the season, if warted tubers are left in the ground, decay sets in and the resting sporangia are released into the soil. Although generations of prosori producing crops of zoospores or potential gametes continue to be formed throughout the season, it appears that resting sporangia do not germinate forthwith but require a rest, sometimes of a year's duration, before maturation is complete. By repeated division of its nucleus prior to germination a resting sporangium becomes possessed of several hundred free, haploid nuclei (presumably a ' reduction ' division occurs meanwhile), which finally enter into the formation of a corresponding number of zoospores developed within the sporangium itself. When germination is about to take place, the thick

the nucleus. 25, resting sporangium develops a membrane. 26, a second division of host cell places sporangium in the third layer of cells. 27, dead contents of host cell deposited as epispore upon sporangium. 28, ruptured empty sporangium. 29, surface view of a living sporangium during maturation of zoospores. (Nos. 1, 15, 28, 29, ×585; Nos. 5-10, 12, 14, 24-27, ×300; Nos. 4, 16, ×1240; No. 11, ×130; No. 13, ×600; Nos. 2, 3, 19-23, ×1500) (after Curtis, *Phil. Trans. Roy. Soc., Lond.*)

sporangial wall breaks open from pressure set up by the swelling of the inner wall, on the rupture of which the zoospores escape into the soil, and the life-cycle of the organism is complete [8, 50].

These two stages in the life-history of the organism do not follow each other in strict order of time, and the change-over from production of summer sporangia to that of resting sporangia is not due to seasonal change of temperature, because prosori may still continue to be formed into the autumn, and resting sporangia may be found along with prosori comparatively early in the season.

In the absence of a potato crop the resting sporangia may remain viable in the soil for many years, periods of 9 to 12 years being recorded in many instances, and it is even suggested that no system of crop rotation can starve these bodies out of the soil. But the organism may, perhaps, be capable of surviving in the soil in some other way than resting sporangia, and it is possible that the zoospores liberated from them may, under certain conditions, live saprophytically in the form of amoebae, and during conditions adverse to this saprophytic life the amoebae may perhaps pass into an encysted condition[8], but such bodies have not, so far, been found.

The resting sporangia may be carried from one place to another by any means that ensures the transport of contaminated soil, adherent to seed tubers or on the underground parts of any transplants such as roots of vegetables, on workers' boots, feet of birds or animals, on cultivators, cart wheels, etc. Moreover, the sporangia remain unharmed if warted tubers are eaten raw by farm stock and are still in a viable condition in compost manure heaps into which warted tubers have been thrown. They are known to be very resistant to extremes of temperature, high and low, and can withstand the temperature of boiling water for 8 to 12 minutes ; in the soil they are viable to a depth of 8 inches.

Although other plants, as above mentioned, besides the potato can be successfully inoculated with the organism of wart disease, it is not likely that any of them are responsible for spreading the disease under natural conditions ; if any such carrier hosts exist, infection in them must be latent, for no species has so far been found to show any outward signs of wart formation. It is known, however, that on rare occasions stolons of the potato plant may develop the resting sporangia without producing any obvious signs of abnormal growth [1].

Wart disease is much worse during wet seasons than in dry ; thus it used to be more prevalent in the wetter northern and southern parts than in the drier southern and eastern counties of Britain [13]. A very high degree of soil moisture is necessary to bring about infection by the winter sporangia [5, 13]. The most favourable conditions for infection are stated to be periodic flooding followed by drainage and aeration. Although good germination of the resting sporangia may take place at a relative soil humidity of 45 per cent., zoospore emission is best at higher humidities of 90 to 100 per cent. Zoospore discharge takes place over a wide range of temperature, the optimum being between 14° and 24° C. It is very active if the oxygen content of the soil is increased, and the presence of dilute solutions of nitrogenous salts is also said to favour the germination of the resting sporangia [12].

Infections from zoospores and zygotes appear to take place over somewhat different ranges of temperature. Thus, infection from zoospores emitted from the resting sporangia is favoured between 10° and 28° C., and from zoospores (potential

gametes, which form zygotes) discharged from soral sporangia the range is wider, from 0° to 30° C., the average temperature in the field for infection being about 21° [48]; and it is noteworthy that the susceptible organs of the potato plant develop well at about the same temperature range favourable to infection [12]. It does not appear that the action of frost, continuous or intermittent, or any special treatment [20], is conducive to a greater degree of germination of the resting sporangia. There is, again, no clear evidence that the physical character of the soil has any bearing on the relative amount of infection due to wart disease, light sandy soils reacting in much the same way as heavy field soils [13]. The most favourable soil-reaction is from neutral to slightly acid conditions, the range being approximately from *p*H 3·9 to 8·5 [48], and the organism appears to be sensitive to high degrees of alkalinity [34]. Three strains of the parasite are recognised in Germany [4 a].

As already indicated, wart disease has been successfully controlled by the introduction of potato varieties possessed of complete resistance to, or immunity from it, and moreover these varieties have shown no deterioration or breakdown in this quality, and they remain immune under all conditions in the field [40, 47]. It has been recorded, however, that under experimental conditions in the laboratory some immune varieties have yielded to infection but the lesions never developed actual warts; in some cases development of the parasite proceeded as far as the stage of summer sporangia but the injury was soon thrown off by the formation of necrotic tissue below the lesion, and other varieties tested in the same way resisted infection entirely [15, 16, 26, 27]. It is very difficult to say what property or properties possessed by these varieties confer upon them immunity from wart disease (they are not immune from other potato diseases), but whatever the nature of this complete resistance may be it appears to be controlled by hereditary factors [4]. Experiments conducted on the selfing or crossing of susceptible varieties have given only susceptible kinds; and selfing of immune varieties in certain cases gave a ratio of 3 : 1 immunes to susceptible, and the crossing of certain kinds of immunes and susceptibles yielded variable results [9, 29]. Amongst varieties which are not immune there are distinct gradations of susceptibility and resistance. Some of these varieties react readily or very slowly or not at all to the hypertrophic stimulus which follows upon infection; susceptible varieties which respond readily have a high degree, and varieties more or less resistant have a low degree of reaction to infection with the result that warts arise on susceptible but not on resistant tubers [22]. No anatomical differences have been revealed in the shoots of susceptible or immune varieties of potatoes in respect of relative resistance to wart disease [6].

Attempts to determine the existence of a possible resistant principle in the sap of immune potato plants have been made by several workers, but with no definite results. It has been ascertained that no materials manufactured by the leaves of susceptible or immune varieties are responsible in any way for the presence or absence of warts on the tubers [18, 19]; and all combinations of grafting of immunes with susceptibles, or vice versa, had no effect on the response of the tubers to the disease [35, 36, 37]. Certain differences have been reported to exist, however, between warted and sound tubers; thus, the hydrogen-ion concentration of warted tissue is said to be higher in comparison with healthy tissue of the same variety of tuber, but it is not believed that differences in acidity of the varieties are in any

way associated with immunity from the disease [46]. Furthermore, quantitative ash analyses of healthy and warted tissue showed a greater amount in the diseased tissue of most of the mineral constituents, especially of iron, manganese, copper and nitrogen, which appears to suggest that the stimulus to hypertrophy may be due to the diversion of these substances to the seat of infection [45].

Despite the immediate advantages obtained over the disease by the planting of immune varieties, however badly contaminated the soil may be, numerous attempts have been made in several countries to kill the organism in the soil with chemicals. Of these, the application of sulphur has given good results, the lethal effect being due actually to the formation of thiosulphuric acid in the soil, but so far no treatment of the soil for eradication of the resting sporangia has proved to be satisfactory without at the same time retarding the growth of the crop [3, 38, 39]. Recent experiments in Pennsylvania have shown the efficacy of ammonium thiocyanate, applied to infested soils at the rate of 2,000 lbs. per acre [16a]. Moreover, none of the ordinary seed disinfectants applied to potato tubers was found to be capable of destroying these sporangia in soil adherent to the tubers [49].

In Britain and in several European countries new varieties of potatoes are constantly being tested for reaction to wart disease and new kinds are added periodically to the official list of varieties approved for planting in contaminated soil. The following are some of the commoner immune varieties [1a, 1b]:

Early: Arran Crest, Arran Pilot, Ballydoon, Di Vernon, Herald, Immune Ashleaf, Snowdrop, Home Guard, Strathearn, Ulster Premier, Ulster Prince.

Second Early: Alness, Arran Comrade, Arran Luxury, Arran Signet, Ben Lomond, Catriona, Dargill Early, Edzell Blue, Ulster Ensign, Ulster Emblem.

Early Maincrop: Abundance, Ally, Arran Banner, Doon Star, Gladstone, Great Scot, King George, Majestic, Stormont Dawn, Ulster Cromlech, Dr. McIntosh, Ulster Early, Arran Viking, St. Aidan, Ulster Commerce, Venus, Red Fife, Ulster Leader.

Late Maincrop: Arran Cairn, Arran Consul, Arran Victory, Champion, Doon Pearl, Golden Wonder, Kerr's Pink, Redskin, Stormont Star, Craig's Bounty, Ulster Supreme.

Wart disease is notifiable within the operation of the Destructive Insects and Pests Acts, and any occupier of land on which the disease exists must report the occurrence to the Ministry of Agriculture, or to the Local Authority [1].

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Watery Wound Rot of Potato, *Pythium ultimum* Trow

This soft rot of potato attacks chiefly the larger 'ware' tubers soon after the crop has been dug. It is liable to appear on tubers lifted prematurely, on which the skin is imperfectly suberised and sensitive to injury, for it is only through wounds that the organism causing this rot can enter. Such wounds are often inflicted when the crop is turned out with a spinner, or when roughly treated during handling or grading. Loss of tubers through this disease is severe if the weather is hot soon after lifting. In storage again, losses may be high owing to the difficulty of detecting early stages of the trouble in small wounds; and on account of the rapid nature of the disease, wastage during transit under warm, close conditions may render an entire load or shipment worthless [2, 5]. The same trouble is known to occur in tubers cut for seed, soon after planting, whether on light or heavy soils, but seldom if whole tubers are used [4]. Sometimes, affected 'sets' disintegrate completely into the soil before making any growth, while at other times tubers only partly affected produce weakly growth.

Starting from a wound or abrasion, the rot causes the skin around the lesion to become dark and moist, and a black zone arises between the rotted and sound

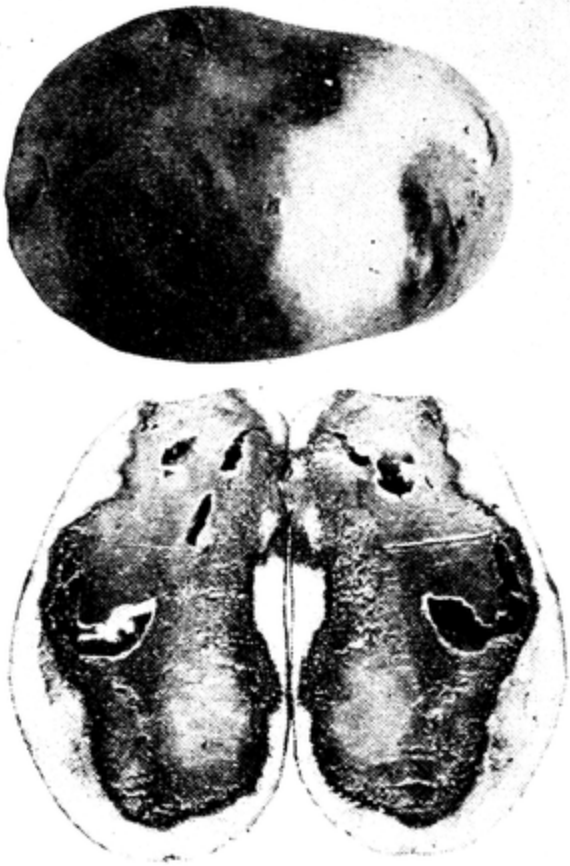


FIG. 251.—Watery wound rot of potato (*Pythium ultimum*). Top, tuber showing a dark, affected portion. Below, another tuber cut in half, showing cavities and dark line of demarcation between diseased and outer sound tissues (Lft. 292, Minis. Agric.)

parts of the tuber (Fig. 251). With further progress into the tuber the fungus brings about a shrinkage of the tissues, and over the affected area the skin becomes stretched and collapses when touched. A great amount of watery material oozes freely through the broken skin, a feature which distinguishes ' watery wound rot ' from ' pink rot ' of the potato (*Phytophthora erythroseptica*, p. 509), which leaves the tuber whole but soft like india-rubber. This watery rot is also much more rapid in its progress than pink rot, and, under favourable conditions to the fungus, a good-sized tuber may be completely rotted in a day or two.

When a partly diseased tuber is cut open, the black zone is still seen to be confined between the rotted and sound tissues, but very quickly, on exposure to air, the entire affected part turns grey, then brown, and finally almost black but with splashes of pink here and there. This pink discoloration is again reminiscent of the symptoms of pink rot, but in the latter disease the pink colour appears first and finally changes to a velvety black. According to variable external conditions or, perhaps, to the variety of tuber, watery rot is sometimes checked when it approaches the firm tissues of the vascular ring ; usually, however, the central tissues of the tuber break down and, when the watery contents are completely discharged, the interior gradually becomes dry and the tuber becomes more or less hollow. Beyond a slight ' fishy ' odour the rot imparts no unpleasant smell to the decaying tuber until saprophytic fungi and bacteria complete its decay.

Watery rot is caused by *Pythium ultimum*, a soil-inhabiting fungus [1, 5, 9]. It attacks numerous plants besides the potato ; namely, begonias, tobacco seedlings, pumpkins, melons, tomatoes, sweet-potatoes (*Ipomoea*), and others [3, 7, 8]. The branched, non-septate mycelium is developed in abundance in the rotted tissues, isolating the cells by dissolving the middle lamellae, but leaving the contained starch intact ; the mycelium is inter- and intracellular [2, 6, 7, 9]. Under natural conditions no reproductive cells of any kind have been found in the diseased tubers. In artificial culture, however, on potato, corn-meal, oatmeal, or potato-dextrose agar, a white, cottony mycelium is formed which produces sporangia, sexual organs, and oospores [8, 9]. The multinuclear sporangia, developed on unspecialised hyphae, are spherical when produced terminally and measure from 12

to 28 μ in diameter, but if intercalary, they are barrel-shaped and range from 17 to 27·8 by 14 to 22·9 μ [9]. The sporangia germinate direct by germ-tubes, and have not been seen to form zoospores [2, 8, 9]. The oogonia are terminal and spherical, rarely intercalary, from 19·6 to 22·9 μ in diameter; antheridia, one to each oogonium, are spherical, 14·7 to 18·3 μ in diameter; oospores are smooth, thick-walled, yellow, uninucleate, and may germinate immediately or after rest, up to 7 months, to form one or more germ-tubes which may [1a], or may not [9] bear vesicles with zoospores. Optimum growth of the fungus in culture occurs at relatively high temperatures of 25 to 28° C., the minimum and maximum being 4 and 40° C. respectively [4, 8]; optimum *p*H for growth lies between *p*H 6 and 8 [4].

The rotting of cut sets after planting, in all types of soil, increases as spring temperatures begin to rise. The disease is severe after a hot dry summer, and extensive rotting occurs if hot weather prevails soon after harvesting [2, 5]; it spreads rapidly in tubers stored at high temperatures, under poor ventilation, and heavy losses are incurred in store and transit under these conditions [4].

There can be little doubt that *Pythium ultimum* survives in the soil from year to year, in what form is not known, but tubers rotted by it and left in the soil never fail to repeat the trouble. All rotted tubers should, therefore, be gathered up and destroyed. As infections are contracted through wounds in the skin, all care should be observed during lifting and handling, and if practicable the use of a spinner avoided, especially if it is desired to lift the crop before it is mature, and if hot weather prevails at the time [5]. Only sound, unbruised tubers should be placed in store, and under cool conditions. Whole 'sets', in preference to cut tubers, will largely obviate the risk of contracting the disease at planting.

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Pink Rot of Potato, *Phytophthora erythroseptica* Pethybr.

This disease of potatoes was first observed in 1909 in County Galway, Ireland [11]. About eleven years later the same trouble was recognised in Scotland, and during the summer of 1921 it occurred in two localities in England on the varieties Majestic, Great Scot, and King Edward [5]. It now occurs in several parts of the British Isles, in Holland on reclaimed marsh soils, Bulgaria, the United States, and in the Dutch East Indies [1, 2, 7, 8].

Pink rot causes a wilting of the haulms as well as a moist rot of the tubers. The trouble starts while the tubers are developing and may be found on early varieties lifted early in July and on others from August onwards. In its action on the tubers 'pink rot' is much more rapid and destructive than 'potato blight'; losses in stored tubers are usually greater from pink rot than from blight, and a 50 per cent. loss in storage is not uncommon. The wilt is preceded by a chlorotic appearance



FIG. 252.—Pink rot of potato (*Phytophthora erythroseptica*). Affected tuber cut in half; the general effect is a pink discoloration finally changing to black (Adv. Lft. 292, Minis. Agric.)

of the foliage, leaves are rolled upwards and inwards, become dry and crisp. The leaflets show scattered spots of brown dead tissue isolated or joined to the already browned withered margins. All, or only some, shoots of the plant may be thus affected, but a cluster of aerial tubers close to the soil is a common feature. The most characteristic symptoms of the disease are, however, not fully revealed unless the plant is pulled up long before normal time for lifting, when the more pronounced symptoms are to be seen on the larger-size tubers; other tubers on the same stool may be entirely free or only partly affected. Unless the infection, in some way or another, has got into the new tubers through wounds or the eyes, pink rot always spreads into a new tuber from the stem through the stolon, that is, it starts at the 'heel end', spreading quickly towards the distal or 'rose end' of the developing tuber. These symptoms are manifest on tubers whether near the surface or deep in the soil. The rot also extends into the bases of some or all of the stems, but never much above soil level; stolons and roots are also affected, but the more striking symptoms of the rot are on the tubers. All stages of infection proceeding from the stems towards the developing tubers may be seen; thus, on the same stool, as already indicated, some tubers may be entirely free though the disease may be present in the stems, and in some cases it may progress along the stolons without reaching as far as the attached tubers, but in other cases it is evident that infection can proceed right along to enter the tubers at the 'heel end' and often entirely destroy them. Wholly affected tubers are discoloured brown and have the consistency of india-rubber, exuding juice when pressed, and with much sticky soil adhering to them. Tubers partly diseased show a black zone of varying width between the discoloured parts and the sound tissues; sometimes small black pin-points, coinciding with the lenticels in the skin, may be seen on the affected portion. On cutting a partly affected tuber the diseased portion is a dirty white colour and more watery in comparison with the sound portion. A striking feature, to which this disease owes its name, is the gradual change of colour the cut surface of the diseased tuber passes through on exposure to air. First it turns pink, then in about half an hour a deep salmon-pink colour appears which changes after a few hours' more exposure into a purplish-brown hue or nearly black (Fig. 252). These changes are the result of progressive oxidation and do not occur in absence of air. Moreover, the reaction of expressed juice from rotted tissue is quite acid at first but after the blackening process is complete the reaction is alkaline [11].

Several species of Phytophthora may produce, to greater or lesser extent, the above symptoms of pink rot. They are *P. erythroseptica* (Pethyb.), *P. cryptogea* (Pethyb. & Laff.), *P. megasperma* (Drechsl.), *P. cactorum* (Leb. & Cohn) Schroet, and *P. erythroseptica* var. *atropae* (Pethybr.) Alcock, but in no case are the symptoms more pronounced

and the disease more vigorous than when associated with *P. erythroseptica* (Pethybr.) [2, 3]. The description of the parasite is confined here to this organism.

Affected stems cut across show a brown discoloration in the vascular bundles. The fungus is found in the xylem and its presence there no doubt accounts for the wilting of the foliage, but it does not extend much into the shoot above soil-level. The epidermis and cortical tissues are decayed, without, however, showing as much browning as seen in the vascular bundles ; but the pith is often destroyed, the cavity being lined with disintegrated tissue, and it is in this region that the fungus mainly develops its reproductive organs, oogonia and antheridia. These organs may also be developed within rotted roots and stolons, rarely in the tubers. The mycelium is abundant, coenocytic, much branched, entirely intercellular, without production of haustoria, and in affected tubers starch remains unchanged. The fungus forms sporangia and resting oospores, but the sporangia are by no means typical of the *Phytophthora* group. Thus, they are not borne on aerial sporangiophores and are not adapted for aerial dispersal : they are produced only under aquatic conditions. Contrary to the prominent part played by the sporangia of *P. infestans* in spreading potato blight, the rôle of these sporangia of the pink-rot fungus in spreading infection is uncertain, and while the oosporic stage of the blight fungus is an uncommon phase of that disease, the oospores of the pink-rot organism are formed in great abundance in the stems, roots, and stolons of affected plants, and tide the fungus through the winter.

The sporangia, developed sympodially from undifferentiated hyphae, are obovate, non-papillate, 32 by 20 μ ; they are not dispersed but germinate in water while still attached, either directly by germ-tube, or indirectly to form, within an extruded vesicle, zoospores which, after coming to rest, develop a wall and round-off to form spores, 8 to 14 μ in diameter, which germinate direct (Fig. 253 A).

Oogonia and antheridia (amphigynous) (Fig. 253 B-D) develop on separate hyphae ; oospores are thick-walled (2 μ), 29 to 30 μ in diameter [10, 11]. They germinate direct both by long and short germ-tubes, in the latter case usually producing a large terminal cell which functions as a sporangium to form zoospores [9, 12].

The fungus is much easier to grow in culture on oatmeal or malt-extract agar [4], and on wort-gelatine than on the tuber itself [11]. The minimum, optimum, and maximum temperatures for growth are 5°, 25°, and 31° C. respectively. Light has no appreciable effect on culture

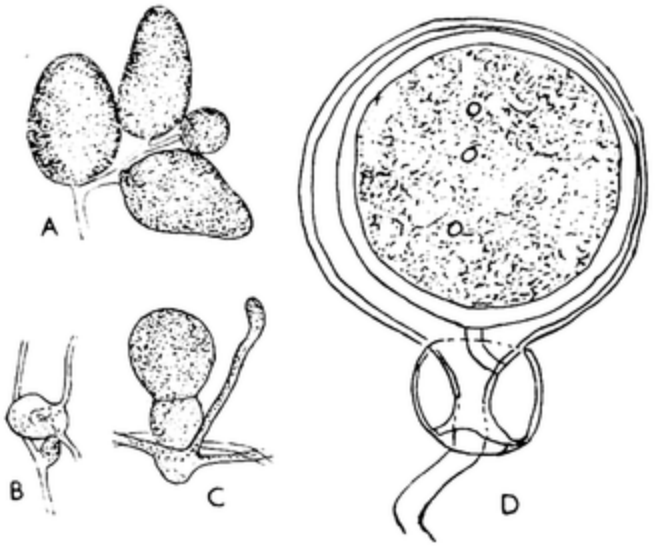


FIG. 253.—*Phytophthora erythroseptica*. *A*, a sporangiophore and sporangia showing sympodial development. *B*, the smaller (as yet) oogonium below, about to enter the base of the larger antheridium. *C*, the, now larger, swollen oogonium having broken through the antheridium, at base (amphigynous antheridium) (after Pethybridge, *Proc. Roy. Soc. Dub.*). *D*, an oogonium showing the large oospore and the empty fertilisation-tube (after Foister ; unpublished research)

growth, and growth under acid conditions (limit of *p*H 3·2) is vegetative, sexual organs and oospores being freely produced in neutralised media (*p*H 6 to 7) over a range of 5° to 25° C.; sporangia have not been observed on gel media [4], but were produced when small portions of a pure culture were transferred to water, sporangia being developed in a few days, both the sporangia and resting oospores being stimulated by adding a little potassium permanganate (0·01 to 0·02 per cent.) to tap water (until fairly pink); when incubated on a slide in a moist petri-dish, at 16° to 20° C., the culture produced sporangia in about 72 hours, and when the slides bearing them were exposed to room temperature zoospores were produced in 2 hours; germination of oospores can also be observed in this way [9].

The organism of pink rot is believed to survive in the soil for long periods of at least 4 or 5 years [2, 13], in what condition is not known, but in all probability as oospores. As already stated, these resting spores have been found in abundance in the dead host, in the pith, bases of stems, stolons, and sometimes roots, and in a few instances under the skin of tubers decayed in the soil [2, 12]. There is evidence again that *P. erythroseptica* can exist in the soil as a saprophyte, growing vigorously and even producing oospores in various types of sterilised soil [6], and it would also appear that it can grow under certain conditions in unsterilised soil and infect tubers in it. It has been established, however, that this organism competes very unfavourably with others in artificial culture and it is doubtful whether it can survive for sufficiently long periods in a normal soil as saprophytic mycelium to cause infections in subsequent seasons [2].

This disease is contracted from contaminated soil and in all probability infection starts from oospores liberated into the soil from remains of haulms, stolons, and sometimes roots, rarely from tubers [11]. It can possibly be carried also into clean land in soil adherent to ' seed ' raised on contaminated land, or through the planting of diseased tubers. In the former case experience shows that the risk is slight, for under moist conditions during storage, tubers are quickly attacked and are visibly unsound by planting time. However, under drier storage conditions this possibility of spread from contaminated ' seed ' cannot be ruled out, and the obvious precaution is to wash the tubers free from all soil at lifting, and drying them thoroughly before storing. The spread of disease from planting of infected tubers is doubtful, since tubers internally affected usually rot, becoming mummified during storage, and the planting only of normally sprouted sets will ensure the freedom of the crop from disease. Pink rot is not transmitted by contact of infected tubers with healthy tubers in dry storage, but under conditions sufficiently moist it is believed that the loss in storage is due to contamination from soil adhering to the tubers or in soil used in clamp construction rather than from direct transmission from diseased to sound tubers [2].

From the planting of sound ' seed ' in soil contaminated with pink rot, infection takes place only under excessively moist conditions of the soil. Indeed, it has been established without doubt that practically all the features on the tubers associated with pink rot can be repeated merely from simple asphyxiation such as would follow planting in water-logged soil or in land liable to flooding [2]. In the case of actual infection with the parasite, soils below 19 per cent. moisture-content did not encourage the disease, but above 24 per cent. infection set in,

increasing with rise of water-holding capacity until at maximum moisture all tubers became diseased (4). Under such favourable soil conditions, warm moist weather for a period of several days encourages attacks of pink rot. Infection of the germinating seed tuber (best between 20° to 25° C., rarely below 10° or above 30° C.) takes place at the ' rose ' end, through the ' eyes ', and unless infection is so severe as to destroy the sprouts before emergence — indicated by a gap in the row — the fungus soon enters the base of the stems, thence into the stolons, and to some extent into the roots. Infection of the growing plant having thus, to some extent, been established systemically, but not migrating much into the aerial stems, the developing tubers contract the disease via the stolons and, as stated, their infection, therefore, begins at the ' heel end '. Thus, there are two ways in which tubers can become infected, namely, from the soil at planting time, through the delicate tissues of the ' eyes ', mainly at the ' rose end ', and, as mentioned above, through the ' heel end ', while still attached to the plant ; tubers in storage also become infected through wounds or through the ' eyes ' (from contaminated soil adhering to them), the rot increasing with rise of temperature as spring succeeds winter.

Since excessive soil moisture is clearly the most important factor encouraging pink rot, best control can be obtained by attention to deep drainage. The trouble is practically unknown in moderately moist soils. Tubers from ground known to be contaminated with pink rot should not be used for ' seed ', since resting oospores may be carried on them in adherent soil. As the disease is soil-borne, a proper rotation should be observed for at least four years before restoring the potato crop. There is no strong evidence that the disease is spread through planting of infected seed, since such seed hardly ever survives till planting time, but a safe measure is to plant only vigorous, sprouted sets, thus ensuring perfectly healthy growth from the start. All infected debris of stalks, roots, and stolons (since they are the acknowledged means of returning the resting oospores to the ground), should be collected and destroyed by burning and not gathered to the manure heap.

It is recorded that varieties of potatoes with luxuriant foliage, encouraged in their growth with organic and potassic fertilisers, gave good results against this disease on reclaimed marsh soils in Holland, with deep drainage ; but no variety of potato is known so far to resist the action of pink-rot disease under moist conditions in contaminated soils of any kind.

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Potato Blight, *Phytophthora infestans* (Mont.) de Bary

Blight is, no doubt, the most important of all diseases of the potato in Britain and one which causes serious damage to haulm and tubers in humid weather during the growing season. It first swept through the potato fields of Europe in 1845. Reports of spot infections had been established in Flanders, near Liège, in Kent, the Isle of Man, possibly Ayrshire, and probably in Ireland late in the previous year; but epidemiological considerations based on the dates and distributions of the first attacks in 1845 preclude an earlier introduction of observable magnitude in 1844. Whence the disease came is uncertain; it was prevalent in New York and some of the New England States for two or three years before the European outbreak, and perhaps also in St. Helena, then an important port of call for home-bound European ships; but the likely home of the disease is Mexico, where it is endemic on wild native species allied to the cultivated potato, and it is not now possible to decide how or where it passed to the cultivated species, which is rare in that country (7, 9, 13, 22-26, 28, 68).

On the Continent, from a focus in Flanders, where blight was seen early in July 1845 (9), it spread with great rapidity through Holland, northern France, and eastern Germany. In the same year there is a doubtful reference to the disease in Jersey in June, and unmistakable evidence of its presence in Cornwall in July, Guernsey and south-east England early in August, the Isle of Wight a little later (13), and in the south-west of Ireland in September, over three weeks later than the date of the appearance of the disease in that area in the following and subsequent years. The resulting loss of tubers from the late attacks in 1845 and in the field crop in the following year had consequences in Ireland unparalleled in any other country, for the potato was the staple food of well over half the people, and famine, pestilence, and emigration reduced the population by a million and a half in the following five years (see p. 103). Subsequent spread has been so complete that probably every potato-growing country with suitable climate is infected.

The first sign of the disease above ground is the appearance on the leaves of small brown patches, which in suitable weather increase rapidly so as often to involve the whole surface (Fig. 254). Extension to the stalk quickly occurs in bad cases, and the entire crown may fall over in a rotten pulp in a few days. The influence of the weather is most marked. In dry, clear weather successful infections are limited in number and the resulting spots remain small, brown, and dry, while the stems may escape altogether. In warm, muggy weather with few periods of sunshine and a ground fog or drizzle, the colour rapidly changes to black, the rot is wet, the stems are quickly attacked, and a pronounced smell of decaying vegetable matter is given off and becomes one of the most marked features of the disease: the wet rot and smell are due in great part to secondary organisms, especially bacteria (butyric and others), which rapidly invade the killed tissues (49).

On the under surface of the brown spots (occasionally also on the upper) the fungus forms a whitish haze consisting of the sporangiophores bearing sporangia in great number, from the interior of the leaf. This growth is scanty or even absent in dry weather and dense but often evanescent on moist, cloudy days. The under-

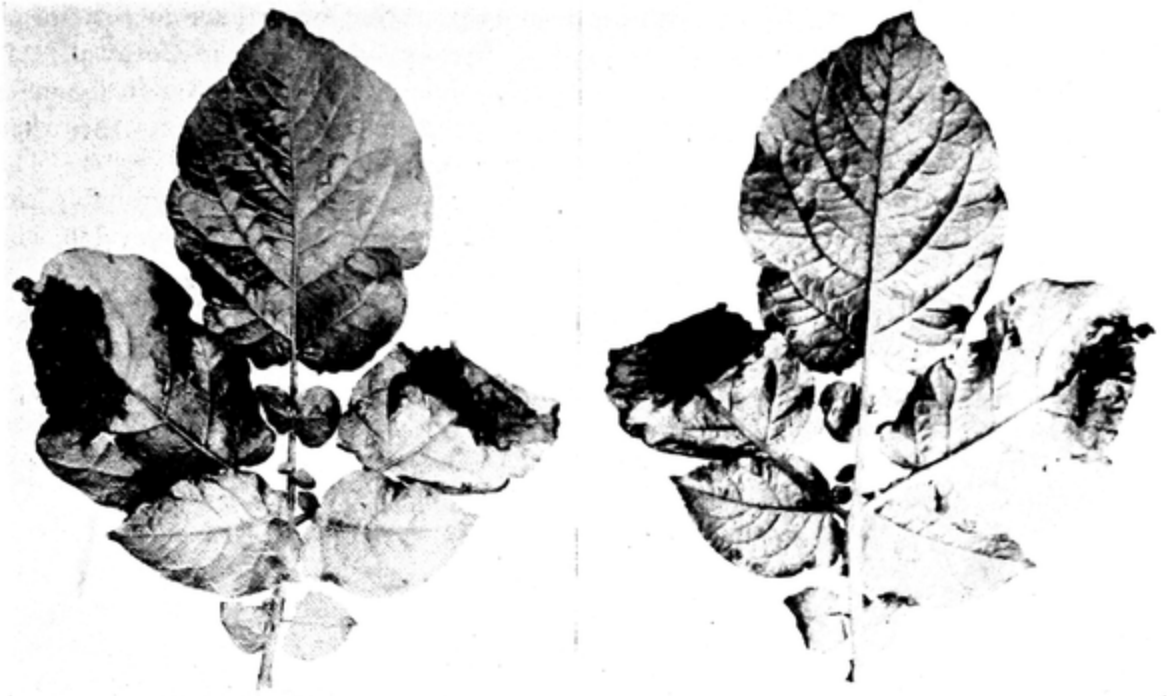


FIG. 254.—Potato blight (*Phytophthora infestans*). On the leaves. Left, upper surface. Right, under surface of same (photos by Foister & Noble)

ground parts, especially the tubers (Fig. 257), are also affected. Sometimes the attack is confined to them and cases have been described, for instance in Australia [36a], where there were no obvious symptoms above ground but the tubers were found well infected. The effect on the tubers, apart from a natural diminution in size and number when the green parts are early damaged, is a dry rot which does not soften the tissues but causes rusty-brown markings just below the skin and extending inwards for a variable distance in an irregular fashion, while the surface is marked by corresponding depressions due to collapse of the underlying cells. In a dry soil nothing more than this may result, and, with proper methods of storage, slightly infected tubers will remain unaltered until next planting season, when they give rise to sound or diseased plants, according to circumstances. In damp, heavy soils, however, a wet rot is usually set up by the action of secondary organisms and many of the tubers may decay before harvest; this wet rot may continue to spread in storage by contact, but the blight fungus itself does not pass from tuber to tuber, and nearly all the blight damage that occurs during storage is due to contamination of the tubers during lifting, or earlier, with spores from diseased leaves and haulms [26a, 48, 49, 52, 53, 81]. Tuber infection is much heavier in clay than in sandy soil, perhaps because the lenticels in the latter are more completely suberised than those of tubers grown in clay [34].

Phytophthora infestans, the cause of potato blight, belongs to the Peronosporales, or downy mildews; it occurs on a number of other Solanaceous hosts, the only one of importance being the tomato [7, 29, 39a, 71, 82, 90]. Some of the races of the parasite on these two hosts do not pass readily from one to the other, but others do; in Jersey, where large field crops of potatoes and tomatoes suffering severely from blight grow close together, the tomatoes may be a serious menace to potatoes though passage in the opposite direction

does not readily occur [82]. It is interesting to note its recent occurrence in Britain, on *Solanum crispum* in Devon and Somerset, and on *Lycium halimifolium* in Cornwall [47 b].

The mycelium in the tissues consists of intercellular hyphae 4 to 8 μ in diameter (Figs. 255, 256), and is frequently provided with lobed diverticula (Fig. 255 E); when the latter extend around the cells they may be mistaken for haustoria [19, 86, 87]. The haustoria in the tubers of some varieties are often thickened by a deposit probably laid on by the invaded cell (Fig. 256). In resistant varieties few haustoria develop, and

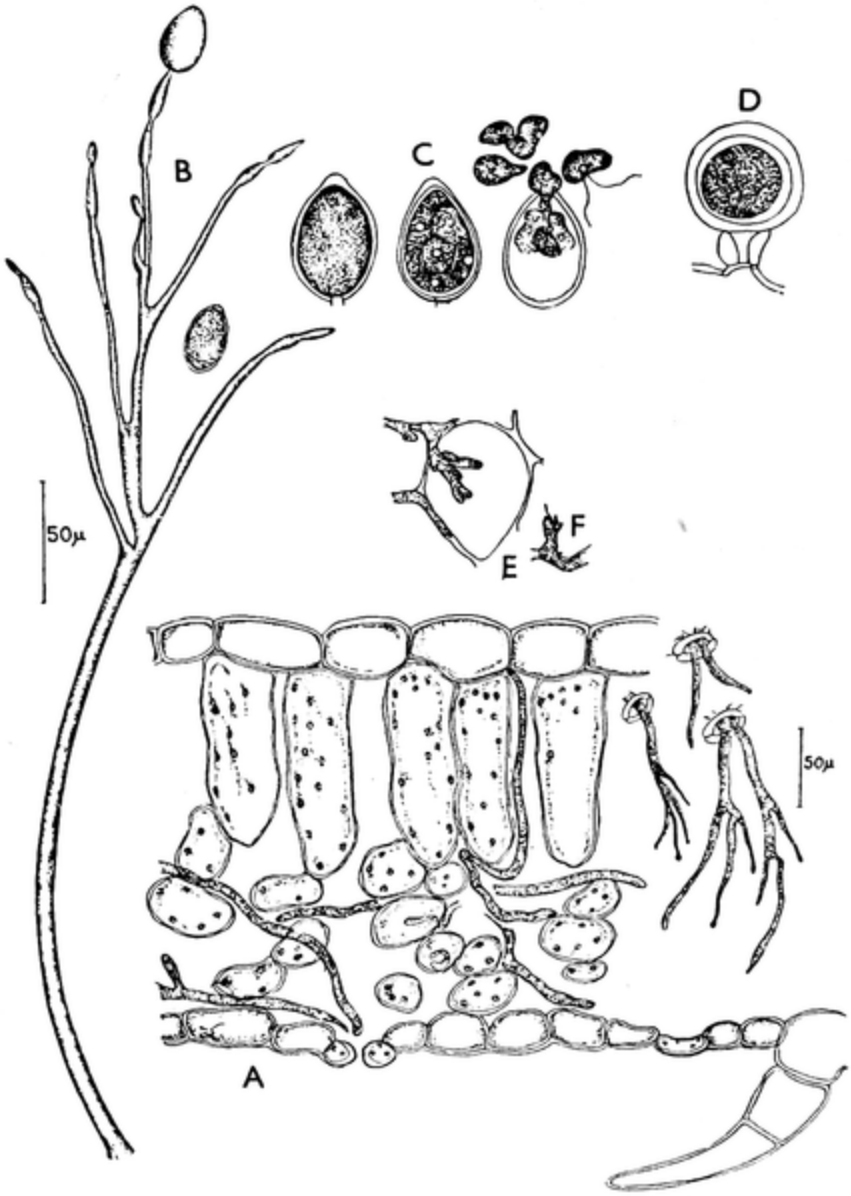


FIG. 255.—Potato blight (*Phytophthora infestans*). *A*, transverse section of leaf, at early infection, showing hyphae about to emerge at a stoma; at right, young sporangiophores emerging at stomata. *B*, a mature sporangiophore showing characteristic constrictions. *C*, sporangia showing indirect germination with formation of zoospores (after Ward). *D*, an oogonium showing amphigynous antheridium and oospore (after Pethybridge & Murphy). *E*, lobed diverticulum probably mistaken for an haustorium from mycelium in potato tuber (after Jones *et al.*, U.S. Dept. Agric. Bur. Pl. Ind., *Bull.* 245). *F*, haustorium in leaf (Butler's *Fungi and Disease in Plants*) (see Fig. 6).

thickened as well as uncovered haustoria are apparently only found in susceptible tubers. Reaction to *P. infestans* varies even among cells of genotypical identity. Further evidence of this is the observed difference between tubers and foliage in respect of resistance. This property in the foliage does not necessarily imply its presence in the tubers though resistant tubers produce the same type of leaves [47].

In favourable periods fructifications may appear on newly infected leaves in 4 or 5 days. The incubation period in the tubers may be much longer, for though early signs of infection may be visible around the lenticels after 3 or 4 days [36, 40], under normal conditions incubation may take about 28 days [53].

The branching fructifications or sporangiophores arise directly from the internal mycelium, emerging from the leaf through the stomata in little groups of about 3 to 5 together (Fig. 255); emergence through or between the cells of the epidermis is also sometimes found. On the tubers they mainly arise from lenticels or abrasions in the rind. The stalk is rather slender (about 10 μ), not rigid, and divides above into 2 to 4 branches of variable length (up to 1 mm.). Sporangia are formed at the tips of the branches while they are still short, but growth continues just below the spore, which is pushed over to one side and usually falls off; at each point where growth has been renewed there is a little nodular swelling in the stalk, as many as 9 or 10 such on a single branch. The sporangia are lemon-shaped, colourless, and measure from 22 to 32 by 16 to 24 μ (Fig. 255 B). When sown in water the apex becomes papillate and zoospores are liberated (Fig. 255 C). This indirect germination is the normal method when the fungus is active and the environment favourable; otherwise germ-tubes are given out from the sporangia, which thus behave as conidia and germinate direct [15, 50, 59, 60, 91].

Oospores were first seen in artificial cultures of the fungus [11] but later were obtained on diseased tubers under natural conditions [51, 63] (Fig. 255 D). Observations, however, have been few, and spread of infection is mainly by the asexual sporangia. The oogonia are pear-shaped to almost spherical, 31 to 43 μ across (average 36 μ), yellow or hyaline. They are fertilised by club-shaped antheridia [45], borne apparently on a separate hypha from that forming the oogonium, or are produced without fertilisation. The oospores have a thick smooth hyaline wall and vary from 24 to 35 μ (average about 30 μ) in diameter [11, 63]. In culture, thick-walled, pale amber-tinted chlamydospore-like bodies may form at the ends of hyphae [21] but these have not been germinated.

The fungus is not very readily cultivated on artificial media but can be successfully grown on various substances such as quaker oats, raw aseptic potato plugs, sterilised soil, white bean agar with 40 per cent. saccharose, and haricot bean agar [14, 15, 20, 33, 61, 79, 85].

Infection takes place through any part of the epidermis on the green parts, most freely on the under side of the leaves, and through wounds, lenticels, and the outer scales of the eyes on the tubers [40, 53]. As infection of the latter is ordinarily from shed sporangia, including the zoospores from sporangia, tubers near the surface are most often attacked, unless (as happened not uncommonly in the early days), a misdirected effort is made to save the crop by lifting early while the green tops are still shedding viable spores.

Primary outbreaks of blight in the field usually come from the planting of infected tubers [1, 18, 32, 38, 39, 54, 62, 73]. It has been conclusively established that the mycelium in the tubers frequently survives without causing any great damage, coming sometimes from crops that showed little disease; on planting, it renews active growth, passing into the tissues of the young sprouts chiefly between the

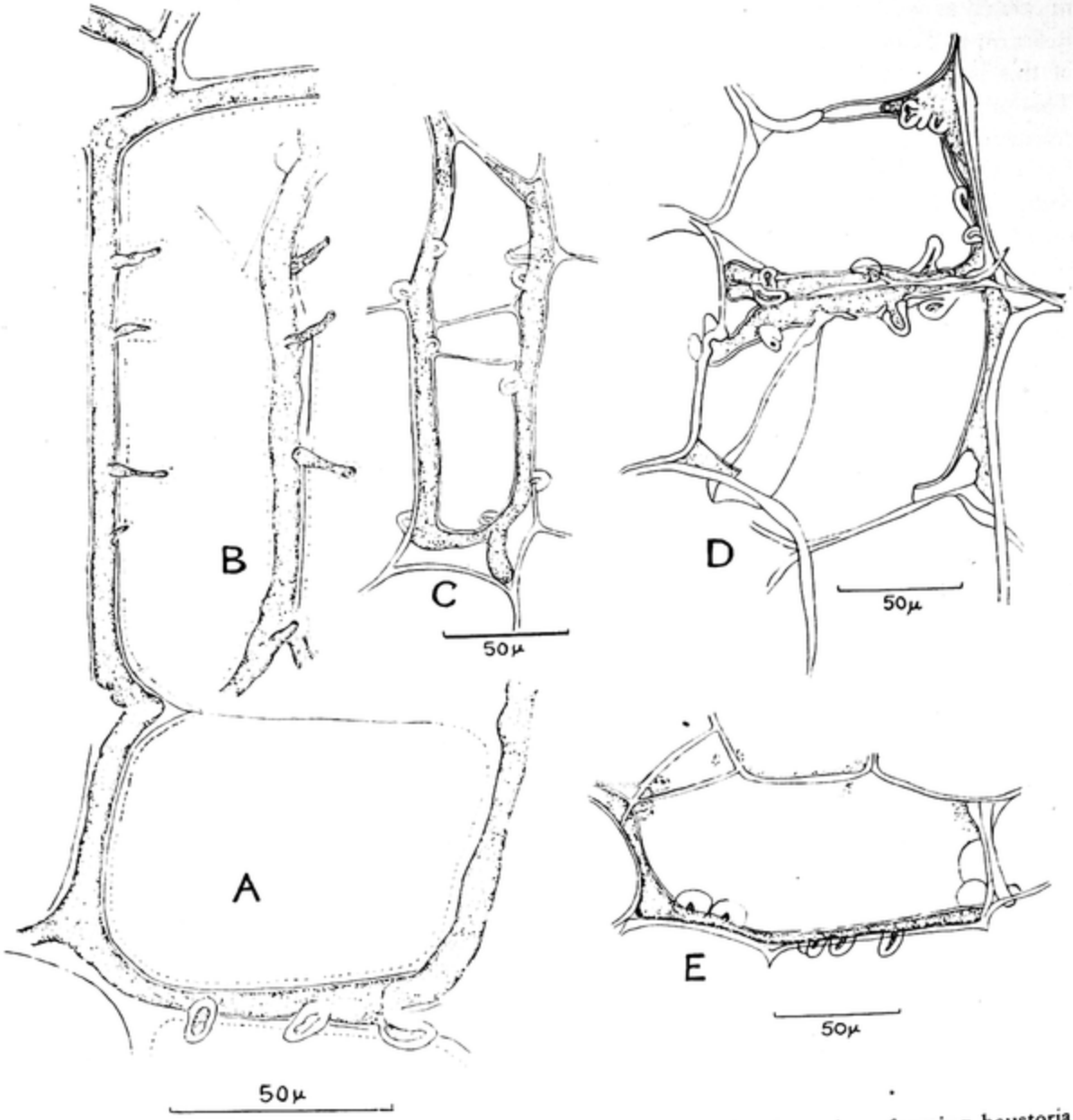


FIG. 256.—Potato blight (*Phytophthora infestans*). The fungus in the tuber, forming haustoria (from work still in progress at Royal Holloway College, by courtesy of Miss E. M. Blackwell). *A*, *B*, from variety Majestic, sections of fresh material treated with iodine and 70 per cent. sulphuric acid, stained with cotton blue in lactic acid, from a clamp, in January. *C*, from variety Arran Banner (October). *D*, *E*, from variety Up-to-date (over-wintered in the clamp and examined in April); *C*, *D*, *E*, treated with lacmoid. All mounted in glycerine.

cells of the cortex, and sporulating on the sprouts above ground if the weather is favourable (Figs. 97, 257). In storage, infected tubers sprout earlier than sound ones [62], and if the same occurs in the field, early sporulation will result. Wind dispersion of the spores thus produced starts secondary infections. Sometimes the sprouts from infected tubers may be healthy or some may contain the fungus and others not (Fig. 257). It is possible also that in soils of open texture sporulation may occur before the sprouts emerge and underground transference of spores is not precluded [41, 57]. Little is known of the life of the fungus in the soil except

that it can survive for eleven months in sterile clay [20]. It has also been suggested that spread from diseased seed tubers may take place just as has been observed to occur in the vicinity of old clamps, presumably from waste diseased tubers left about the site or buried when the new crop is planted [8d, 32]. Experiments in Ireland and in Russia have shown that the fungus from such material can sporulate in or on the surface of the soil [54, 57], and it has also been established that the sporangia can remain viable in soil for nearly two months under summer conditions [34, 35]. Survival on old haulms or shoots from ' ground keepers ' (volunteer plants) has also been observed in the West of England and blighted haulms are found throughout the growing season in parts of the United States [15]. Nevertheless, though these observations may provide a partial solution of the difficulty that blight rarely develops in the field before the plants are 2 to 4 months old, seldom indeed until they have finished flowering, whereas spread from primarily infected sprouts would be expected much earlier, attempts to account for this by proving that the fungus can persist in the soil normally for long periods, as from season to season, have failed. The view has also been expressed that sprout infections are insufficient to start the widespread outbreaks that occur [8d, 33]. It was at one time thought that susceptibility increases as the plant matures, but this is not true, for the fungus can establish itself more quickly on young than on old foliage [15]. The tendency for the infection to start at the base of the plant is due largely to the more favourable conditions of the microclimate (see p. 168), such as abundance of humidity provided by the dense growth in that situation. It has been observed that an attack may develop with differing intensity on two equally susceptible varieties according as their habit of growth induces a more (Suicisse) or a less (Early Rose) favourable microclimate [32, 32a].



FIG. 257.—Potato blight. The fungus in the tuber. A cut tuber of the variety Great Scot (photo by permission of Pathological Laboratory, Ministry of Agriculture) and a sprouting tuber producing a diseased shoot and two apparently healthy shoots (after Alcock & McIntosh, *Ann. App. Biol.*)

The climatic and physical conditions favouring infection and the development of the parasite in the tissues have been referred to earlier (see Chapter III, p. 109) [4, 5, 16, 32, 43, 88, 89, 92]. Aerial emergence of the hyphae takes place only in an atmospheric humidity exceeding 85 per cent., while sporangia apparently

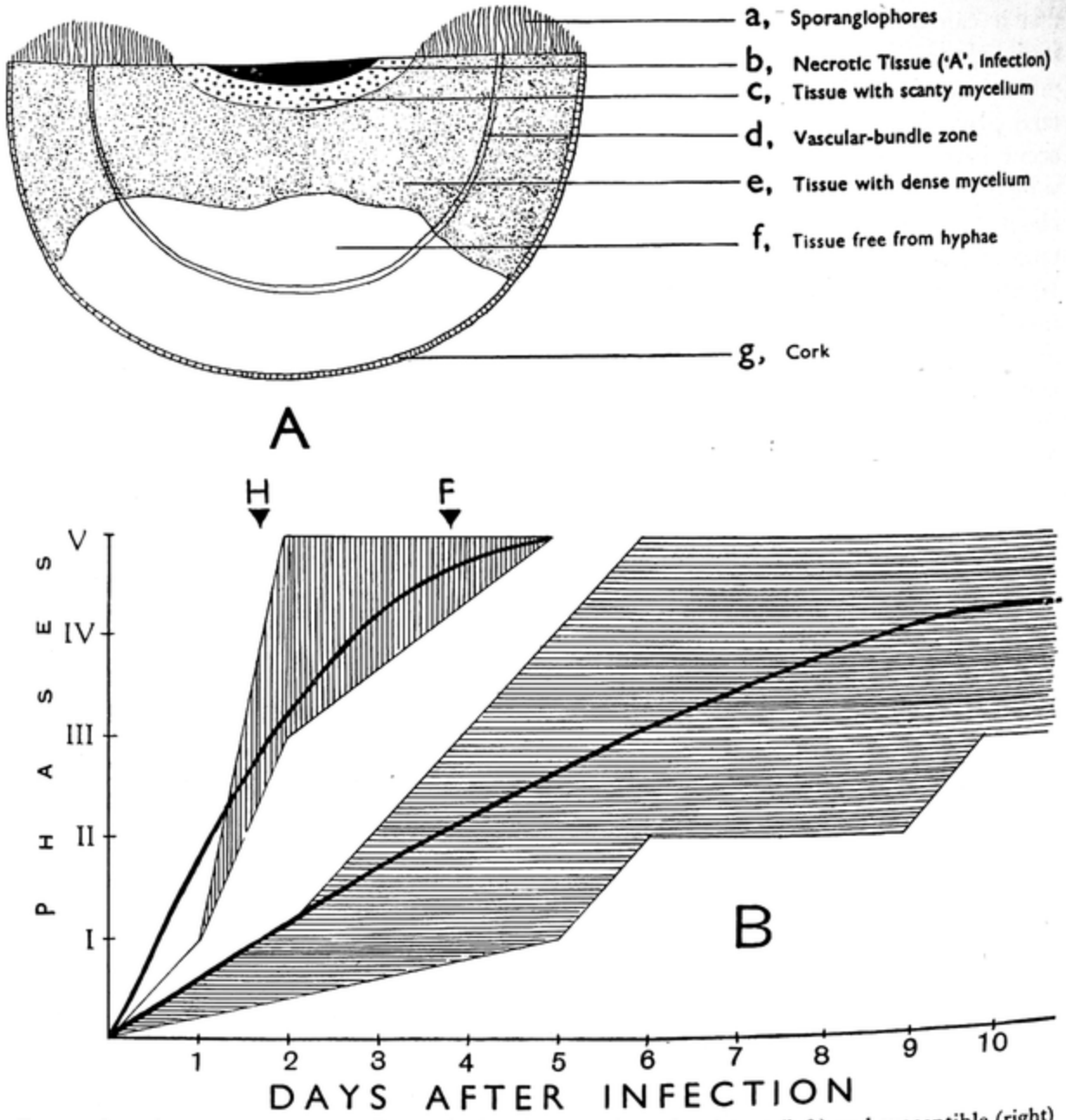


FIG. 258.—*A*, see text, p. 522. *B*, graph showing reaction of resistant (left) and susceptible (right) tubers to infection curve. *H*, *F*, incidence of haustoria and fructifications. Phase I, no change; II, plasma granular, nucleus swells; III, plasma fibrous, brownish; IV, nucleus shrinks, walls browned; V, cells dead (after Müller)

form only above 95 per cent. [16] and do not become abundant under about 97 per cent. [15]. Germination of the sporangia occurs on immersion in a film of water and is quicker if their water content has first been lowered [58]. The most favourable temperature for the production of sporangia is about 18° to 22° C. and for development of zoospores about 10° to 13° C. (extremes 6° to 15°), but direct germination occurs through wider limits, 4° to 30° C. [15, 50, 59, 91]. Darkness appears to inhibit production and germination of the sporangia. The rate of mycelial growth in the tissues is directly proportional to the water content of the leaves or perhaps to the ratio of water to nitrogen [12]; there is evidence that

the plants are more susceptible to the fungus when nitrogen is withheld [74], and growth is most rapid (shortest incubation period) at 20 to 23° C., though good growth has been got at 27° C. [39].

Where the mean temperature exceeds 25° C. (77° F.) blight is practically unknown. Rapid development often follows a period of unusually cool weather bringing heavy dews or fog and stimulating the germination of the sporangia at the critical time, followed by a warmer moist spell to secure a short incubation period. The efforts to forecast outbreaks of blight and the physical data on which they are based have already been discussed (see Chapter V, p. 168).

Cells of leaf and tuber tissues in contact with or near the mycelium turn brown, both walls and contents being affected. In the rotted tubers there is an increase in the pentosans and crude fibre and a decrease in dry matter [31]. Starch is not hydrolysed until the cells become invaded by secondary organisms [84].

The pandemic in the middle of last century swept many of the existing varieties of potatoes out of cultivation, including most of those (except the Regent) then grown in the British Isles. It also greatly stimulated the production of new varieties from true seed, and before long there was evidence of the existence of varietal resistance, sometimes in the foliage, sometimes in the tubers, sometimes in both [21, 26 b, 39 a, 64, 70]. The first great variety produced was probably Nichol's Champion, raised from seed of unknown parentage at Ochterlony in 1862, though Myatt's Ashleaf raised in 1853 and the Rock, grown widely in Ireland until the Champion came, had considerable resistance [17]. The running out some twenty years or so after they have reached their prime, which has been the fate of most of the improved varieties thus continually introduced down to the present day, is attributable to the accumulation in them of virus diseases rather than to any direct increase in their susceptibility to blight (see Chapter VIII, p. 256).

The two principal methods employed in the control of potato blight, as of so many other diseases, are direct attack on the parasite by spraying and dusting, and the cultivation of resistant varieties. Spraying to check blight is probably more widely employed and gives more satisfactory results than with any other field-crop disease. On the whole, no fungicide is superior to Bordeaux mixture against this disease, but Burgundy mixture is preferred in many parts of Ireland [55, 56] and dusting is popular elsewhere (see Chapter VII, p. 235). Correct timing is more important than the choice of the fungicide [6]. Forecasting services help in determining this in some areas [6 a], but elsewhere it is the practice to give the first application when the plants are 6 to 8 inches high, repeating every ten days or a fortnight, sometimes until six applications have been given. In Britain the early crop is not usually treated, as it matures before the onset of the disease. Main crop varieties may require the first treatment between the middle of June and mid-July, depending on the district and state of the crop and of the weather. Frequent earthing-up of the growing crop diminishes the risk of tuber infection, while spraying to destroy the haulms two or more weeks before lifting has become increasingly popular in recent years, 100 gallons per acre of 10 to 20 per cent. sulphuric acid or 5 per cent. copper sulphate, or 12 lb. of the sulphate plus ¼ lb. caustic soda in 40 gallons water, being used [3, 37, 80]. Tar-acid sprays are effective non-corrosive substitutes for the acid [93]. In view of the danger of surface

contamination from diseased haulms these should not be used to cover the clamps. The tubers should be stored dry and the clamps as well ventilated as possible. Immediate disinfection on lifting tubers destined for seed has been beneficial in some tests, 0·1 per cent. corrosive sublimate or a dip of a half to one minute in an organo-mercury compound being advised [26a].

Breeding for blight resistance has been methodically pursued in several countries during recent times [8-8c, 21, 21a, 27, 40-47a, 65-69a, 72, 77, 78, 83a, 88a]. The first step, as usual, has been to obtain a sufficient range of variable parent material for intra- and interspecific crossing and subsequent selection. The inoculation of tubers for tests is usually performed with zoospores, maintaining the tubers at a constant temperature of 19° C. [47a]. Since the European varieties are probably descended from only a few primitive types [43], a wider choice of immune and partially immune varieties has been sought by various expeditions to Central and South America [28a] (p. 250). The wild immune hexaploid *Solanum demissum* from Mexico, when used as the female parent, has yielded many hybrids with cultivated potatoes [41, 42, 43]. Promising results have already been secured by crossing cultivated varieties with certain wild races of Central or South American origin known as the 'W' races. Müller [47, 47a] in Germany has found the reactions of these races to inoculation to proceed in five stages, and with the aid of a 'vital' stain (methylene blue) these reactions of the tubers could be examined step by step, until the cells were killed, the necrotic tissue forming a barrier to further spread of the disease. These stages follow rapidly on one another in resistant varieties but slowly in susceptible ones, the *genes for resistance acting simply as accelerators*. Fungal development in resistant varieties is poor, haustoria and sporangia are few or absent, the final phase being reached considerably earlier than in susceptible varieties [47a] (Fig. 258). The reaction is one of *speed of production* of a substance toxic to the fungus, and not one of the presence or absence of a toxin. A complication in the breeding for blight resistance is the discovery of new strains of the fungus, 31 in all being recorded in experimental areas in Germany.* Attempt has been made to group these into '*A*', '*B*', '*C*', etc. strains, but these designations are a little unreal as all gradations of host reaction towards them exist. Strain '*A*' is the most important as it attacks all commercial varieties, but to which the 'W' types are highly resistant though not immune. Thus the German variety Aquila is resistant to British strains '*A*' and '*C*', but susceptible to strain '*B*' of blight. In Fig. 258A, a 'W' tuber is shown inoculated with strain '*A*', and the reaction of the tuber has been so rapid that a necrotic area (black) has been formed. Local 'immunisation' ('vaccination') has been set up in a zone of tissue extending for some 20-30 cells below the necrotic area as shown by subsequent (1 hour) inoculation with a strain of *P. infestans* to which the tuber is susceptible. This strain avoids, or grows sparsely into, the 'immunised' zone, but normally into the tissues below, which the immunising effect, following the first inoculation, had not reached. The reaction between the tuber and *P. infestans* is not specific, and a similar reaction may take place against other parasites. The substance exosmosed from the hyphae is, however, specific and has been isolated by Müller and his students.

* Communicated by Professor K. O. Müller.

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Black Scurf of Potato, *Corticium solani* (Prill. & Delacr.) Bourd. & Galz.

' Black scurf ', ' Rhizoctonia disease ', or ' stem canker ' of potatoes is found wherever the crop is grown. It is caused by a soil-inhabiting basidiomycete, *Corticium solani* [21], better known as *Rhizoctonia solani* [12], the name applied to the sterile mycelial form of the fungus. The latter exists as a brownish mycelium

on the tubers and sometimes on the basal parts of the plant itself, forming small, shiny black sclerotia, superficial and easily removable, spreading over these parts in black, scurfy incrustations (Fig. 259). The fungus attacks an unusually wide range of hosts, some 230 plants belonging to 66 families being listed, in 1930, as susceptible hosts [3]. They include ornamental plants and weeds, as well as plants of economic importance of such diverse habit as coniferous trees, lettuce, sugar beet, tomatoes, cucumbers, etc.

Reports vary considerably as to the importance of black scurf in respect of losses in the potato crop. In Britain the disease does not assume serious proportions, and if cultivation is good, and proper rotations are observed, losses are small; but crops grown in the same land every year, especially if the soil is heavy, wet, and undrained, may show heavy losses [1]. In Washington [7], from the point of view of losses sustained, it is reported to rank second only to virus diseases in this host, and it is of increasing importance in Germany [16a, 26a].

While the effects of the disease on the tubers themselves, in the form of superficial sclerotia, are not of a serious nature, the attacks of the fungus on the basal parts of the stem, by reducing the vitality of the green shoots, may impoverish the



FIG. 259.—Black scurf of potato, *Corticium* (*Rhizoctonia*) *solani*. Affected plant showing down curvature of the leaves near the top, indicating an attack by the fungus on underground part of the stem (this feature is sometimes mistaken for a symptom of primary 'leaf roll' due to virus infection; see Figs. 164, 269). Insets, left, dark mycelial strands of *Rhizoctonia solani* around an eye (enlarged); right, the black sclerotia of same (photos by McKay)

whole underground system of the plant, with the result that the yield of tubers is reduced. It is still uncertain to what extent the fungus causes a rotting of the tubers [15a]. The symptoms on the subterranean parts arise by direct action of the parasite on these parts; those shown by the green shoots follow indirectly upon underground infection and disturbance of the normal functions of the rooting system [7]. The fungus in the soil attacks the young sprouts on the germinating tuber from the earliest stages of growth, destroying them on the seed-piece, or causing them to develop lumps like small tubers instead of shoots. Sprouts which survive these early attacks may become infected at any time before or soon after breaking through the soil; as many as six or more unsuccessful attempts may be made before the germinating tuber eventually produces a shoot above ground [7]. Young tips may be attacked and browned, or lower down on the sprouts, brown, sunken, circular or elongated spots may appear. The growing points of stolons, like those of young shoots, are especially liable to attack. These infections during early growth are said to be correlated with prevailingly low temperatures, and it appears that above 21° C. the young sprouts are able to make such good growth as to outpace the fungus, which, moreover, loses much of its virulence with rise of temperature [18]. This phase of the disease is common with early plantings, due presumably to lower prevailing temperatures, but there is no evidence that it

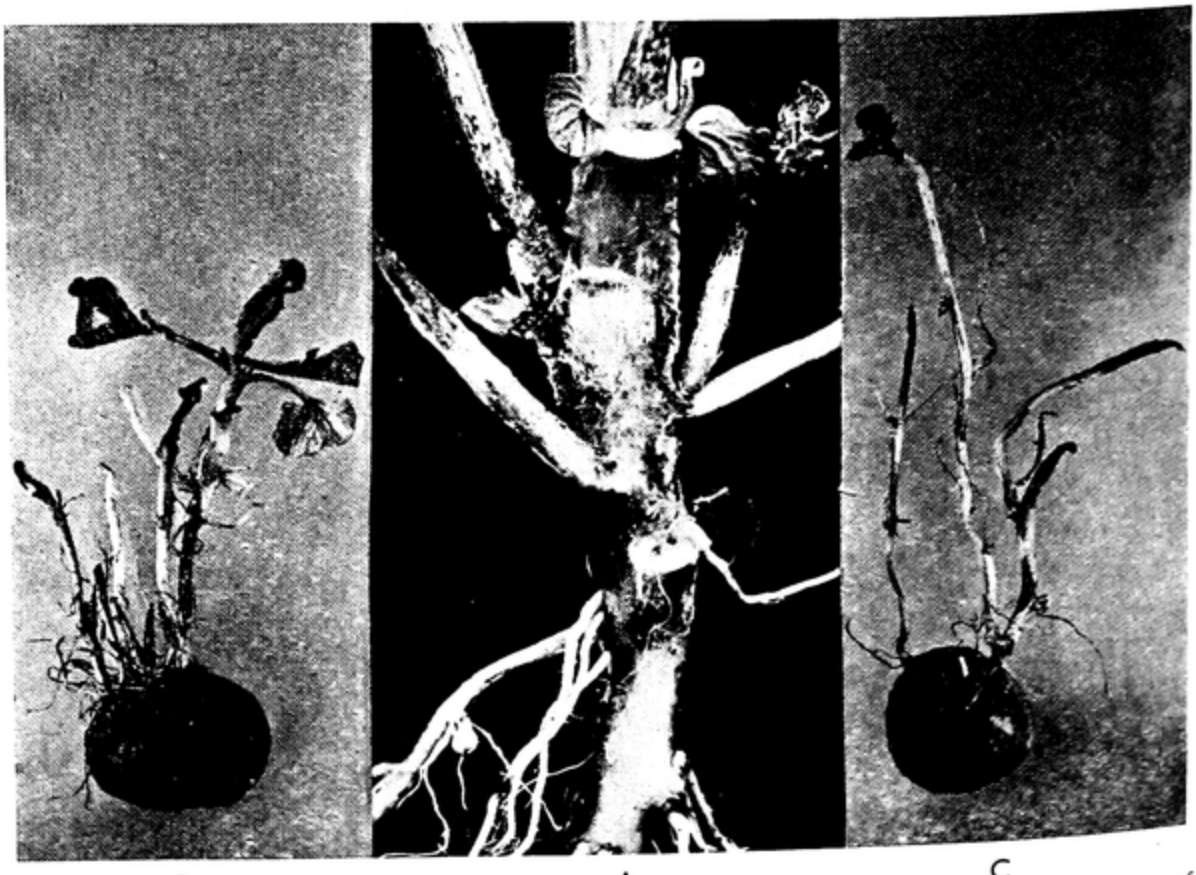


FIG. 260.—Black scurf of potato. *A*, base of stem of potato plant showing the white fruiting stage of *Corticium solani* (photo by McKay). *B*, *C*, potato sprouts affected by the canker phase of the disease (photos by Millard)

attacks early more than late varieties of potatoes [25]. But probably the most destructive stage of the disease is the production of one or more cankers which form irregular elongated brown areas on the underground portion of the main stem, at, or just below, the surface of the soil (Fig. 260). This canker phase of the disease seems to be serious around a temperature of 18° C., and even at as low a temperature as 9° C. damage may be extensive [18]. While some doubt exists as to the capacity of several strains of *Rhizoctonia* to destroy the sprouts before emergence, it is said that practically all strains pathogenic to potato are able to cause stem canker, with or without wounding [19]. The canker may be so severe as to girdle the stem, so that the part above the lesion dies. Below the seat of injury, however, one or more new buds may develop to produce fresh shoots which often escape infection and grow into vigorous plants. By the destruction of the cortical tissues of the stolons, cankers may form in precisely the same way on these underground branches as on the stem, and if the stolons are girdled the crop of young tubers may be lost entirely. Partially girdling cankers on stems and stolons are, of course, not so serious, but they interfere appreciably with the translocation of food and swelling of the tubers. Moreover, buds towards the base of the stem, instead of contributing to the formation of stolons, may swell abnormally and give rise to ' aerial tubers ' and, following upon the impoverishment of the rooting system, the parts above ground turn yellow, the uppermost leaves tend to become ' bunchy ', and there is often a peculiar rolling of the leaf edges (Fig. 259) which is by no means easy to distinguish from true ' leaf roll ' due to virus trouble [7]. Infected plants which have become more or less established may show early signs of new-tuber infection by the presence of brown discoloured areas extending from the affected stolons. These areas may consist of a mere russeting of the skin or there may result a more severe corrosion, marked by narrow cavities of irregular shape, sometimes as much as $\frac{1}{2}$ inch across ; such cavities may usually be found lined with the brown mycelium of the fungus. As the tubers mature, sclerotia begin to appear and, before harvest, the tuber finally develops a slow type of rot which starts at the ' heel end ', and, if placed in storage, decay sets in in the form of a dry rot which is delimited from the firm flesh by a layer of toughened dead tissue [7]. Sclerotia are typically formed on the tubers (Fig. 259) and less frequently on the basal parts of affected stems and on the roots. They are of irregular shape, black, closely adpressed to the skin, and vary in size from a pea to a pin-head. Their formation on the tuber is stimulated by falling temperature and they occur more in dry than wet soils, and arise in greater number on the under than the upper side of the tuber in the ground. They are heralded at various spots on the tuber by small groups of white cottony hyphae composed of closely interwoven filaments derived from the brown investing mycelium (Fig. 259) ; the groups gradually darken and harden to form the black sclerotial bodies ; they develop in the same way in culture, from small, white, woolly masses of mycelium. Being entirely of superficial origin, sclerotia do little material harm to the tuber and in no way affect the vitality or keeping qualities of the potato [7]. But, carried on the surface of the planted seed tuber, or liberated into the soil, sclerotia are probably the principal means of perpetuating the disease, and these resistant bodies are known to be still viable in the soil after more than a year [16].

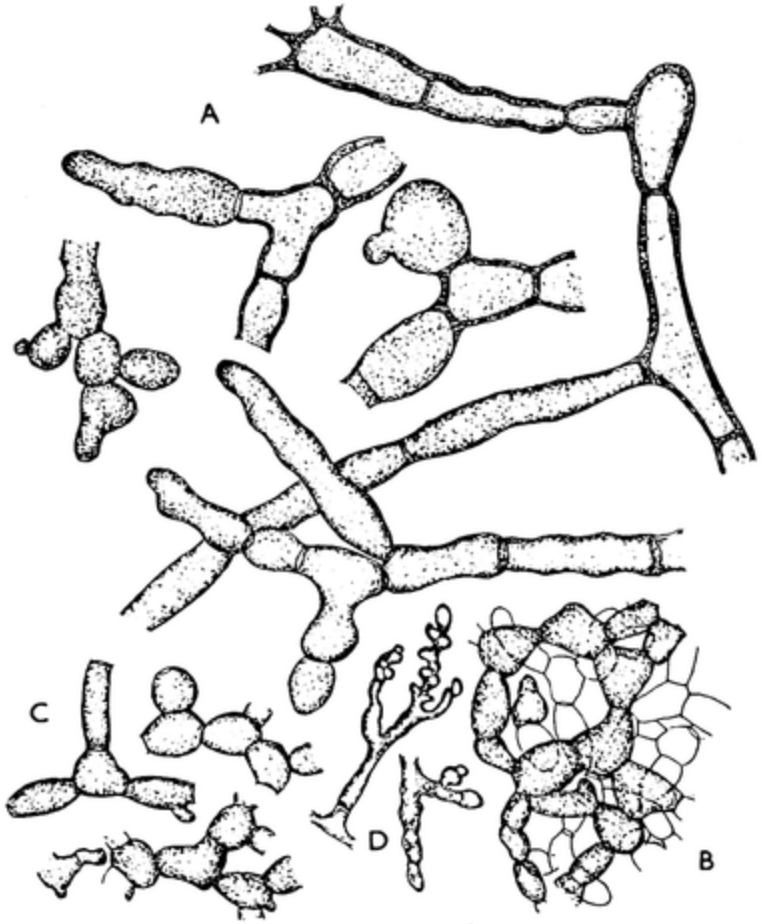


FIG. 261.—*Rhizoctonia solani*. *A*, lobulate, moniliform, and 'elbowed' cells of the mycelium, from a culture. *B*, from a section of a sclerotium on potato. *C*, cells isolated by maceration of a sclerotium. *D*, young hyphae from a young sclerotial tuft on lettuce (after Duggar)

The mycelium of *Rhizoctonia solani* varies considerably in appearance with age, both under natural conditions and in culture on a wide range of media ; at first colourless and vacuolated, later it deepens to a yellow and finally to a rather deep-brown colour, the cell-contents meanwhile becoming granular and finally disappearing almost entirely. A very characteristic feature is the constriction of the hyphal branches at the points of origin (Fig. 261). Though fusions between hyphae are fairly common, no clamp connections are formed. The organism grows in culture over a wide range of hydrogen-ion concentration, but variation in degree of acidity produces marked differences in growth-characters of different isolates, and affects also the size and colour of the sclerotia[15]. Under natural conditions the fungus appears to thrive best in acid soils.

The perfect, basidiomycete stage, *Corticium solani* (Fig. 260 A), has been observed by several authors. Though it has been stated that the fungus is homothallic [16], a study of this organism causing a disease of cotton in South Carolina showed, however, that the *Corticium*-stage was produced under artificial conditions on a non-living substrate only when multispore transfers were made [28]. A cool period and an abundance of moisture in the soil appear to be the necessary conditions for the development of this perfect reproductive stage. The fructifications are not at all typical of the higher basidiomycetes ; there is no well-defined sporophore, but a somewhat diffuse ashy-grey mat of mycelium arising from the dark-brown mycelium, prior to sporophore formation, gathers in greater profusion at some parts of the host than others so as to form a network chiefly around the base of the plant, and sometimes extending for several centimetres up the stem, and even extending as far as the stalks of the lowermost foliage leaves, but only for support. The hymenium consists merely of a layer of hyphae arising from the greyish mycelial mat, in which the septated hyphae show a multinucleate condition until they give rise, especially towards the margin of the diffuse hymenium, to a number of binucleate hyphae which finally bear the club-shaped basidia furnished with four sterigmata and spores ; the latter are colourless, oval to ovate, from 9 to 14 by 6 to 8 μ ; there are no cystidia [16]. There is no evidence that the basidiospores ever cause infections, and attempts at artificial infections are reported not to be successful, but further investigation

is desirable since it has been found that isolates obtained from spore collections yield a very high percentage of virulent strains while other strains appear to be harmless [23]. Moreover, as above mentioned, the complete life-cycle of the fungus is effected only when multispore cultures are grown [28]. The fungus possesses a great number of different strains, and attempt has been made, on various grounds, to arrange them in groups. Some strains appear to be associated with certain types of lesions on the stem, either large and deep, or mere superficial flecks; and some strains are more liable than others to attack and check the development of the tuber-sprouts [9, 23]; and while many strains differ considerably in virulence on the same host under like conditions, variability also exists in the amount of mycelium, capacity to form sclerotia, the size of sclerotia, degree of enzyme activity [8], and ability to attack the host only within certain limits of temperature [15, 27]. Certain isolates have a different host range [14]; thus, as mentioned elsewhere in this book (p. 583), the strain attacking sugar beet (the *Corticium*-stage has not been found on the beet) does not attack the potato, and vice versa; but there is still a great deal to determine in respect of physiologic specialisation within this species [14, 16a, 23].

There is little doubt that the organism of black scurf is capable of existing in the soil as a saprophyte [2, 4, 26a]; it possesses a considerable degree of resistance to the metabolic products of other soil organisms [16]; and from mycelium and sclerotia which have over-wintered in the soil, or from sclerotia carried on seed tubers, infections of potato plants start anew.

The rejuvenated mycelium in the soil, or the mycelium given forth by the germinating sclerotia, attacks the tuber at the eyes, and as already stated proceeds to penetrate the young sprout at the apex, or a little lower down, to form brown, sunken lesions. Inoculation experiments show that the tips of the infecting hyphae exert considerable mechanical pressure on the cuticle and epidermis of young shoots, and after entry the epidermal cells are rapidly destroyed. The middle lamellae of the underlying tissue are early dissolved and the fungus soon finds its way into the meristem of the young shoot. Thus the growing point may become invaded and, subsequently, the tissues further back, by inter- and intracellular invasion [16]. Young shoots thus infected turn brown and die; older shoots which have escaped infection may be entered by the fungus from an infected lateral bud, and the disease may spread internally from infected to healthy shoots.

Black scurf of potato is frequently associated with cold, wet weather during early spring; plants partially infected make good recovery during warm, dry periods in late spring and early summer. Planting in cold soil infected with the fungus has been known to reduce the crop by a half [19], whereas in warmer soils in irrigated areas the disease is seldom a limiting factor [25]. Not only are serious losses incurred in early sown crops but those grown at high altitudes and in mountain valleys where soil temperatures are low also suffer severely. The general indication is that the prevalence of comparatively low soil temperatures *during the first few weeks after planting* determines the greatest amount of damage to the crop [19, 20]. But the temperature factor cannot be considered apart from that of the moisture content of the soil. While it has generally been observed that the disease is more severe when soil is dry and cool during early growth than when warm and moist, certain strains of the fungus have been found to be more pathogenic in dry soil than other strains which make poor growth in the same medium.

It is very probable, therefore, that there are other factors besides temperature and moisture-content of the soil which influence this disease, but whether they affect the host or the relative virulence of the parasite has not been determined [24].

It is obvious that seed tubers bearing sclerotia should be rejected for planting; and since the young sprouts are so liable to attack, the danger of early disease is greatly reduced if only sprouted tubers with sturdy, well-developed shoots are used, and to induce quick emergence shallow planting is advised [16a, 26a]. The treatment of seed tubers with disinfectant appears to be of doubtful value [5, 26]. Mercuric chloride, formalin, and potassium permanganate are recommended for tuber-steeping; a 5-minutes' treatment in cold, acidulated mercuric chloride, 1 part each of chloride and commercial hydrochloric acid, in 500 parts of water, destroys the majority of sclerotia on the tubers but large sclerotia need a longer time than small ones [4, 13, 22]. Formaldehyde treatment, 1 in 120, at a temperature between 122° and 124° F. for 4 minutes is effective; after steeping, the tubers are allowed to dry [10]. One per cent. solution of permanganate is also effective as a steeping agent [17]. The disease is best controlled by crop rotation, but it is important to know the range of host plants susceptible to the particular strain of the pathogen in the area [11]. Having in mind the longevity of the sclerotia, rotations of from 4 to 6 years are advisable. In New Zealand rotations of 2 or 3 years with cereals, brassicas, and legumes have proved effective, and laying the area affected down to grass for 4 years practically eliminated the disease [6]. The use of rye as a green manure in a 2-years' rotation with oats is also recommended [11].

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Black Dot of Potato, *Colletotrichum atramentarium* (Berk. & Br.) Taubenh.

This disease usually attacks potato plants from midsummer to autumn, during comparatively dry seasons. It is much more common in warmer latitudes than in countries of temperate climate like Britain, though appreciable losses may be experienced in this country under dry, warm conditions. The disease is known in many parts of Europe, in the United States and Canada, South Africa, Brazil, and Australia [7].

'Black dot' is caused by *Colletotrichum atramentarium* [2, 11] (Fungi Imperfecti), and the same organism causes a similar disease of the tomato plant [3, 4, 5, 8, 12, 13].

Affected plants usually exhibit much yellowing and drying of the foliage, and if the drying is relatively slow a number of axillary buds become swollen, and in certain varieties of potatoes aerial tubers may be developed. If infection is early, the plants remain small, especially under dry conditions. The stems are commonly affected towards the base, even down to the parent tuber, and there is considerable destruction of the stem tissues. The greatest amount of tissue injury is in the cortex, which may peel off from the dried stem in some quantity, and the vascular cylinder may also become involved, a delicate amethyst colour pervading the entire vascular system of the rotted stem. A characteristic feature of the disease is the development of numerous tiny black sclerotia embedded in the moribund tissues on both sides of the vascular cylinder (Figs. 15A, 262). They may also be formed on the inner side of the cortex, which by this time is more or less loosened and easily removable from the stem.

The stolons are attacked even more severely than the stems and become infected at any stage in the development of young tubers. Not uncommonly a stolon may be completely severed by the disease, so that young tubers are left stranded, and at digging time may be seen to carry wisps of dried, rotted stolon, which may often be found to carry small sclerotia. Older tubers usually bear sclerotia on the side uppermost in the soil, and these tiny bodies may vary in number from comparatively few to several hundred on a single tuber. Sclerotia may also be found on tubers still attached to the stolons, though the latter may to all appearances be quite free from infection. The roots are also liable to be attacked at all stages of growth, and, like the stem, suffer decortication. Infected plants pulled up will often show only stringy remains of the roots on which bits of cortex carrying the characteristic sclerotia are again found [7, 10].



FIG. 262.—Black dot of potato (*Colletotrichum atramentarium*). The sclerotia on decayed stems. (*A*, photo, Adv. Lft. 296, by permission of Minis. Agric. *B*, *C*, photos by Foister & Noble)



FIG. 263. — *Colletotrichum atramentarium.* The spores (×685) (after Duke, *Trans. Brit. Myc. Soc.*)

The sclerotia are found on practically all parts of the plant near, or in, the soil, on the lower parts of the haulms, or within the pith cavity of the stems, on the roots and on the stolons. They are small, round, black bodies, varying from 100 μ to 0·5 mm. in diameter, and may or may not be furnished with stiff bristles or setae which vary in length from 80 to 350 μ ; as many as 70 setae may be found on a single sclerotium. The sclerotia are rich in oil, and in germination produce a globular pink mass of spores on the surface. In culture (at 21° C.) the fungus produces a white, silky mycelium which, according to the type of medium, produces an amethystine or yellowish colour in the medium, a portent of sporulation. Later, the mycelium disappears and the culture passes over to the formation of the characteristic sclerotia which develop in concentric rings, and often in such great profusion as to cover the medium with a dark crust. When spores are developed in culture (Fig. 263) they may either be formed singly at the ends of short hyphae or in a close layer from a number of conidiophores ; the latter are from 10 to 30 μ in length, occasionally branched, and septated ; the spores are oval, continuous, slightly curved, hyaline singly, pink in the mass, and measure from 17·5 to 22 by 3 to 7·5 μ ; they are 1- to 3-guttulate. Saltations are frequent [6, 7, 9].

The fungus survives from season to season in the form of sclerotia in host debris, and may thus pass into the soil in which they are known to live for more than a year [7]. The disease may also start anew by the planting of seed tubers bearing sclerotia. The first infections are probably contracted by the roots whence the fungus passes into all the other parts, below soil-level ; the mycelium has even been found to pass from the vascular cylinder of the stem into the leaves [7].

Much can be done to avoid this disease by preventing the return of sclerotia into the soil, and all infected haulms should be destroyed by burning. Crop rotation should also be observed, to starve the sclerotia out of the soil.

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Dry Rot of Potato, *Fusarium caeruleum* (Lib.) Sacc.

Fusarium dry rot of potatoes, in Britain, attacks the tubers only in storage. In America, certain species of *Fusarium* also cause potato rot by direct penetration during the growth of the tubers on the stolons [9], but this mode of attack has not been observed in this country. It is true that contamination with the fungus

occurs while the tubers are in the ground, but the disease does not develop until some time after lifting [1a].

Dry rot tends to increase as the tubers become more mature [5, 10]; it is most serious on seed tubers of early varieties, and seems to worsen from December onwards; but in some years it may occur, as in 1934 and 1935 in England on second early and maincrop varieties, as well on the early kinds; in 1936 it was mostly on early varieties. The general impression is that dry rot is more in evidence following a dry season [3a]. The rot may start at any point on the tuber, usually from a wound, and if it appears at numerous distinct spots, which later become joined together, the probability is that infection originated in a number of the lenticels [5]. At such infected areas, the skin is slightly sunken and dark in colour. As the area spreads and the disease enters more and more into the flesh of the tuber, the overlying skin develops a series of wrinkles which are arranged concentrically around the seat of infection. With progressive rotting and loss of water, the tuber gradually shrinks, becoming dry and almost hard. White fungal pustules, changing to pink upon exposure to light, soon make their appearance on the wrinkled skin, and when cut, the decayed tuber will show one or more cavities filled with mycelium which is distinctly blue in some parts and the flesh around the cavities may also be similarly coloured (Fig. 264).



FIG. 264.—Dry rot of potato (*Fusarium caeruleum*). Below, two tubers showing the white mycelium on the wrinkled surface (photo by Foister & Noble). Top, tuber cut open, showing cavity filled with mycelium, with dark-coloured tissue around (photo by McKay)

While there are very numerous fungi [1a, 6] reported to be associated with decay of potato tubers, and these include numerous species of *Fusarium* [5, 7, 11], there is general agreement that the most virulent parasite causing dry rot of tubers is *Fusarium caeruleum* [4, 6], and strains of this organism appear to differ in morphological features pertaining to growth and production of spores, in pathogenicity, and in colour production in cultures.

The pustules or stromata on the tuber consist of closely interwoven hyphae which give rise to numerous branched conidiophores bearing sickle-shaped conidia which are blunted at the ends (Fig. 265). The conidia may be 1- to 4-celled, the latter predominating; they measure, for the non-septate kind, 14 by 3·7 μ; the 1-septate, 19 by 4·1 μ; the 2-septate, 21 by 4·3 μ; and for the 3-septate, 27 by 4·8 μ. Chlamydospores (Fig. 265 D, E) also occur occasionally on the pustules; they are spherical, and average 8 to 9 μ in diameter, smooth, thick-walled, bluish in colour, and are produced singly, in pairs, in chains, or in irregular groups [5]. In culture, on a wide range of media, conidia and chlamydospores develop in great abundance, forming slimy masses (pionnotes) on the

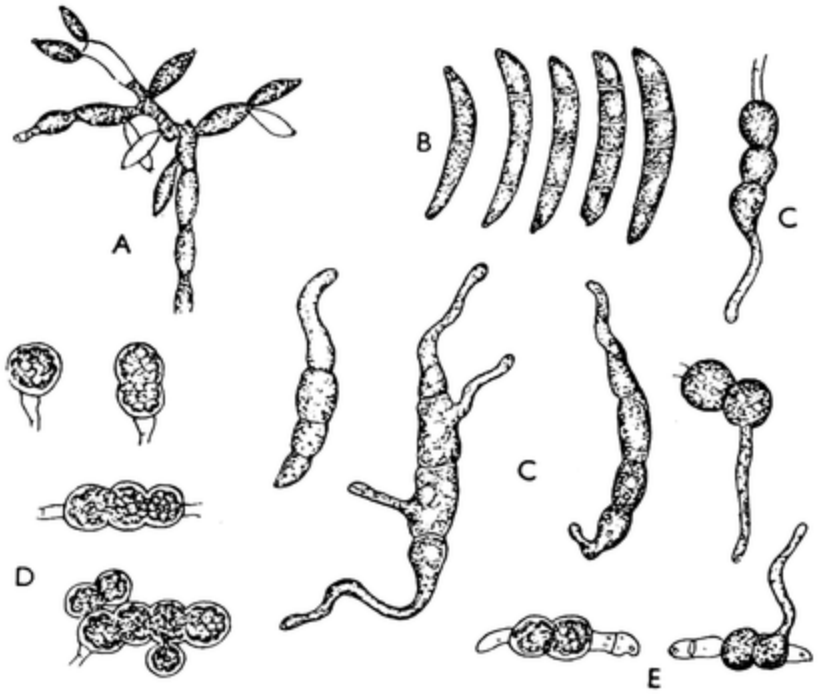


FIG. 265.—*Fusarium caeruleum*. *A*, branched conidiophore ; note, a ' collar ' at the end of each sterigma, except on the one on left which is producing a young conidium (× 330). *B*, conidia. *C*, germinating conidia. *D*, *E*, chlamydospores (after Pethybridge & Lafferty, *Sci. Proc. Roy. Dub. Soc.*)

surface of the substrate ; on rich media, at low temperatures, growth is purely mycelial ; no sporodochia are formed in artificial cultures on agar [10]. The colour of the spores and staining of the medium apparently depend on various factors, such as length of exposure to light, reaction of the medium, high carbon-nitrogen ratio, and degree of aeration ; the spores remain colourless when submerged, as in liquid cultures [2] ; in the dark the conidia are greenish blue ; in acid media the colour is wine red, in alkaline media, deep blue, and the fungus in its growth on acid media changes the colour from red to blue [5]. The maximum acidity tolerated by the fungus is near pH_3, and growth is still possible at $pH_{10\cdot5}$. It is noteworthy that the reaction of the expressed sap of healthy tubers is acid while that of the rotted tubers is alkaline.

F. caeruleum has a high capacity for producing chlamydospores, especially under conditions of reduced aeration [3]. They are often formed in abundance when sporulation is scanty [3, 9]. One or more cells of a conidium may become thicker-walled than the other cells, to form a chlamydospore, and even when conidia germinate in the ordinary way, the tip of the germ-tube often expands into a swelling which becomes thick-walled before its separation as a chlamydospore.

The fungus may be carried in field soils and in soil on the dug tubers, on contaminated knives used in seed-cutting, seed-boxes, etc. [1a, 2, 5, 8]. The disease spreads from tuber to tuber in storage, mainly through wounds and, probably under unusually moist conditions, through the ' eyes ' and lenticels as well. It is recorded that healthy tubers became infected after being steeped in a spore suspension, and infection has also been observed to start at ' powdery scab ' (*Spongospora subterranea*) lesions [1], and on unsprouted ' eyes ' which were apparently quite free from any injury [5]. The mycelium in the tuber is intercellular in those parts where it is advancing into the healthy tissue, but in the rotted, brown parts it occurs both between and within the dead cells ; the starch contents are not attacked.

F. caeruleum attacks potatoes in storage over a wide range of temperature. It is equally active during low night temperatures of 0° to 6° C. as it is during a day temperature of 25° C., but is inhibited at 30° C. The optimum temperature for growth of the fungus lies between 15° and 25° C., the minimum and maximum

being slightly below 5° and 30° C. respectively [3].

Early infections are greatly encouraged by high atmospheric humidities, though a 50 per cent. saturation is often sufficient [5]. Once infection becomes established, however, progress of the disease is practically independent of the amount of moisture in the air. But there is little doubt that rapid evaporation taking place at the surface of a wound, by encouraging the formation of callus, helps the wound to heal quickly and under such conditions infection may be checked.

The varieties Eclipse, Windsor Castle, and Epicure are usually less susceptible than Sharpe's Express, Ninety Fold, Early Puritan, May Queen, Duke of York, Arran Pilot, Catriona, and Midlothian Early, while main-crop varieties are, in general, less subject to dry rot than the early varieties [5]. Doon Star is most susceptible, and Majestic is frequently attacked [1, 3a].

Since dry rot is contracted mainly through wounds, care should be taken at riddling and handling of the tubers, to avoid breaking the skin [1]. If the mechanical digger is employed it should be manipulated so as to throw up sufficient soil to prevent bruising the tubers. To avoid ' sweating ' and premature sprouting the lifted tubers should be placed in shallow boxes in a cool store adequately lighted and ventilated. Knives, old sprouting boxes, and baskets used for carrying tubers, should be thoroughly disinfected before use. During storage, tubers should be examined periodically for symptoms of dry rot and affected ones removed or boiled if fed to stock.

Good control over the disease may be obtained by the use of an organo-mercuric dip, immediately after lifting and the tubers should be thoroughly dried after dipping, otherwise soft rots are liable to set in [2]. Since contaminated soil is the chief source of infection, washing the tubers greatly reduces the risk of disease [8a]. Dipping in 1 per cent. solution of formalin for $\frac{1}{4}$ to $\frac{1}{2}$ minute also gives effective control [1a].

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Silver Scurf of Potato, *Spondylocladium atrovirens* Harz.

This disease of potato tubers has been known in Europe since 1871 [5]. Its appearance in the United States was considerably later, in 1908 [3], and in 1914 was reported in Australia [1]; it is now widely distributed in most potato-growing areas [4, 6, 7, 8]. It attacks no other host.

Silver scurf disease, confined to the tubers, is mainly superficial, and no rotting

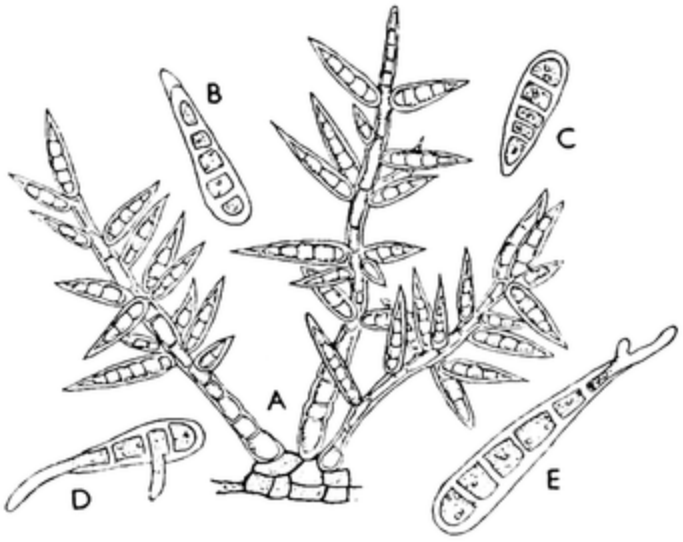


FIG. 266.—Silver scurf of potato (*Spondylocladium atrovirens*). *A*, conidiophores and conidia from tuber. *B*, *C*, conidia. *D*, *E*, germinating conidia (× approx. 380) (after Burke, *Cornell Bulletin* 692)

of the tissues follows upon infection. It attacks tubers which are mature or nearly mature. At harvest time the trouble is barely evident, but there is considerable disfigurement of the tubers under too moist and too warm conditions in storage. The early symptoms of disease, if looked for soon after digging, are very difficult to detect, but occur chiefly towards the stolon end of the tuber as tiny, buff-coloured spots with a finely fimbriated margin. The spots are seen to better advantage if the affected part is wetted, when the whole area presents a glazed or silvery appearance, producing a striking effect on coloured varieties of tubers where the pigment is destroyed over the affected area. Usually only a part of the surface is affected though a fair portion of the tuber may often be covered [2]. The effect of the disease is either to cause a sloughing-off of patches of the cork layers, forming ' scurf ', or if the affected spots remain intact the overlying cork layers are raised up so that air pockets are formed, hence the silvery appearance of the tubers when wetted. In any case, the ultimate effect is a gradual drying of the tubers, which soon present a shrivelled and wrinkled appearance in storage. The grey, ' silvered ' areas soon become covered with an olivaceous mass of conidia and in severe infection the whole surface of the tuber may be rendered dull black and sooty.

Silver scurf is caused by *Spondylocladium atrovirens* (Fungi Imperfecti) [5]. Its mycelium in artificial culture or on moist tubers is slow-growing ; the optimum temperature is 24° C., no growth occurring at extreme temperatures of 3° and 33° C. ; and high humidities of 95·6 per cent. or more are essential. The mycelium in culture or in the tuber lesions is hyaline at first, then brown, the hyphae being straight and septated ; later, the mycelium consists of round to barrel-shaped cells of a very dark colour, and on the tubers similar globular cells become massed together to form tiny bodies resembling microsclerotia. Conidia appear in the cultures in about 10 to 12 days, forming a greyish-black mass over the medium. The dark-olive or brown multiseptate conidiophores are very variable in length, from 150 to 375 μ, and rounded at the base ; the conidia are borne at the apex, or at the distal end of the intermediate cells, in whorls, down to about half the length of the conidiophore (Fig. 266). The elongate-ovoid conidia attached at the broader end are highly variable in dimensions, ranging from 35 to 61 by 7·5 to 12 μ, the majority being from 35 to 40 by 8 μ, and 5- to 7-septate [2, 7] ; they germinate from the narrow end usually, but any other cell may produce germ-tubes as well [2]. Sporulation occurs over a wide range of temperature from 6° to 27° C. [2].

Primary infections appear to take place at the stolon end of the tuber, arising probably from contact with the infected mother-tuber, and not from infection

travelling along the stolon. Early infections may also start at any other point on the tuber, probably through lenticels. These early infections make little progress until the haulms are dead and the tubers more or less matured in the soil before they are lifted. The exact method of penetration is not known but the mycelium gathers chiefly below the cork layers, and conidia appear in 3 or 4 weeks after invasion. Infection and creation of new lesions continue during the storage period under conditions of high humidity and warm temperature.

The fungus probably thrives in the soil as a saprophyte [8], especially in humus soils [2], but is probably carried from season to season with the ' seed ', particularly by infected tubers left in the soil from a previous crop.

The disease may be held in check by keeping the tubers stored at as low a temperature as possible ; no fresh lesions were seen to occur nor the disease to spread at temperatures below 37° F. and at humidities below 90 per cent. [2]. Steeping the tubers as a possible seed treatment against the disease is also effective ; dipping in a 1 per cent. solution of malachite green for 5 minutes, or a mixture of 0·5 gm. mercuric chloride and 0·5 gm. mercuric cyanide in a litre of water, applied for the same period, prevented spore formation without harming the tubers.

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Skin Spot of Potato, *Oospora pustulans* Owen & Wakef.

This tuber disease of potatoes causes superficial blemishes on the skin but is much more serious in its effects on the ' eyes ' as, by its destruction of the buds, tubers may be rendered ' blind ' and therefore unfit for ' seed ' purposes. In Britain, losses of 20 to 30 per cent. have been noted in stocks of seed tubers, due to this disease [4].

Skin-spot disease was first recognised in Britain in 1904, on potatoes from Lancashire [3]. The trouble is difficult to detect at lifting time, and it is essentially a disease which develops in storage and clamps. It does not affect all varieties of potatoes in the same way. In Scotland it was observed that, while the variety Golden Wonder remained free from the trouble, others such as Kerr's Pink, Arran Chief, and Arran Banner were frequently badly affected with a general spotting of the tubers but without damage to the sprouting eyes, while crops of Ally, Majestic, and King Edward suffered from blindness as well [1].

On coarse-skinned potatoes, such as Arran Chief, the spots are like tiny pimples over which the skin remains stretched, unbroken and shiny and of the same colour as the rest of the tuber when dry, but a darker brown when wetted. On a smooth-skinned variety, such as King Edward, the spots are round and sunken but with a slightly raised centre, brown or black in colour. Spots of an intermediate character may also occur but not usually on one and the same tuber [7] ; the pimple form has



FIG. 267.—Skin spot of potato (*Oospora pustulans*). Affected tuber from which the skin has been lightly peeled, showing the dark, dead tissue beneath each spot (photo, Adv. Lft. 279, by permission of Minis. Agric.)

been observed on King Edward tubers to become depressed at the centre as the spots get older [5]. In any case, the spots may occur singly here and there, or in groups, and may join together so that large areas of a tuber are frequently covered with them, though one side is often more spotted than the other. But there is little invasion of the disease into the flesh and the tuber remains firm throughout, without rotting (Fig. 267).

The disease is caused by *Oospora putsulans*, a member of the Fungi Imperfecti (*Hyphomycetes*). In the diseased spots the mycelium consists of very narrow, septated filaments, from 2 to 4 μ wide, hyaline or pale brown in colour; it penetrates between the cells of the parenchyma to a depth of some 12 to 15 layers of cells from which the starch contents have entirely disappeared (Fig. 268). Some of the affected host cells are browned, thick-walled, and crushed; others remain hyaline and thin-walled, and the lesion finally becomes delimited from the deeper tissue by a broad concave layer of cork [5]. The first conidiophores to break through are long and decumbent, but the majority are short and erect, cutting off, by acropetal budding, long chains of cylindrical or oblong, hyaline conidia which measure from 6 to 12 by 2 to 2·5 μ. The organism is easily cultured on a wide range of vegetable media, at an optimum temperature of 12° C., growth being practically absent at 0° and 24° C. [7].

The disease is not usually evident on the tubers in the autumn or early winter, but under cool, temperate conditions in the spring it develops in storage and clamps and many of the tubers fail to sprout [4]. The trouble is believed to start from the soil and to attack the tubers during growth, but in what form the organism exists in the soil, its method of attack, and the exact time of infection, are not clearly known. It has been known to break out in land which had been in grass for at least twenty years [4]; and seed tubers planted in contaminated soil may or may not produce a diseased crop according to



FIG. 268.—*Oospora pustulans*. Vertical section through a skin-spot pustule on the potato. Note the dark, slender hyphae and the minute globose conidia (after Millard)

circumstances which have so far not been determined [7]. It is probable that the tubers are attacked before digging and that infection remains latent, possibly in the lenticels, for some time before undergoing further development, so that there is apparently a lag between infection and development. This hypothesis receives support from the fact that, in a method of tuber disinfection aimed at checking the growth of the fungus in the skin, the treatment afforded good control provided it was applied immediately the tubers were lifted, but had little effect if delayed [4].

When the 'eyes' are attacked at the delicate tissues of the bud scales, the latter become spotted like the rest of the tuber and the fungus penetrates so deeply that infected buds are killed, but other buds at the same eye may escape and produce sprouts, and buds may be destroyed on which there was no previous evidence of spots, infection having probably entered from the tissue around the base of the eye, for the fungus penetrates into this region as well as into the buds [7].

As the disease is of importance on seed tubers, these should be selected as soon as the crop has been lifted and boxed immediately for sprouting in a dry, well-ventilated store. Tubers which fail to sprout or produce only weak growth should not be used for planting. As above stated, a certain degree of control has been obtained by treating seed tubers, immediately upon lifting, with a fungicide, the process consisting of an 'instantaneous dip' of $\frac{1}{2}$ to 1 minute in a solution of organic mercury [4]. But obviously the best precaution against this disease is to plant tubers with clean, vigorous sprouts.

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Potato Leaf Roll

As already mentioned in Chapter VIII, virus diseases are of great economic importance and account for greater losses than any other of the numerous diseases which affect potato crops. It is a common experience that potatoes lose considerably in cropping power when seed for planting is saved from year to year from the previous crop. This falling-off in productivity, accompanied in many cases by more or less well-marked symptoms of disease in the green shoot, is now generally accepted as being due to the presence of one or more viruses in the plants, and not, as was once believed, to a natural 'running out' of the plants following upon prolonged vegetative propagation. Moreover, it has long been noticed that this deterioration occurred much more rapidly in some areas than in others, and that a change of seed was beneficial. It is now almost a routine practice for growers to obtain their seed potatoes from Scotland and Ireland, and it is estimated that over 300,000 tons of seed potatoes from these areas are imported into England

every year [58]; another estimate is that English growers spend on an average about £700,000 every year on fresh seed to replace their own stocks, which become exhausted within a comparatively short period of two or three years through the action of one or more virus diseases [14].

Leaf roll is the most important virus disease causing degeneration of potatoes, and has long been recognised as a serious problem in Europe, Canada, and the United States [2, 66, 75]. While other virus troubles, such as 'severe mosaic' (p. 548) may account for greater reduction in yield, leaf roll occurs more frequently and losses due to it are heavier [14, 58]. Fortunately this disease is not common in the best seed-growing areas in Scotland and Ireland, where the climate is too cool and moist for insect vectors to thrive, and there are certain parts of Wales where the crop can be cultivated with a high degree of freedom from this disorder [79-81].

This disease is not equally virulent on all potato varieties. Thus, in crops of Up-to-Date and Great Scot losses may be from 40 to 50 per cent. whereas they may be as high as 80 to 90 per cent. in King Edward and May Queen. The recognition of leaf-roll disease in the growing crop is not always an easy matter, for the feature of leaf curling may arise from various causes, some physiological, due perhaps to a derangement of the water supply to the roots, or to mineral deficiency, and some to fungal diseases, such as that due to *Corticium solani* (Fig. 259). It is not usual for healthy plants, naturally infected during the first season, to show the characteristic symptoms of a curling of the leaves, but sometimes this feature may be found in the *upper*, youngest leaves of a plant, a condition which is then described as *primary* leaf roll. *Secondary* leaf roll, when the *lower* leaves show marginal curling, is usually manifest in the second and subsequent seasons following primary infection [1]. But the younger the infected plant and the more rapid its growth, the more quickly secondary symptoms appear, and the two stages frequently occur together on the same plant [49]. In some varieties, however, which appear to be more tolerant of the disease than others, the uppermost leaves may show no signs of leaf roll, but a rolling of the leaves at the base of the plant is a fairly safe diagnosis of the trouble, and with the continued growth of the shoot the entire foliage develops a rolled appearance (Fig. 269).

Though the complete symptom-picture of leaf roll may vary according to the particular variety of tuber, there are features which are fairly common to all plants affected with leaf roll. In addition to a rolling of the lamina, affected leaves are thicker, the plants are stunted and present an unnatural, stiff appearance, the leaves being harsh to the touch; the plants have a leathery texture and rattle when one walks between the drills. Discoloration effects on the leaves are highly variable and liable to change even in individual plants. More exact descriptions of leaf roll must, of necessity, be given on particular varieties. Thus, in Arran Victory after an interval of some 30 days following infection, the primary symptoms are manifested by a general pallor of the upper and youngest leaves, the actual rolling following later, starting from the base of the lamina, where there is also a brownish-black discoloration. The secondary stage quickly follows, in which the lower leaves show a marked interveinal pallor, as well as a leatheriness and a harshness to the touch. These features are again followed by leaf rolling and discoloration, and the whole plant becomes stunted and may develop a purplish



FIG. 269.—Leaf roll of potato. *A*, in the variety British Queen; medium effect. *B*, single leaf of same. *C*, in the variety President, showing severe stunting, typical of the disease in erect varieties (photos by McKay)

hue all over. The presence of aerial tubers is often a feature of leaf-roll disease in Arran Victory [62]. Again, the variety President (Fig. 269 c) is very susceptible to this disease, and while a curling of the leaves is not a striking symptom in this variety, it shows much stunting and a decided unnatural stiffness in its branches. The young leaves usually become yellow on top and pinkish below and eventually become highly necrotic. On account of its high degree of susceptibility, this variety is rapidly going out of cultivation. In the variety King Edward, in addition to a general pallor of the young leaves and a slight rolling of the leaves at the base, a pink colour develops on both sides of the leaves. An interveinal pallor is also shown by Arran Chief and Kerr's Pink and the leaves early develop a pink colour. In the variety Great Scot, leaf rolling is not a prominent feature, but here, again, there is the same pallor accompanied by a stiffness of the lower leaves [60, 62].

Once within the plant, the virus travels to all its parts, including the new tubers. On the latter, however, there are no visible symptoms of virus disease, so that, externally, they are indistinguishable from virus-free tubers. Virus-laden tubers often remain hard and unrotted in the ground, but this feature is not always a clue to virus trouble. Tubers infected with the virus when planted give rise to shoots in which leaf rolling is a characteristic symptom and such tubers offer a ready means of perpetuating the disease in the progeny, so that successive crops ' run out ' within a comparatively short time.

The infective principle of leaf-roll disease is a single virus, and to distinguish it from others it is designated *Solanum virus 14* (Appel & Quanjer). *Potato phloem necrosis virus* (Quanjer 1913), *Potato leaf roll virus* (Appel 1911), *Potato leaf curl virus* (Pethybridge 1911), *Potato virus 1* (J. Johnson), are synonyms [62]. The virus is not sap-transmissible and its natural spread in the field is through the agency of insects, the principal vector being the peach aphis *Myzus persicae*. A peculiar biological relation seems to exist between the virus of leaf roll and the insect responsible for its transmission. A delay of some 48 to 54 hours within the body of the vector appears to be necessary before the virus becomes sufficiently potent to bring about infection [60]. The infective power is, moreover, retained within the insect even after it has fed on plants not susceptible to this disease, but the insect does not transmit this power to its progeny. The new brood can become infective only after feeding upon plants affected with leaf roll. A close relationship thus exists between virus and vector [33, 34, 55, 60].

After feeding on the sprouts or leaves of a plant affected with leaf roll, the insect spreads infection by puncturing the sprouts or leaves of plants in the close vicinity. Although it is easier to infect young plants, the age of the plant to be infected, or of the plant from which the virus was obtained, is of little significance. The prevalence of the vector *Myzus persicae* is of primary importance in the spread of leaf-roll virus in the field. It is true that the disease may be transmitted artificially by grafting a shoot from an infected plant on a healthy one, but artificial transmission by sap inoculation, so frequently successful with other viruses, is not effective, and the insects are entirely responsible for the spread of leaf-roll disease. Experiments conducted in numerous areas amply confirm the relationship between the incidence of leaf roll in the field and the times of appearance of the aphides [49]. According to observations in eastern Ireland [52], the disease in that locality spreads

in the direction of the prevailing winds, infection occurring during a six-week period between May and early July, this period being found to coincide with the aphis season. Years of least spread of the disease had unusually wet weather in June, a condition which appeared to limit the increase of aphides during the critical period. But a high degree of infection was induced by normal rainfall and temperature in June, which favoured rapid plant growth while at the same time increasing the aphid population (52).

The insect vector *M. persicae* may collect the virus not only from infected potato fields, but from certain weeds growing in the vicinity of the crops, especially from cabbages on which the insects hibernate. During early growth of the potato crop, therefore, the presence of the insects on sprouts of infected tubers or cabbage plants in the vicinity, is no doubt, the limiting factor, and the health of the potato crop depends on the scarcity of the hosts on which the aphides can tide the winter and on the remoteness from potato fields (52).

The pathological effects of the virus on the tissues have been described for certain varieties of potatoes. These effects are confined to the vascular strands and are seen in the form of a '*phloem necrosis*' which, however, appears to be confined to the primary phloem, and may occur both in the outer group and in the inner group of this tissue in the bi-collateral bundles of the potato; sometimes the necrosis affects only one group, outer or inner, and may be severe in one and slight in the other. The condition may spread both up and down the stem, and always precedes the symptoms of the curling of the leaves. In severe leaf roll, necrosis can be found throughout the main stem almost to the growing point, and may spread into all the branches and into the petioles of all but the youngest leaves; its constant presence in the stem of the plant, just at or below soil level, will help in the diagnosis of leaf roll, even before curling has started, however lightly the plant may be affected by this virus (75). It has been observed, however, that certain varieties of potatoes, e.g. Katahdin, Chippewa, and Sebago, very seldom, if ever, exhibit phloem necrosis, and the phenomenon apparently depends on various factors, such as the relative susceptibility of the variety to the disease, the severity of the attack in a particular plant under examination, and the time of season at which the examination is made, and possibly other undetermined factors (66). A pathological condition within the tuber itself, also confined to the phloem of the vascular strands, has been observed in certain potato varieties and referred to as '*net necrosis*'.

A characteristic feature of plants suffering from leaf roll is the abnormal retention of starch in the leaves, the leaves of healthy plants growing under the same conditions showing little or no starch. The familiar iodine test, even without a previous clearing of the lamina from chlorophyll, will show the difference very strikingly (Fig. 270). Rolling of the leaves follows rapidly after the



FIG. 270.—Leaf roll of potato. *A*, starch test, to show retention of starch in the leaf. *B*, healthy leaf (photo by McKay)

commencement of starch accumulation and apparently the actual leaf curling is the direct effect of the starch collecting more in the spongy than in the palisade mesophyll, and since the former, looser-knitted tissue is free to expand in all directions except on that side where it abuts on the better-consolidated palisade tissue, the result is, therefore, a downward and lateral extension causing an upward rolling of the leaflets [48].

While in normal potato plants, as in all healthy green plants, the translocation of starch from the leaves takes place during the night, the leaves being practically free from starch by the morning, in potato plants affected with leaf roll the starch is retained in the leaves, and there is little, if any, translocation into the tubers. It is difficult to account for this check to translocation. Since a necrosis of the phloem has been observed to take place comparatively early following upon infection (Fig. 175), it is asserted by some that this pathological condition of the phloem may check the flow of carbohydrates throughout the plant, while others state that the inhibitory effect begins in the affected leaf itself, and that it gradually extends into the stem, though it is possible that phloem necrosis, once it has set in, may add to the difficulty of translocation [48]. In the healthy leaf it appears that the accumulated starch first begins to disappear, during evacuation, at or near the leaf tips, and then gradually vanishes from the lower portions. In the diseased leaf, however, the reverse takes place, the starch first disappearing from the bases of the leaflets and proceeding for a lesser or greater distance towards the apex [48]. While the translocated carbohydrate in the healthy leaf is believed to be sucrose, in plants affected with leaf roll it is deemed to be hexose, and the suggestion is made that the reduced photosynthesis results from the accumulation of starch, the reaction in rolled leaves being the conversion of starch to hexose, hexose to sucrose, and sucrose back to starch [8]. Another explanation of the problem is that both necrosis of the phloem and the accumulation of starch result from a derangement of protein metabolism. As already stated, virus-laden seed tubers are often found in the ground hard and undecomposed, and while such tubers are said to germinate later than healthy ones, the proteins pass into the shoots much sooner than the carbohydrates. This complete migration of the proteins into the shoots apparently puts an end to diastatic activity in the tubers, so that the starch remains unaffected and the flow of sugars to the developing shoots is arrested [59].

For the eradication of potato leaf roll, the selection of healthy seed is obviously of primary importance. To this end seed-growing areas should be situated where the aphides concerned are least likely to breed. *Myzus persicae* is usually very scarce in wet, windy districts, so that in such localities the migration of the winged forms is impeded ; it has been observed that heavy rain may relieve a potato crop from infestation for a considerable time, or for a whole season, if the weather remains cold and damp. Seed crops should not be established where peach or apricot trees are near, on which the aphides may tide the winter, and the same applies to proximity to market gardens where winter cabbages and weeds serve as temporary hosts to the insects [32, 35, 73].

The annual inspection of potato crops has done a great deal to ensure that seed potatoes should be free from virus troubles, and a standard of certification is now demanded of growers. In 1937 this standard allowed not more than 0·5 per cent.

of leaf roll (and ' severe mosaic ', p. 548), and not over 1 per cent. of all readily visible virus diseases. The highest grade Stock Seed Certificate, in Britain, requires not more than 0·25 per cent. of visible virus diseases, with fewer than four plants per acre (about 0·025 per cent.) showing leaf roll or severe mosaic [58]. Even certification of seed in the field is not very reliable, for virus troubles are often masked and late-season infections cannot always be detected, and a recognised technique of advance-testing of tubers of seed stock in the greenhouse gives a good index of the virus disease contents [16].

Potato breeding for virus resistance has shown that some form of resistance exists among various cultivated varieties of potatoes. Moreover, each form appears to be controlled by heritable factors, and there are possibilities that more useful forms of resistance in ' wild ' material may be discovered, and it is hoped that good control may result from the introduction of new varieties which may show a higher degree of resistance than the varieties now in cultivation [15, 15a, 39, 46, 58]. In Eire, recent observations have shown the varieties Shamrock and Skerry Champion to be highly resistant under field conditions, the latter variety showing almost complete immunity [46a].

Potato Mottle, Mild, or Simple Potato Mosaic

This disease, caused by *Solanum virus 1*, better known as *Potato virus X*, is widespread in all potato-growing areas everywhere. The great majority of commercial varieties are apparently never free from it, and it is often found in potato plants which have already been tested in the experimental glasshouses [58].

It is remarkable that most varieties of the host either carry the virus with no visible symptoms of disease, or when infected, show only an interveinal mottle with slightly noticeable dwarfing or deformation of the leaves (Fig. 271 A). The varieties Arran Crest, Epicure, King Edward, and others, however, are very susceptible to artificial infection, develop *top necrosis* and die, and yet none of these varieties has shown the disease in the field, so that they serve as useful indicator hosts for this virus [28, 62]. These varieties, experimentally infected, whilst not immune from this virus in the usual sense, do possess a peculiar type of immunity for they are so intensely susceptible when artificially infected that they are killed off. Presumably, under natural conditions the reaction of the plant at the spot at which infection might take place becomes at once so lethal that there would be no danger of the disease gaining access to other plants [73].

Though there are no visible symptoms on the great majority of potato varieties, yet the growing plants lack robustness and vigour and in many cases a reduction of 2 to 4 tons per acre may well be encountered in the crop [73]. It is recorded that 90 per cent. of potatoes grown in Australia are infected by this virus and the losses due to the disease caused by it are estimated to be about equal to the combined losses caused by all other virus diseases in the crops, estimated in value at about £350,000 every year [4].

The following synonyms are listed for the causal virus *Solanum virus 1* Orton [62]: *Potato virus X* (Smith 1931, 1939); *Potato common mosaic virus*



FIG. 271.—*A*, mosaic (virus X) of potato on leaf of variety President. *B*, aucuba mosaic of potato on leaf of variety Kerr's Pink. *C*, rugose, or severe mosaic (virus Y) of potato on the variety Arran Crest, on left; healthy plant on right (photos by McKay)

(Quanjer 1923); *Potato mosaic B virus* (Fernow 1925); *Potato mottle virus* (J. Johnson 1925); *Latent potato virus* (Schultz); *Potato virus X strain S* (Salaman 1930); *Potato simple mosaic virus* (Murphy 1932); *Potato mild mosaic virus* (Samuel 1943).

The virus is known to possess a number of strains which differ in the severity of the symptoms produced. Some of the more severe types may exist along with milder ones, and in extreme cases a limit to the multiplication of a severe strain is imposed by the damage it does to the host tissues; in other cases an approach to an equilibrium may be obtained [3, 6, 7]. As many as six strains have been differentiated by inoculation of a wide range of plants [65, 70]. Together with *virus A*, *virus X* is responsible for the composite mosaic disease known as 'crinkle', described below.

Though the means of transmission of this virus still remain to be elucidated, the general opinion is that it spreads throughout the crop by leaf contact, mainly under the influence of wind [42, 43]. There is no evidence of its transmission through the agency of insects, and the risk of underground infection appears to be negligible [42]. That insects are likely to transmit the virus by visiting the flowers is not clear; observations in Scotland showed that of 14 potato varieties which flower sparingly or not at all, 10 were shown to contain the virus rather commonly, while the other 4 were always free from it; none of the freely flowering commercial varieties were, however, free from the virus [23].

The presence of the virus in the plant appears to reduce slightly the amount of carbohydrates in the infected leaves, and starch formation and hydrolysis are impeded. At all stages of growth, however, there is a greater content of nitrogen in the diseased than in healthy leaves, and it has been suggested that the disturbances in carbohydrate metabolism and the pathological conditions created, are direct manifestations of a disorganised nitrogen metabolism [26]. It is further suggested that the loss in yield due to this disease may be traced to the unavailability of the protein reserves, by virtue of their conversion into 'virus proteins' which the plant fails to reconvert into mobile substances for storage in the tubers. That some varieties of potatoes suffer greater loss in yield than others may, therefore, be due to their infection with more severe strains of the virus, which by their greater power to immobilise the proteins are enabled to multiply all the more to greater concentration in the plant than the milder strains [5].

Histopathological effects of the virus are sometimes evident in a necrosis of the phloem in the stem, and there is a thickening of the cell walls in the primary phloem, with a gummy deposit in the intercellular spaces; sometimes, necrosis may spread along the medullary rays into the wood [12, 62].

Investigations on the breeding of potatoes resistant to this disease are already showing considerable prospects of success. It has been established that field immunity from *virus X* is inherited as a Mendelian dominant [18, 24]. The breeding of varieties by selection and reassortment should therefore not be a difficult problem [28]. As there are usually no visible symptoms of this disease in the crop, control by roguing is rendered very difficult. The raising of high-quality seed from tested *virus X*-free tubers has already been carried out on a limited scale [22, 70], and all that remains is to multiply these on a large commercial scale with due

attention being given to the means of transmission, should any other method be found than that by contact of diseased with healthy plants [58].

Potato Rugose or Severe Mosaic

This virus disease, second to ' leaf roll ' in importance, is common in the south and east of England where it accounts for most of the degeneration of potatoes in those areas [14]. It appears to be rare in Scotland and Ireland, but has been noted in France and in the United States, where it is known as ' vein-banding ' [62].

It is not possible to give a general symptom-picture of this disease, for it varies with different varieties [68]. On Majestic, Arran Crest, and President for example, soon after infection, local lesions consisting of black necrotic spots appear on the under side of the leaves. Later, the uppermost leaves develop a wrinkled appearance, become yellow or mottled, and are usually the only leaves to remain on the plant, the rest having collapsed against the stem or left hanging by mere strands. Black necroses appear on the under side of the veins of those leaves situated about half-way up the stem, and the collapse follows after the infection has become systemic, when necrosis has spread along the veins into the petioles and finally into the stem itself on which brown longitudinal lesions become apparent, just below the still greenish epidermis. The leaves then collapse or fall off, from the lowermost upwards. This primary phase has often been called ' *leaf drop streak* '.

When the virus-laden tubers are planted, the symptoms on the growing shoots are strikingly different from those described above (primary) on the parent plants. The leaf symptoms are not now so evident, and the more obvious effect of the disease is the decided check to growth, the plants being dwarfed and brittle, with the leaves mottled, twisted, and closely bunched together, hence the name *rugose mosaic* for these symptoms of the second year (Fig. 271 c). On another variety, Arran Victory, the symptom picture is simpler than on Majestic and President, for there is only one type of infection, and this is not systemic. In the first and subsequent years of infection Arran Victory shows only a mild crinkle ; the leaves are slightly mottled and waved, but there is no leaf drop and little or no necrosis [14].

This virus had been variously named [62] : *Solanum virus 2* Orton ; *Potato virus Y* (Smith 1931) ; *Streak virus* (Orton 1920) ; *Leaf-drop streak virus* (Murphy 1921) ; *Acropetal necrosis virus* (Quanjer 1931) ; *Stipple-streak virus* (Atanasoff 1922) ; *Hy. II virus* (Hamilton 1932) ; *Vein-banding virus* (Valleau & Johnson 1930) ; *Potato severe mosaic virus* (Samuel 1943).

The virus, better known as *Potato virus Y*, is sap-transmissible and is spread through the agency of insects, namely the peach aphis *Myzus persicae* and *Aphis rhamni* [37, 45] ; a variant form of the virus has been identified [63]. The tobacco plant (*Nicotiana tabacum*) and the thorn-apple (*Datura stramonium*) serve as ' differential hosts '. On the former, the virus produces a clearing of the veins of the youngest leaves, followed by a banding of the veins of the older leaves, without necrosis ; on the latter, the virus has no effect, the immunity of this plant, therefore, being useful in eliminating *virus Y* from any complex of viruses in which it may occur. The tomato, henbane (*Hyoscyamus niger*), woody nightshade (*Solanum dulcamara*), black nightshade (*S. nigrum*) are also susceptible. It is interesting

to note that the early symptoms on the tomato in the form of vein clearing and mottling disappear with growth, and the adult plant shows no external symptoms of the presence of the virus [62]. Experiments at Cambridge have also shown that this virus can be carried in a masked form by turnip, cabbage, kale, brussels sprouts, red clover, garden peas, and the common bindweed. Its transmission to potatoes from the crucifers was effected both by grafting and through the agency of the insect vector *Myzus persicae*. The plants above named, especially the cabbage and the troublesome bindweed may, therefore, quite possibly serve to harbour the virus and give succour to the vectors over the winter [72]. It is said that the virus may be carried to a small extent in the true seeds of the potato berries [57].

Potato Crinkle

Crinkle disease of potatoes is caused by a mixture of two viruses, *Solanum virus 1* (*virus X*) and *Solanum virus 3* (*virus A*). Some varieties grown in the south-west of Scotland were found to fall into four groups in respect to infection by these viruses, singly or in combination : (*a*) varieties lethally necrotic to both, (*b*) lethally necrotic to A but not to X, (*c*) lethally necrotic to X but not to A, and (*d*) non-lethal to both [26].

Though the symptoms of crinkle disease vary according to the variety, affected plants, in general, are dwarfed and bushy with pale, puckered leaves which curve downwards (Fig. 272). A diffused, slightly yellowish spotting occurs in the leaves, which later changes to a rusty-brown discoloration before the plants die. The leaves are brittle and easily injured [47]. In those varieties which develop a necrotic reaction to the disease, the symptoms first appear on the terminal leaves as a yellow, blotchy mottle, and as the necrosis develops, spreads from leaves to petioles, and from the apex of the shoot to the main stem until the entire plant finally collapses [28].

As we are dealing here with a mixture of viruses (one, *virus X*, as stated in a previous section (p. 547), being not transmissible by insects, while the other, *virus A*, is so transmitted) the disease can only be brought about in those plants which are already infected with *virus X*, if *virus A* is brought to them through insect agency. Artificial infection, followed by typical symptoms of crinkle, may be effected from potato to potato by grafting, and sap inoculation will transmit only one of the component viruses, namely *virus X* [62].

In its physiological effects on the host plant this disease does not greatly disturb the metabolism of carbohydrates, and the translocation of sugars to the tubers is not seriously impeded, but there is a decided interference with nitrogen metabolism. It is suggested, therefore, that heavy losses in yield due to the incidence of crinkle may well be due to changes in the building-up of nitrogenous compounds [9, 11]. Experiments on the variety President showed that chlorotic parts in the leaves were thinner than the green parts, due to a shortening of the palisade cells as well as to a reduction in the size of the intercellular spaces [19].

The varieties Up-to-date, British Queen, Kerr's Pink, Arran Crest, Epicure, and Great Scot are intolerant of these combined viruses, while Champion, President, Arran Chief, Arran Victory, and Arran Banner are severely subject to crinkle disease [47, 62].

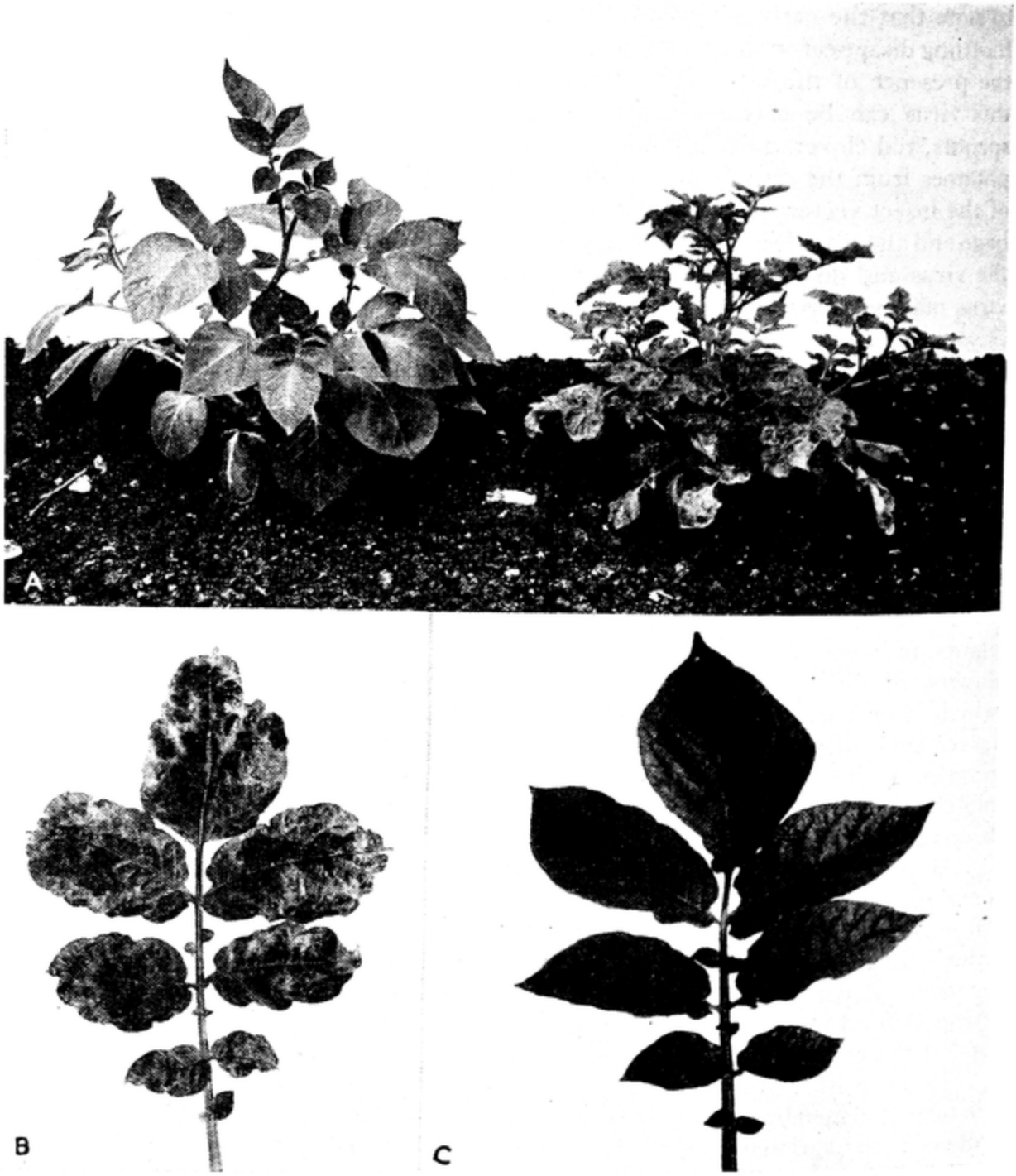


FIG. 272.—Potato crinkle. *A*, healthy specimen of variety President on left; diseased, on the right. *B*, a single leaf of the same, with crinkle. *C*, single leaf of the same, affected with virus '*A*', a constituent of the mixture causing crinkle (photos by McKay)

Potato Aucuba Mosaic

This virus disease of the potato is so named because of the strong resemblance of the affected leaves, with their yellow spots, to the foliage of the Japanese laurel

Aucuba japonica [54, 55]. The disease is common on many varieties in Great Britain and North America [62].

The characteristic symptom of aucuba mosaic is the bright-yellow and green variegation of the foliage (Fig. 271 B). This feature is clearly distinguishable from the type of mottling attendant on plants infected with other viruses, such as *virus X* or *virus A*, and always appears first on the lower leaves, which sometimes are the only leaves to be so affected. In the variety Irish Chieftain, however, the leaves become yellow all over, but whether this general discoloration, with its marked brilliancy, is due to the presence also of another virus (*virus A*) (Fig. 272 C) is not clearly known [21]. In addition to a variegation of the leaves, some affected varieties develop necrosis in the tubers, viz. President, Majestic, Champion, Great Scot, Dunbar Yeoman, and British Queen, while others, such as Early Regent, Epicure, Arran Crest, Arran Banner, and Arran Victory, show no tuber necrosis [62]. In the varieties affected, tuber necrosis accounts for the greater part of the reduction of yield though a general loss of vigour is also contributory.

The following are synonyms for the *Virus of aucuba mosaic* (Quanjer 1922): *Solanum virus 9* (Murphy & Quanjer), *Potato virus G* (Clinch, Loughnane & Murphy 1936), and *Non-infectious chlorosis virus* (Murphy).

The virus is transmissible by the aphis *Myzus persicae* and may also be conveyed by sap inoculation. Inoculation experiments, performed by rubbing infective sap with a ground glass spatula on the lower leaves of young plants of President in pots, resulted in the appearance of the typical symptoms of the mosaic in the foliage and tuber necrosis developed later. When tubers so affected were planted the next season, typical aucuba mosaic mottling duly appeared on the leaves. *Datura stramonium* the thorn-apple, and tobacco (var. white burley), when similarly inoculated showed none of these symptoms, but that the virus had been transmitted was shown by the development of the typical symptoms of aucuba mosaic, when the inoculated plants were grafted back on healthy President plants [21].

While the green areas of affected leaves show normal green chloroplasts with the inclusion of small starch grains, the yellow mottled areas when tested for starch in the early morning give a much deeper reaction than the remaining parts of the leaf. Even in older leaves, whether they were removed and tested in the morning or evening, starch was found to be present in the yellowed parts. The effect of the virus in the leaves, therefore, is clearly to impede the translocation of carbohydrate. The final effect on the green plastids is their degeneration and the liberation of droplets of fat into the affected cells. On varieties of tubers subject to necrosis, external symptoms are usually manifested in the form of irregularly shaped, brown patches, first starting at the heel-end, which later develop into sunken, brown, dry areas. Necrosis does not extend into the 'eyes', or into the vascular tissues. It develops chiefly during storage, and is favoured by darkness and high temperatures [21].

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Soft Rot of Turnip, *Bacterium carotovorum* (Jones) K. B. Lehmann

Bacterial ' soft rot ' affects the succulent parts of a wide range of cultivated plants. Biennials and perennials swollen with food reserves are its principal hosts ; these include turnip, swede, carrot, parsnip, potato (tuber), onion and hyacinth (bulb), arum lily (corm), iris (rhizome), the succulent tissues of broccoli, cabbage, celery, etc. [1, 7, 13]. It is also found as a foot rot of cucumber and melon, a root rot of runner beans [4], a rot of cultivated violets, and a stem [5] and fruit rot of tomatoes, but cultivated fruits of trees and shrubs, in general, are not subject to this bacterial decay. It frequently follows in the wake of numerous diseases caused by fungal parasites attacking a great variety of plants, the bacteria thriving more or less under saprophytic conditions.

This soft rot has been ascribed by different authors to an organism which has from time to time been variously named, but *Bacterium carotovorum* is now generally accepted as the correct designation [5a, 9], and though there are undoubtedly numerous strains of this parasite differing in minor morphological and cultural features these differences are not considered to be sufficient to establish these variants as distinct species [2, 2a, 3, 6, 8, 12, 17, 18, 19, 20, 24].

The symptoms of soft rot in which this organism pursues a parasitic career are fairly typical on turnip or swede in active growth in the field [10, 14, 15, 16]. Plants of all ages, from young seedlings to mature ' bulbs ' may be attacked. Infection in all cases is effected through wounds such as are caused by slugs, insects, or by careless thinning, weeding or hoeing. It is not usual for very young seedlings to become rotted and killed unless the infected wound is at the growing point of the young shoot. Plants injured later, in the same way, may make good the loss of the primary shoot by developing a number of secondary shoots around the seat of injury. The injured apical bud makes an ideal entry and the rot soon makes rapid progress into the succulent tissues in the wound.

A cursory glance at the turnip crop during the summer usually reveals little signs of the presence of the rot, except perhaps a few gaps here and there in the drills due to early death of seedlings. Later, when the bulbs are beginning to show



FIG. 273.—Soft rot (*Bacterium carotovorum*). *A*, on swede; the section shows the internal tissues rotted brown, this discoloration being characteristic in spongy, aerated tissues (photo by Foister & Noble). *B*, on carrot (photo by Ogilvie, by permission of Long Ashton Res. Station). *C*, section of white turnip (Green Globe) showing advanced stage of rot, the interior hollow, but vascular connection in the rind, between root and leaves, still functional; in sappy, watery roots the diseased pulp remains white (photo by Jones, *J. Agric. Sci.*)

fair growth, with more or less luxuriant foliage, the extent of the rot in infected bulbs is by no means always revealed in the appearance of the expanded leaves. But some affected plants in advanced growth may show evident signs of the disease by the outer leaves wilting and drooping, becoming yellow and shrivelled in appearance, the inner leaves in turn going the same way, until the entire rosette of leaves perishes, the whole root collapsing to the ground. The internal tissues of the bulb meantime become converted into a white, pasty, putrid mass which disappears into the soil after a shower of rain leaving the resistant rind derelict on the surface, and from which slugs and marauding insects pick up infection [14, 15, 16]. In other plants, particularly those well established and infected late, in which only the youngest tender leaves at the centre of the rosette were wounded, leaving the rest of the foliage unharmed and normal, it is often not possible to detect the extent of the rot in the bulb except by inserting a probe into the wound concealed by the luxuriant foliage. Indeed, the extent of internal damage is often not fully revealed until the time of lifting when the harvester lops off the foliage, only to discover that the roots are practically dried out and hollow (Fig. 273 C). Very frequently the rot starts from a gaping wound at the side of a bulb, caused probably by hoe or cultivator, and when pulled up by the leaves at harvest, the top of such a bulb breaks away easily from the less affected root which remains in the ground. Why some plants, as in the first case cited above, suffer from a complete collapse of the foliage during the progress of the internal rot, or as in the second case when

the foliage and rind remain practically intact to the end despite disease in the bulb, is not known, but it appears that certain varieties of turnip, e.g. Green Globe, though very susceptible to the rot, have a much more resistant rind than other varieties.

In a vertical section of a diseased turnip the soft, putrid core is everywhere delimited from the sound tissue by a narrow brown zone, and if infection starts at the apex, this zone finally takes the shape of a flask extending to within about ¼ inch of the rind; it does not extend into the narrowing basal part of the plant, that is, into the region of the true root, where it forms the flat or convex base of the flask (Fig. 273 C). From an examination of cut plants, at all stages of growth, it is apparent that the brown zone steadily advances in front of the white rot, and becomes obliterated behind as the rot progresses towards the rind. It is clear that the tissues are killed in advance of the organisms, for comparatively few bacteria are to be found in the brown zone. Despite, therefore, considerable destruction of the tissues of the core and much disintegration of the more internal parts of the vascular system of the bulb, the fact that the vascular elements at the base of the root and at the periphery of the bulb still remain more or less intact, no doubt accounts for the retention and healthy turgescent appearance of the bulk of the foliage of many affected plants.

The initial mode of attack of this disease in the field is very difficult to detect. Close examination of the growing plants will often show, however, intermixed in the drills with diseased bulbs, plants with luxuriant foliage but having brown, dry empty cavities opening to the exterior where the youngest tender leaves have apparently been destroyed, and in which there is no sign of soft rot at all; but such cavities frequently harbour slugs or their ova. These plants show, like the diseased roots, either the young foliage at the growing point bitten off, or the entire absence of the apical shoot, the scar left by the latter having become completely healed and surrounded by luxuriant foliage from secondary crowns. Slugs and insects which have been feeding on diseased turnip no doubt carry the disease through the crop, but how the organisms survive in the field from season to season is not known. Quantities of turnip trash left behind after harvest harbour the organisms over long periods even into the depths of winter and never fail to yield the bacteria when cultured. Possibly carrier weeds, especially crucifers, in hedgerow or headland may become infected by slugs or insects and serve to carry infection to the new crops.

Bacterium carotovorum is a short, rod-shaped organism occurring singly or in pairs, and on sugar-rich media long, unequally segmented filaments are often formed. Individual bacteria measure from 1·3 to 3·0 by 0·75 to 0·9 μ [10], but they are of variable dimensions [8, 11, 15, 17]. They are non-sporing and are furnished with a long, single polar flagellum, but strains with peritrichic flagella are also reported [20]. On neutral beef-broth-peptone-gelatine, the colonies are large, greyish, translucent, with a fibrillated margin, the colonies becoming deeply concave by solution of the medium. On agar media the colonies are round, slightly lobed, smooth, and opalescent; sometimes filmy areas with a dentritic margin are formed; buried colonies are small, oval or spindle-shaped. The organism is aerobic and facultative anaerobic, acid-forming, without evolution of gas in sugar media, and nitrate-reducing. It dissolves the middle lamella of the tissues and a toxin is pro-

duced (Figs. 274, 275). Acid vegetable media must be made neutral for maximum growth in culture [6, 10].

Artificial inoculations of turnips at any stage of growth are ineffective on any part of the plant except through a wound. Holding the leaves down in a water or broth suspension of the bacteria failed entirely to induce the organisms to

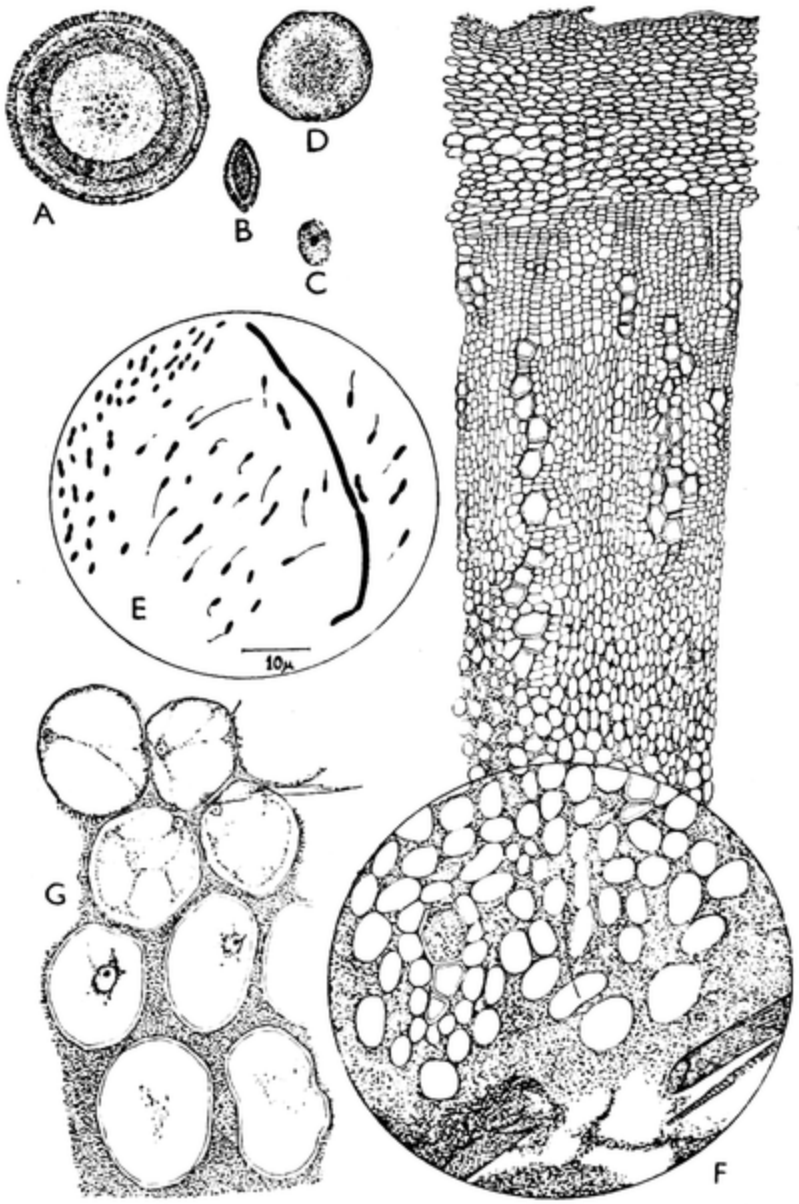


FIG. 274.—*Bacterium carotovorum*. *A*, a colony on nutrient gelatine (×2) (Fig. 275 A). *B*, *C*, spindle- and oval-shaped, small buried colonies in nutrient agar (×6). *D*, a typical lobed colony on nutrient agar (×4). *E*, the bacteria (stained with Loeffler's mordant and carbol fuchsin) showing a single terminal flagellum (long 'filaments' appear on a sucrose medium). *F*, transverse section of white turnip (Green Globe) showing the peripheral tissues and vascular bundles intact, the bacteria attacking only the central tissues, as shown in the circle, where the medulla becomes completely disintegrated, thus separating the cells and lignified elements. *G*, cells of the medulla showing various stages of disintegration; at the top the cells appear normal, but with the accumulation of the bacteria in the intercellular spaces, there is a swelling of the cellulose walls and enlargement of the nuclei before the cells are killed and the walls finally dissolved

enter the stomata or water pores to bring about disease in the bulb, though under other unknown conditions prevailing in the field during growth of the crop the bacteria might possibly enter through these channels, but there is no positive

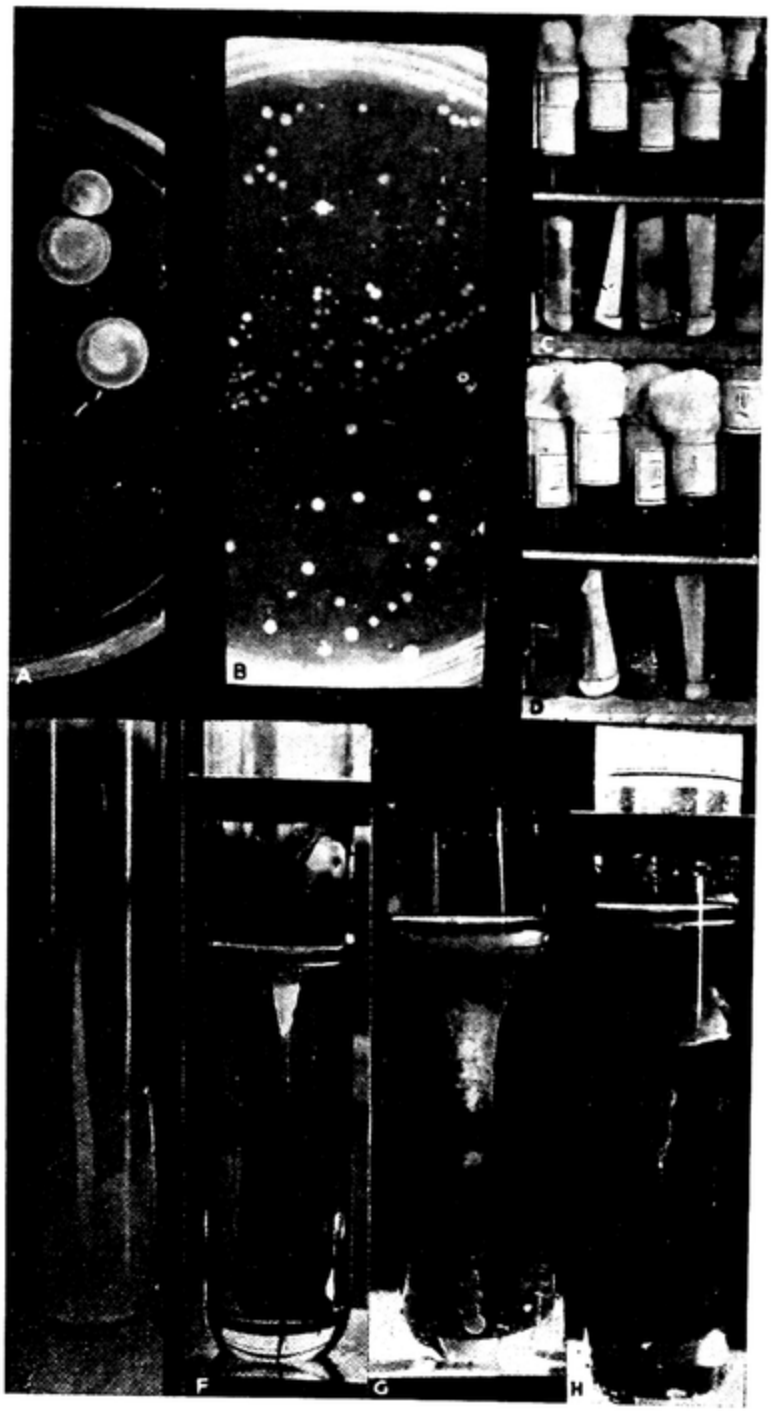


FIG. 275.—*Bacterium carotovorum*. *A*, the colonies on nutrient gelatine showing liquefaction of the medium; 3 days' incubation. *B*, the colonies, in crowded growth, on nutrient agar; 3 days' incubation. *C*, three inoculated chunks of turnip showing water-soaked areas after 3 days' incubation; two alternate tubes, control. *D*, the same after 7 days' incubation, showing complete collapse of infected chunks. *E*, characteristic growth of the organism on agar-slant cultures. *F*, a gelatine stab culture; 3 days' incubation, 18° C. *G*, the same after 7 days. *H*, growth in stab-culture, after sealing of the track, showing growth under anaerobic conditions (photos by Thomas)

evidence of such infection. Infections are successfully performed if a pure culture of the organism is deposited in a small abrasion made in the centre of the leafy rosette, the inoculated part being kept moist for a time. But the progress of the disease and the extent of browning of the core vary considerably in plants of different varieties, age, and texture of tissues. In young, sappy bulbs progress is comparatively rapid, the diseased core showing the characteristic whitish-grey pasty mass accompanied by the marginal brown discoloration abutting on the sound tissues around. In older roots of a dry spongy texture progress is much slower, and the rotted core is more or less uniformly brown. Apparently the brown discoloration is closely connected with oxidation of the bacterial by-products as they infiltrate into the tissues. The fact that the browning in a succulent root takes the form of a narrow flask-shaped zone between the healthy and diseased tissues is probably due to the toxic effects of the bacterial secretions on the tissues, acting in advance. By the action of the secretions in dissolving the middle lamella, followed by a collapse of the cells as they are killed and loosened, access of air through the diseased core, is prevented and so the latter remains white. In older roots progress is slower in the drier tissues, and the spongy nature of some of the roots allows for much aeration of the affected tissues which therefore remain uniformly brown. Moreover, the absorption of certain nitrogenous fertilisers such as nitrate of soda or sulphate of ammonia is also known to accentuate the brown discoloration in host tissues suffering from soft rot.

In the tissues of the host the bacteria are intercellular, occupying the medulla in great profusion but getting gradually sparser towards the outer tissues of the rind. Sometimes a few cells of the medullary parenchyma and of the lignified vascular elements may be seen to be occupied by the bacteria, but this type of soft rot is not a vascular affection, and the bacteria do not usually invade the living cells. The tissue debris from the pasty core shows parenchyma cells in all stages of digestion, the cell walls and nuclei first swelling after the middle lamella is dissolved, and thereafter becoming more or less completely dissolved, and interspersed amongst the disintegrated parenchyma may be seen the more resistant elements of the lignified tissues, vessels, spiral tracheids, and fibres (Fig. 274 F, G).

Control of soft rot in the field is a very difficult problem. As they are liable to force the growth and induce the formation of watery, sappy bulbs, nitrogenous fertilisers, especially nitrate of soda, should be used sparingly. Drainage should be attended to, as crops planted in low-lying areas are usually the first to suffer decay. Care should be taken to avoid injuring the growing plants when thinning out, weeding, or hoeing. In small areas means should be adopted to reduce slugs, but on a wide scale this is obviously not practicable.

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Club Root of Crucifers, *Plasmodiophora brassicae* Woronin

' Club root ', or ' finger-and-toe ', attacks a wide range of cultivated and garden plants, all of which belong to the *Cruciferae* family. Upwards of a hundred different species and varieties are recorded to be more or less susceptible to this disease [30].

Club root has been known for over two centuries ; it was reported in Britain to have occurred in Norfolk and Suffolk about 1736 [9] and in north-eastern United States about 1867 [21, 36, 37]. The disease is now widely distributed and is responsible for very heavy losses in the commonly edible cruciferous plants, particularly cabbage, turnip, and swede.

The first scientific investigation of club root (on the cabbage) was made in Russia in 1878 by Woronin [38], who described the causal organism, a member of the so-called slime-fungi or Myxomycetes (Myxogastres), under the title *Plasmodiophora brassicae*, a name highly descriptive of the presence of the organism in the host in the form of naked, jelly-like bodies, or plasmodia.

Club root, as the name suggests, is essentially a disease of the rooting system, and the appearance of the ' clubs ' or galls on the stem of the host either follows upon wound infection or is due to secondary migration of the parasite into the stem, from primary infection already established within the roots [19]. The presence of the disease within the host leads to a remarkable increase of growth in the parts affected, causing the roots, for the greater part, to become abnormally swollen ; but the symptoms vary somewhat according to

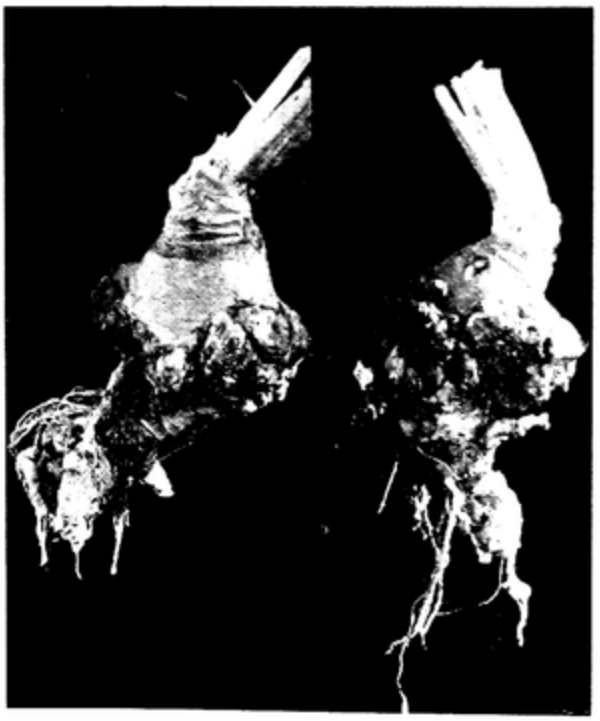


FIG. 276.—Club root (*Plasmodiophora brassicae*). The galls on swede turnip (photo by Scott Wylie)

the habit of the plant. On the cabbage, for instance, furnished with a comparatively slender tap root with numerous lateral roots, the galls take the form of spindle-shaped swellings of variable thickness of the tap root and of practically all the lateral roots. On a swede or turnip (Fig. 276), in which the lateral roots occur below the bulbous hypocotyl, the diseased galls appear on these roots or on the tap root, as warts or tumours, and the ' bulb ' itself is not greatly disfigured with galls [19, 36]. It is true that galls on the bulbous stem arise often enough, but only as a result of infection through wounds caused by rough particles in the soil, or by grubs, slugs, careless weeding, hoeing, etc.

The disease may attack the host at any time as long as active growth is taking place. Symptoms of wilting or a yellowing of the foliage are not usually seen until the plants are fairly well advanced, and despite the diversion of elaborated food from plant to gall as a result of infection, actual interference with leaf functions does not occur until gall development within the root has proceeded so far as to disturb the continuity of the vascular supply from root to leaf. Such a derange-

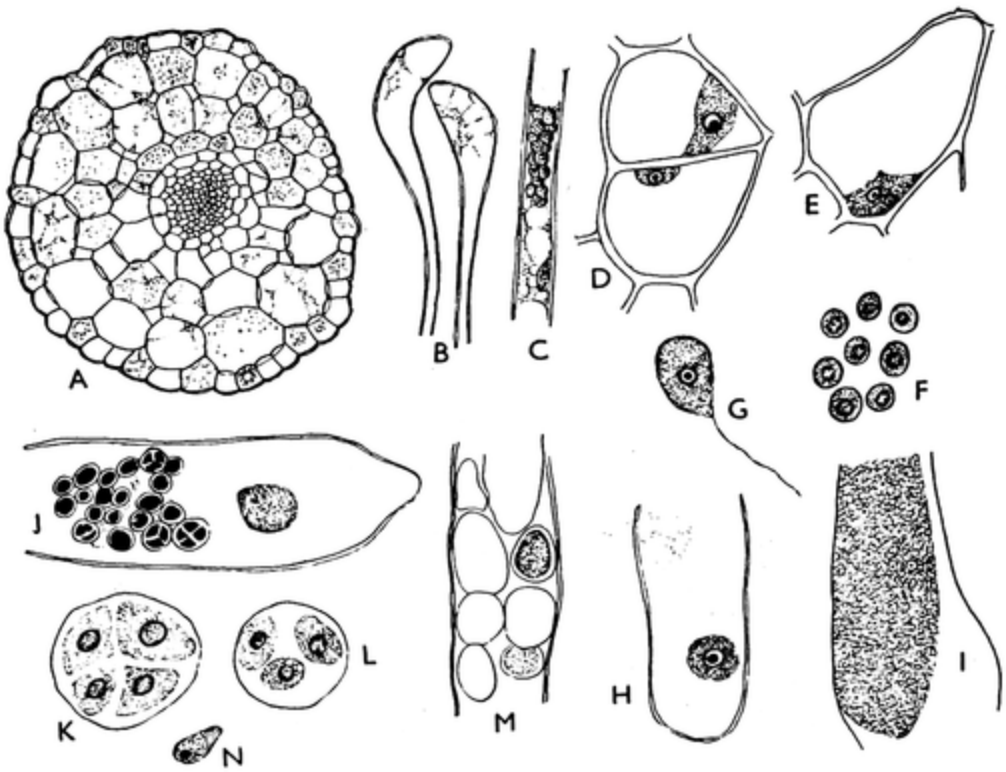


FIG. 277.—*Plasmodiophora brassicae*. *A*, transverse section young root of cabbage showing young plasmodia (shaded) in some of the cells of piliferous layer and cortex. *B*, early infection of two root hairs showing myxamoebae. *C*, root hair containing, probably, a number of zoosporangia (all after Woronin). *D*, uninucleate amoebae in host cells. *E*, young plasmodium in host cell (× 550). *F*, spores (× 1600). *G*, a swarm spore (it has two unequal flagella) (× 1600). *H*, root hair containing swarm spore, the flagella having retracted (× 1250). *I* to *M*, stages in the formation of zoosporangia and zoospores, in root hair. *I* (× 550). *J* (× 1250). *K*, *L*, uninucleate zoospores in zoosporangia (× 2250 and 1600 respectively). *M*, a group of zoosporangia in a root hair showing some in process of development, while others have discharged their zoospores (× 1250). *N*, a mature zoospore, prior to fusion : this constitutes a gamete which will fuse with a similar one to form a zygote which infects the host tissues (× 2250) (all after Ivimey Cook, *Trans. Roy. Soc., Lond.*)

ment of the host tissues must, of course, result sooner or later in a yellowing, wilting, loss of foliage, and a stunting of the plants.

A transverse section of a hypertrophied cabbage root shows the infected areas to consist of several scattered groups of cells, chiefly in the cortex and medullary rays, having denser contents than normal (Figs. 277 A; 278). These cells are practically filled with plasmodia or with a multitude of very minute spores derived from them, the spores being enclosed in no covering save that of the host cell accommodating them (Fig. 278). In advanced stages of disease, consequent upon such increase in the size of the galls as to cause disruption of the outer tissues of the host, a general decay of the galled tissue sets in, and with the access into the roots of other secondary organisms from the soil, host cells are broken down and the spores of club root are released in myriads into the soil.

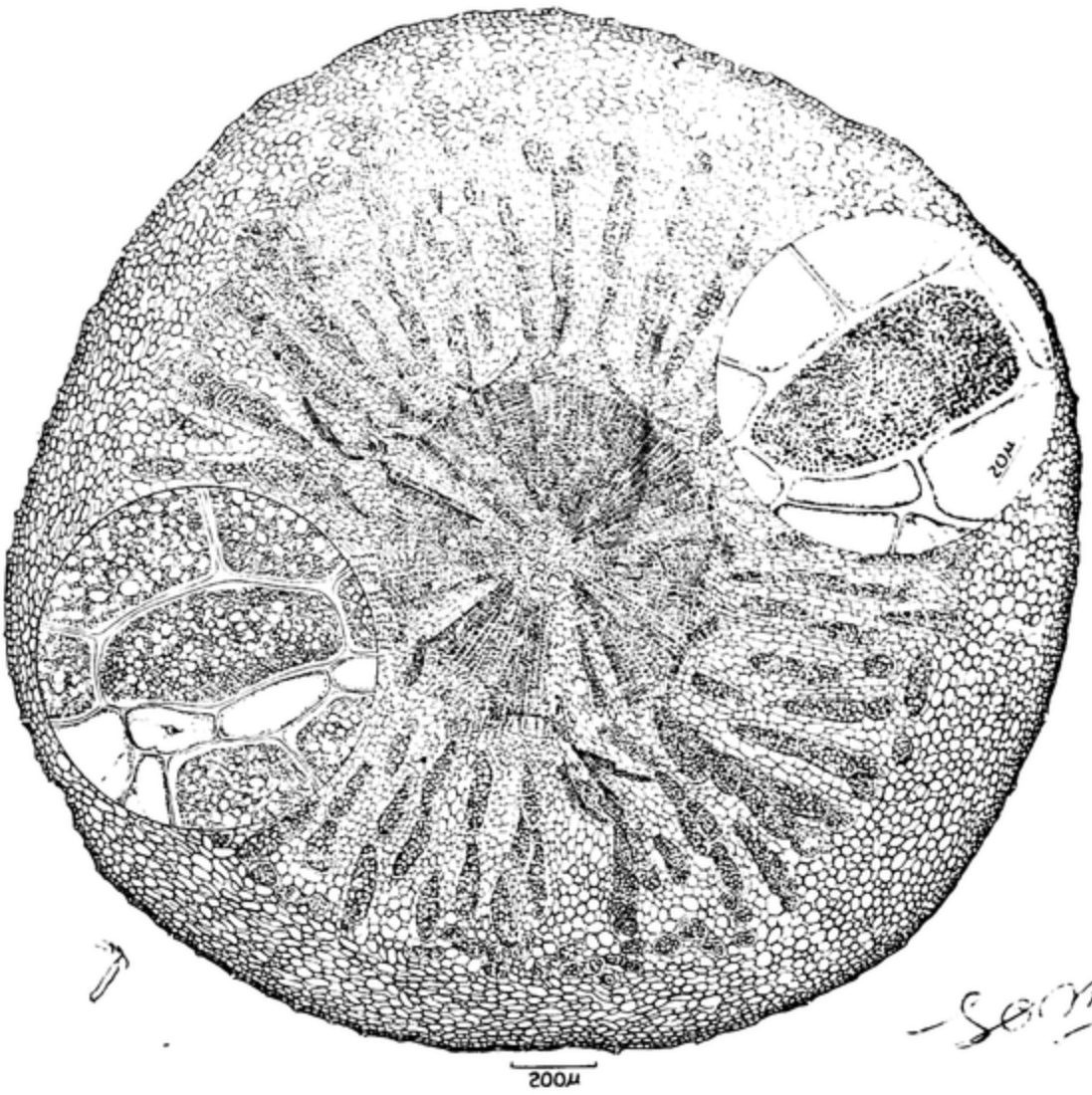


FIG. 278.—*Plasmodiophora brassicae*. Transverse section root of cabbage showing enlarged cells infected with plasmodia, with considerable division of the cells in their vicinity. Note increase in width of medullary rays, causing disruption in vascular cylinder. Insets, left, to show comparison between the enlarged host cells, occupied by plasmodia, and the smaller uninfected cells; note the frothy nature of the plasmodia; right, an advanced stage in the formation of spores, the host cell constituting a sporangium (see Figs. 2, 144) (from a slide made by Gonzalez)

Although the spores of *P. brassicae* are capable of immediate germination, they are known to be viable after periods of 3 to 6 years, even to a depth of 12 inches, in the soil [26, 28, 32]. A few instances are recorded where it was possible that the spores could be carried on the seed, and in other cases of reported transmission by seed, it is not unlikely that the outbreaks arose owing to contamination of the seed with infested soil [39]. It can be spread by any means that may carry contaminated soil from place to place, on farm implements, through biotic aid, or by the use of manure containing remains of diseased crops; and there is little doubt that the practice of feeding cattle and sheep with raw club roots is a means of returning the parasite to the soil. The dispersal of spores by wind is hardly likely except in periods of extreme drought when it is not improbable that strong winds blowing over light soil may carry the spore-laden dust to fresh areas; and although the spore wall is comparatively thin, the spores resist desiccation to a marked degree [7]; but heavy rains and surface flood-waters distribute the spores widely in the soil. In cultivation, however, one of the most fruitful means of spreading the disease is by means of small quantities of soil usually found adhering to transplants from infected beds, or on such organs as bulbs and tubers raised in soil contaminated with club root. The disease, as already noted, has a wide range of hosts among which are some of the commonest weeds of the hedgerow. It is a frequent observation that, soon after renewal of the cruciferous crop, following long periods of rotation with non-susceptibles, the disease often breaks out first in places close to hedges or headlands, a possible explanation being that cruciferous weeds growing in these positions serve as carrier hosts. The spores from these weed hosts would probably be washed into the soil, and during ploughing would perhaps be deposited mostly along the border of the field and around the headlands.*

The parasite of club root is not amenable to growth in artificial culture, but the spores are easily germinable in tap water or in nutrient solutions at room temperature; frost stimulates germination but is not essential [1]. The spores are variable in size; various authors give the dimensions as: 2·8 μ [1]; 2 to 3 μ [7]; 1·6 μ [38]; 3·3 to 4·3 μ [5]; " never over 4 μ " [15]; 1·7 μ (young), 2 μ (old) [37], in diameter. In germination, the spore liberates a single naked, pear-shaped, uninucleate zoospore furnished with two unequal flagella at the pointed end [1, 20]. After a short period of motility it comes to rest and undergoes considerable change of shape, lengthening out at its narrow end into a beak, while at the broader end putting forth portions resembling the pseudopodia of an amoeba. Such a body bereft of its flagella is called a myxamoeba, and some maintain that the body is not flagellated at all [13]. Outside the host plant further stages of spore germination have not been observed.

Infections have been studied chiefly in relation to seedlings of the cabbage, grown in soil contaminated with galls of club root finely macerated to ensure a maximum release of spores. A temperature of 25° C. is favourable for these observations. Natural infection has been observed to take place when zoospores or myxamoebae come to rest on the moist surface of root hairs or other cells of the piliferous layer which presumably they penetrate without difficulty. Infection

* The organism has recently been observed in root hairs of non-cruciferous plants. Webb, 1949: *Nature*, 163, 4146, p. 608.

occurs over a brief period only when the root hairs are young and full of protoplasm. It increases with the density of the spores in the soil and is greatest when the latter is at a *p*H of 6·2, and least at *p*H 7·7 [35a]. Access to the host tissues may also be obtained through wounds on root or stem, and perhaps through the natural openings at the points of emergence of lateral roots [15]. In the root hairs of seedlings, about 4 to 7 days from infection, bodies resembling myxamoebae may be seen, some appearing to move from one end of the root hair to the other. Should multiple infection have occurred, the myxamoebae retain their identity and their fusion in the root hair has not been observed. The myxamoebae still within the root hair now undergo transformation into zoosporangia, each giving rise to about 4 to 8 swarm-spores or zoospores. As the zoosporangia are all in close contact with the wall of the root hair, the swarm-spores, at maturity, are discharged through minute pores made at the points of contact. These new crops of zoospores, formed so early following initial infection, are considered by some to be sexual gametes [7] (they are smaller than the original zoospores) capable of pairing and fusing to form zygotes (cf. *Spongospora subterranea*, p. 495, and *Synchytrium endobioticum*, p. 500), but others have failed to detect fusion between any kind of motile bodies produced by the parasite of club root, the swarm-spores serving merely as additional infective bodies in the history of the disease [1]. Others have observed that the zoospores are sometimes discharged from the sporangia into the cavity of the root hair [35a]. Bodies resembling myxamoebae which presumably have migrated from the piliferous layer have been observed in the inner tissues of the root, in the cortex, and even in cells near the root tip, having reached these destinations probably by progressive penetrations from cell to cell. Whatever the nature of the infecting body may be, whether zoospore, myxamoeba, swarm-spore, or zygote (Fig. 277) (from fusion of swarm-spores as potential gametes), it finally settles down in a living cell, usually in the cortex or in a medullary ray. By growth and active nuclear division, the body proceeds to build up a multinucleate plasmodium in which finally several hundred free nuclei are embedded. A typical plasmodium of *P. brassicae* (Figs. 2 F, G, 278) has the appearance of a frothy mass of greyish jelly containing a number of embedded granules and small oil globules; the numerous minute nuclei are, of course, not discernible except in preparations suitably fixed and stained. At early occupation of a cell, it is not easy to distinguish between plasmodium-mass and host-cytoplasm, and the one lies against the other with no perceptible demarcation. Apparently there is little interference with the normal functions of the infected host cell for an appreciable time and the host nucleus looks quite normal. A host cell may possess more than one plasmodium, and the plasmodia appear to maintain their separate identity, but such a condition may apparently break down, and two or more plasmodia have been observed to fuse together into a common, but still multinucleate mass. Finally, by segmentation of the plasmodium, the whole mass becomes divided into as many units as there are nuclei, and these bodies become rounded off to form a more or less dense aggregation of spherical spores, each spore being furnished with a smooth wall enclosing vacuolated cytoplasm and a nucleus (Fig. 277 F).

The presence of a plasmodium in a cell stimulates not only the occupied cell

to meristematic activity but also the cells contiguous to it. This is effected in two ways : (*a*) by the one or more divisions of the infected cell being accompanied by a sharing of the plasmodium into the daughter and subsequent cells, before the divisional walls are laid down ; and (*b*) by bits of the plasmodium, cut off within an infected cell, migrating through pores in the wall into contiguous cells, with a repetition of the same process within prescribed areas. Both of these methods appear to be operative, though several authors deny the capacity of plasmodia to penetrate cell walls.

While, in the early stages of root infection, the invaded host cells are naturally those of the primary cortex, the parasite, later, seeks out the primary cambium. This meristem, however, is not seriously assailed until an appreciable amount of secondary tissues has been formed by it in the normal manner. The occupation of the cambium, however, initiates a much more serious phase of the disease than was the case in the occupation of the primary cortex. Plasmodial portions within dividing cambial cells are supplied to practically every living cell added by the cambium to the phloem and phloem parenchyma, as well as to the medullary rays, on both sides of it. But the parasitised cells, wherever they may occur, have no prescribed plan of division, and their irregular multiplication at advanced stages of infection takes place on so wide a scale that the proliferating tissues intrude upon the vascular elements of the wood to such an extent as to break the vascular continuity (Figs. 144, 278). Such abnormal meristematic activity, at numerous centres of infection within a root, causes considerable displacement of host tissues, which are therefore forced outwards and finally appear at the broken surface as clubs or galls of hypertrophied tissues [17].

Germination of the spores occurs over a wide range of temperature, according to various authors, from 6° to 27° C. [37]; 6° to 35° C. [24]; the optimum, between 18° and 25° C. [24, 37], or between 27° and 30° C. [5]. In general, the range of temperature for development of the disease is practically the same as for spore germination, but the temperature effects upon the growth of the host may be different [37].

The amount of moisture in the soil is a significant factor in the incidence of this disease. In a graded series of experiments [24], infection occurred at 60 per cent. moisture capacity of the soil but not at 45 per cent., or below, and with a rise above 60 there was a corresponding rise in virulence of the disease. But adequate drainage alone does not keep the disease in check, and it has been established that while the success of initial infection depends largely on the length of time the roots are in contact with water, favourable to spore germination, a fall in the moisture content, after infection has been accomplished, has little effect upon the progress of the disease. As short a period as 18 hours' incubation in the soil is sufficient, under excess of moisture, to establish thorough infection in the root [37].

Long before the true nature of club root was understood, soil acidity was considered to play the most important part in the development of the trouble, and the application of lime to the soil was one of the earliest methods used to combat the disease. But spore germination is not exclusively dependent on relative reaction of the soil [4], and may occur with equal facility in alkaline or acid solutions, under certain controlled conditions, such as maintenance of a temperature not above 21° C. [13]; in the absence of a susceptible host the organism becomes

exhausted sooner in acid than in neutral soils [4]. In general, the organism ceases to cause infection at, or slightly above the neutral point, and the maximum limit appears to lie between *p*H 6·9 and 7·8, perhaps nearer the latter value [6, 29, 30]. The action of liming the soil for the control of club root is still imperfectly understood, and the amount of lime added is not the chief factor in inhibiting spore germination [4]. But the form in which it is added is important, and while hydrated lime is of wide acceptance, its action is said by some to be due, not so much to its neutralising properties as to its direct toxic action on the parasite ; in this respect it was found to be much more effective as a lethal agent in the soil than the sulphates or carbonates of lime [37]. In other cases, however, quicklime is reported to give better results than hydrated lime, or limestone [3]. Again, experiments conducted in Wisconsin in 1930 and 1932 showed that hydrated lime did not give uniform control in the field although the reaction was neutral, but the effects were much more successful under greenhouse conditions when frequent watering could be carried out. It would appear, therefore, that a fluctuation in the amount of soil water, and forced aeration, permit of varying degrees of infection even in slightly alkaline soils [18]. A high concentration of spores may also induce infection in such soils, and this may perhaps explain why liming sometimes fails to control club root [35a].

Lime is best applied in the hydrated form at the rate of $\frac{1}{2}$ to 2 tons per acre [37] ; lime or quicklime should be allowed to slake by exposure, and applied at 4 tons per acre, forked well in, several days before planting [2]. In small areas good results follow the application of disinfectants such as the highly poisonous chlorides of mercury, corrosive sublimate, and calomel [12, 30, 33]. In the planting of cabbages, for instance, the sublimate is employed, 1 oz. in 10 to 12 gallons of water, applying about $\frac{1}{2}$ pint to each hole before planting ; or 2 per cent. formalin, at rate of 2 gallons per square yard may be used 4 weeks before sowing [33]. Calomel may be used in powder form, a 4 per cent. dust raked into the soil at a rate of $1\frac{1}{2}$ oz. per square yard being very effective [12a], or the roots of each transplant may be dipped in the dust at planting [2, 33]. Variable results are reported for the use of lime substitutes such as calcium cyanamide [16] and nitro-chalk [33].

Loam and clay soils are generally less prone to harbour club root than moorland and light soils [25, 30]. The application of organic manures fosters the disease, this being attributed by some to the capacity of the organism to exist in amoeboid form as a saprophyte, and by others [32] to the action of the manures in encouraging the retention of soil moisture favouring spore germination. In the field, such manures as well as acid fertilisers should be applied at other periods in the rotation when non-cruciferous crops are grown. Long periods of rest from crucifers are advisable in order to starve the parasite out of the soil, and every effort should be made to eradicate susceptible weeds from the vicinity of the crops.

It is recorded that club root is most severe on turnips having a high content of sugar, and a direct relationship has been found between relative resistance of the host and the percentage of certain glucosides which, in fermentation, produce highly pungent mustard oils ; and the further suggestion is made of the possibility of controlling club root by breeding and crossing of species otherwise desirable but deficient in the active glucosides, with those containing higher amounts of

these substances [34, 35]. It is interesting to note that a chemical analysis of leaves and roots of healthy cabbage, in comparison with those of plants diseased with club root, showed a difference in protein content; in healthy leaves it was 33·37 per cent., and 28·65 per cent. in leaves of diseased plants; the values in the roots, however, were of a reverse order, being 11·19 per cent. and 32·87 per cent. in healthy and diseased roots respectively; moreover, the amounts of phosphorus and potassium were larger in healthy leaves than in leaves from diseased plants, the reverse being the case in the roots [27].

Immunity from club root is not claimed for any kind of crucifer. The yellow turnip, Bruce Purple Top, The Wallace, Dale's Hybrid, and Irvine's Greentop Yellow, possess high resistance [11, 29a]. In Sweden the first variety is favourably reported upon, as well as another kind, 'Immuna' [30]. In Wales, the Danish strains of swedes are recorded to have superior resistance in comparison with British kinds; while none of the strains was found to be immune, they all gave higher yields on infected land than any of the home varieties, and though they were considerably out-yielded by the latter in healthy soil, they still possessed better keeping qualities. Of the Danish Bangholm swedes, the best-resisting are Hernig, Studsgaard, and also the Øfofte strain of the variety Wilhelmsburger; the Welsh trials also point out the importance of knowing the place of origin of the seed [8]. No varieties of cabbage, broccoli, or brussels sprouts are immune from club root, but marrow-stem kale and some strains of kohlrabi are less susceptible to it.

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Dry Rot and Canker of Swede and Turnip, *Phoma lingam* (Fr.) Desm.

Dry rot of swede and turnip has been known in England for nearly half a century and is common everywhere throughout Britain [13, 14]. It also occurs in Denmark and Canada, and appears to be the most serious disease of root crops in New Zealand [4, 8, 12]. Weeds, such as wild turnip, white mustard, charlock, wild radish, horse-radish, and wallflower, are known to be attacked by the fungus *Phoma lingam* causing this dry rot, and some of these may serve as carrier hosts to the cultivated crops in the field. As there are two fairly distinctive phases of this disease, one a wilt, the other a canker, it is very difficult to assess the losses incurred in the growing crop, and later, when the roots are in storage. Though not usually as serious a trouble as ' club root ', in a bad season losses may be from 10 to 50 per cent. [9, 14], and 50 to 100 per cent. of swede crops have been lost in New Zealand where it is less severe on turnips, though losses up to 40 per cent. have been experienced on that crop as well [4]. On some farms in England, where short rotations are practised, swede crops have had to be abandoned owing to the severity of dry rot [1].

The disease may start on young seedlings as well as on mature plants. All parts may be attacked, including pods and seeds. When seedlings are about a week or a fortnight old, sometimes the cotyledons of affected plants turn grey and shrivel up in 2 or 3 days [9]. In other cases seedlings may perish from being attacked at the hypocotyl or at the root tip ; seeds may sometimes be so severely infected that they decay without germination [7]. Even at these very early stages of the disease on ungerminated seeds and seedlings killed, the tiny black pycnidial fructifications of the fungus are produced in great numbers. These early seedling infections are believed by some to serve as foci of infection for spreading the rot to the surrounding healthy crop. Typical dry rot usually begins about mid-July when the fleshy roots are about $\frac{1}{2}$ inch thick, the plants in scattered places in the field showing a bluish coloration of the leaves, in striking contrast with the green of the healthy crop. Such plants soon wilt and may be so girdled with disease that the upper parts collapse and separate easily from the rooting portion. On the crowns of these plants the tiny black pycnidia are present in great numbers and serve to infect surrounding plants. Spread of infection, however, is slow, and decay within confined areas extends very gradually and is never epidemic throughout the entire crop.

Dry rot does little harm to the expanded leaves of established plants but usually on the older, yellowing, drooping leaves, less commonly on those still green, slightly sunken spots may arise on any part of the leaf blade, usually circular, or angular if against a vein, 1 to 1½ cm. in diameter, of a dirty-white colour, with a light-brown or yellow margin, and when held up to the light each spot is bordered again by a zone of deeper green than the surrounding area [2]. Numerous black pycnidia develop on both sides of a leaf spot (Fig. 279 B), but some authors state that these leaf pycnidia never open in the field to set free their spores [14], dissemination apparently taking place only from those formed on other parts such as the neck, bulb, pod, and seed.

The more serious phase of dry rot, the canker stage, starts at the neck and later penetrates into the interior of the bulb, resulting in the partial or complete breaking across of plants at soil-level. Bulb rot is accompanied by an internal blackening of the tissues and the production of abundant black pycnidia on the lesions or around the lips of the dry, gaping canker (Fig. 279 A). Early signs of the onset of canker consist of small green spots on the neck of the bulb ; they are horizontally elongated and depressed and later turn grey or brown in colour. In small bulbs these lesions may extend inwards and cause early death. At first few in number on the neck, the lesions increase and may become scattered over the entire exposed

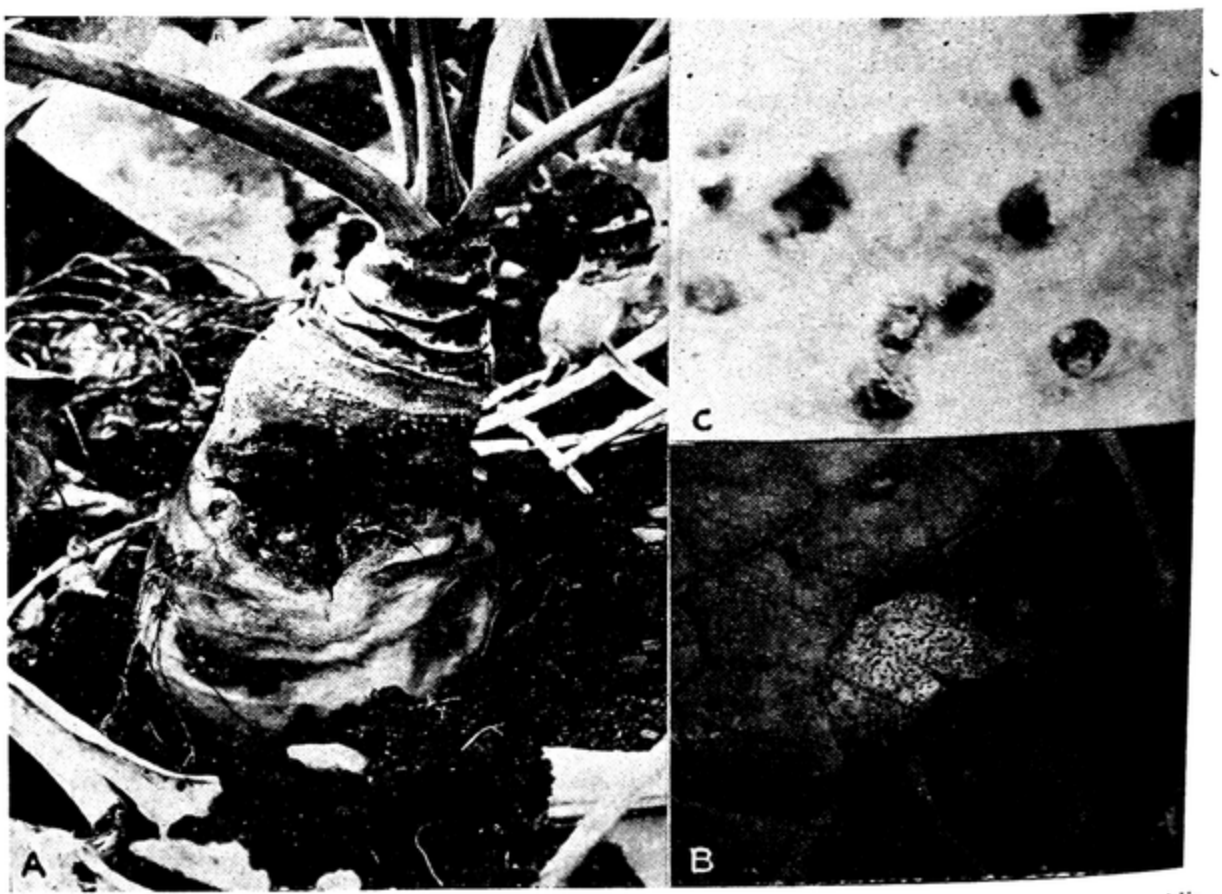


FIG. 279.—Dry rot of swede (*Phoma lingam*). *A*, lesion on the bulb. *B*, the tiny black pycnidia bordering a lesion on the leaf. *C*, pycnidia (enlarged) on the bulb (photos *A*, *B*, by Foister & Noble)

surface of the bulb, but rarely developing below soil-level. Though lesions may be numerous on the more advanced bulbs, the latter are not killed unless the lesions join together. When a lesion is young and growth of the plant is vigorous, a horizontal crack may develop in the rind and girdle the plant so completely that the top falls off, leaving the rooted part behind. Pycnidia are usually abundant in the vicinity of the bulb lesions but are not invariably present [4].

On the flowering stems of plants grown for seed small lesions similar to those described on the neck may frequently occur. After flowering is over, lesions may also be seen on the pods, and these may take the form of indefinite blackened areas either at the point of attachment of the pod to the axis or at the stigmatic end, or of better-defined spots at intermediate points, somewhat similar to those on the green leaves. Infections from such pod lesions may enter any seeds in contact with the affected pericarp, and sometimes penetrate so deep into the seed as to stop its development, but seed infection very rarely goes beyond the testa. It is, however, important to note, despite the fact that infected seeds are considered by many to play but a minor part in the spreading of the disease, such seeds when killed outright, or soon after germination, produce pycnidia and spores (especially in sunlight), and if they can persist in the soil it is quite likely that diseased seeds may act as foci of infection [7]. But the methods for over-wintering of the fungus are discussed again below.

Phoma lingam, the parasite of dry rot, is a member of the Sphaeropsidales (Fungi Imperfecti). The pycnidia are sub-epidermal, black, globose or lenticular, from 130 to 340 μ in diameter, opening by an ostiole ; pycnospores arising from a layer of beaked cells lining the fructification are unicellular, elliptical, slightly curved, from 3·5 to 6 by 0·8 to 2 μ in diameter ; they are hyaline and embedded in mucilage which in moist weather is extruded with the spores through the ostiole, the spores being scattered in raindrops or perhaps borne away by insects. In dry weather the pink spore tendrils dry out, becoming firmly fixed to the substratum ; the spores remain viable in the dried mucilage for over 6 weeks, but without its protection survive for only 5 to 10 days, or less [4]. Different strains of the fungus are known to exist ; in artificial culture these differ mostly in their rate of growth and capacity for staling of the medium ; in general, the colonies are irregularly lobed, the aerial mycelium being of a dirty-white colour above, and slightly olivaceous within the medium (potato-dextrose-agar, at 20° C.) ; monospore cultures, kept moist, produced concentric rings of pycnidia in 10 to 24 days ; the optimum temperature for growth is 25°, and for pycnidial formation somewhat lower, 20° C. ; exposure to strong light favours development of pycnidia [2, 7].

There is general agreement that *Ph. lingam* survives from season to season on the decayed remains of infected crops in the soil, but it does not appear to be capable of long existence in the soil ; it is reported to live at least for one [10] or two seasons [5] ; in New Zealand it was isolated from a moist soil two months after the removal of the infected crop but was not recovered after that period unless associated with host debris [6]. Whether the organism of dry rot can also survive on the seed has long been a vexed question. There is abundant evidence that the seed coat carries infection (pycnidia) which may attack the seedling in its early growth, but according to some, seed infection occurs on too small a scale to account for heavy infections usually witnessed in the field. On the other hand,

in New Zealand, where the disease in many places has been a limiting factor in the raising of swede crops, infection is believed to start from seed, occurring there, too, on swede seed imported from Britain [4]. The severity of the disease in New Zealand is otherwise difficult to explain, but there is evidence from this quarter that certain insect vectors attracted by the mucilaginous spore tendrils may also distribute the disease [3, 11]. Furthermore, there is undoubted evidence that dry rot can start afresh on swede crops after many years of rotation, or on newly broken old pastures where there was no likelihood of the disease having been carried to the new crop in any other way than from infected seed, such as, for instance, by any weeds susceptible to it [7]. It seems fairly clear, therefore, that while in severe attacks the spread of dry rot is mainly from infected debris left in the soil, there can be little doubt that true primary attacks arise from the planting of infected seed. Probably one variety of swede is more prone to seed infection than another ; it is recorded that when seeds from two stocks of the same variety of swede were planted in clean soil, one stock gave 60, and the other only 1 per cent. of diseased roots [1]. Much disease probably ensues from a combination of infected seed and the use of manure contaminated with crop debris.

The various parts of the growing plant become infected from soil-level upwards by the spores of the parasite being splashed by rain and wind, the lower parts becoming first affected and, having formed pycnidia and spores, the latter are carried up a step higher from leaf to leaf, causing fresh infections and pycnidia until finally the pods, and from them the seeds, are in turn infected. Infections are thus external, occurring through lenticels, leaf scars or wounds, not, it is believed, through unbroken rind [14], but uninjured leaves are said to be invaded by the spores, pycnidia appearing within three days after the spots are first visible [9]. The fungus destroys the cells of the leaf, the affected part turning brown, and from groups of hyphae which collect at various points under the epidermis (upper and lower) pycnidia are developed [9]. In the neck and bulb, the intercellular mycelium spreads in the cortex, chiefly in a tangential direction, inward penetration being slower, but when the deeper-seated tissues are invaded the mycelium may also be seen inside the host cells ; cambial and phloem tissues may thus be occupied and the fungus may collect in masses near the xylem. Though pycnidia are early formed at points of infection, later, with the rupture of the lesions due to growth and expansion of the bulb, they continue to be formed at varying depths into the wounded tissues as well, but getting less and less black the deeper they are formed and becoming less efficiently enclosed because of the poor development of the pycnidial wall, since they are protected in crevices in the host tissues [14]. Bulb infection is always followed by death of the plant ; complete decay of large bulbs occurs towards the end of the season, when they dry out and become mummified [4]. The fungus is present in all the lesions on the pods, and after traversing the pericarp wall, may fill the interior of the pod with a flocculent mycelium which may penetrate the seeds, especially those in the vicinity of a lesion [4] ; all parts of a seed may be penetrated and destroyed, but mostly only the testa becomes affected [2]. As already mentioned, young seedlings may contract infection from the testa when this is carried up on the tip of one or other of the cotyledons at germination ; the fungus grows out of the testa into the cotyledons, to produce

a small grey or brown lesion, the tissues being destroyed in the same way as described above in the case of the foliage leaves. Even if affected seeds do not germinate, pycnidia may still be developed on the seed coats in a few days, and young seedlings which contract infection through the hypocotyl, if the testa is not carried up on the cotyledons, usually develop a rot which involves the entire seedling, and pycnidia may appear on any of its parts [7].

Dry rot appears on all types of soils. There is no evidence that severity of the disease is dependent upon high moisture content of the soil; in Scotland it is as severe in the dry north-east as in the wet south-west areas, so that its distribution appears to bear no relation to the amount of rainfall [7].

Since dry rot is mainly contracted through sowing in contaminated soil, suitable rotation should be observed, as practised, for instance, by the Lothian farmers who carry out a six-course rotation, the cruciferous crop following either potatoes or oats [7]. With regard to cultivation, the disease is said to be less severe when the roots grow close together, presumably because the dense overlapping foliage forms a canopy over the bulbs protecting them against rain-splashed spores [16]. In certain parts of New Zealand it is the practice of farmers deliberately to choose a smaller yield by sowing thickly and late, rather than risk the almost certain loss of a better crop obtained by sowing earlier with better spacing of the plants [11].

Various treatments to destroy the fungus on the seed have proved ineffective or impracticable on a commercial scale [6, 7, 11]. No variety of swede is reported to be resistant to dry rot in all localities under similar conditions. Swedes are generally more susceptible than turnips, yellow turnips showing distinct resistance [14, 15, 16].

For the protection of plants cultivated for seed, spraying with Bordeaux mixture is recommended [6]. The most promising line of enquiry towards the eradication of the disease is that of more rigid inspection and selection of seed plants exhibiting less and less tendency to canker and dry rot [7].

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Brown Heart or Heart Rot of Turnip and Swede (*non-parasitic*)

This important disease of turnips, swedes, and allied plants is due to boron deficiency. It has probably arisen through the diminished use of manure and the greater purity of artificial fertilisers, so that it has become necessary to add the

mineral to the soil [3, 4, 8, 9]. The disease is known in England, Canada, and New Zealand as brown heart or heart rot, in Scotland as ' raan ', and by various other titles in Scandinavia, Holland, and Germany. It is fairly common after dry seasons, and in calcareous soils.

Experiments reported from Canada in 1937 (the trouble was stated to be a limiting factor in the growing of swedes and turnips in eastern Canada), from Scotland, Wales, and Norway in 1935 showed that this disease was controlled by soil applications of sodium tetraborate (borax) and other boron compounds. In Canada, it is estimated that losses due to this disorder amount to about 50,000

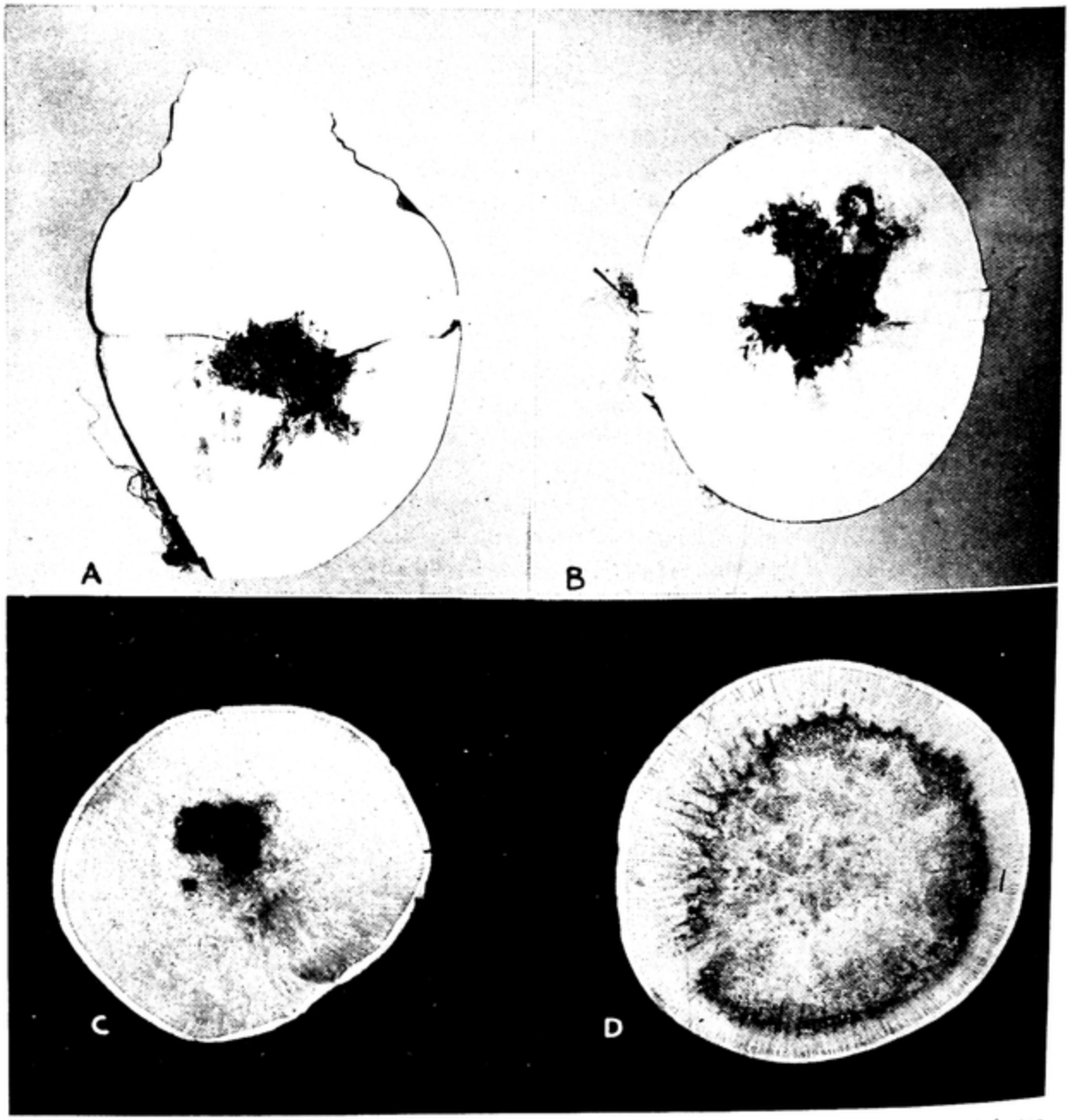


FIG. 280.—Brown heart of swede. *A*, in longitudinal section. *B*, in transverse section (photos by Foister & Noble). *C*, *D*, cross-sections of turnip showing brown heart after six months of storage (photo by O'Brien, by permission of West Scot. Agric. Coll., Auchincruive)

dollars every year [5]. The use of finely powdered borax, at the rate of 10 to 20 lb. per acre, shortly before sowing the seed is now a general remedy.

Brown heart, as seen when the ' bulb ' is cut across, is marked by the development of a well-defined, brownish, mottled, more or less watery area, not extending past the cambial layer, in the root (Fig. 280). No external symptoms are visible at this stage. As the turnip grows older the mottling becomes less noticeable, being replaced by a greyish-brown dry mass of broken-down cellular tissue, but there is no spreading rot. The trouble appears to begin, in seedlings, in the lower part of the tap root and to be associated with abnormalities of the xylem ; in older plants the middle lamella is swollen in the brownish areas. In swedes suffering from boron deficiency there is developed a wide zone of thin-walled cells from the cambium. Greater damage to the cell walls in initial attack was observed in the secondary vascular bundles, followed by the formation of thick-walled cells distinct from all the adjacent tissues [6]. Affected roots have a bitter taste, are woody when cooked, and their feeding value is much reduced, the loss in sugar being as much as 12 per cent. In Eire, in 1940, brown heart affected about 90 per cent. of swedes in south County Kildare on an alkaline light soil, and was controlled by the application of borax, spraying the crop in the third week of July, with the solution, reducing the amount of disease to a greater extent than making soil applications by broadcasting at sowing time, or by applying the compound to the side of the bulbs, at the same rate of 21 lb. per acre, while the spraying was effective at the rate of $7\frac{1}{2}$ lb. per acre [2].

Some varieties are relatively resistant but none appears to be completely so, and all may show symptoms under adverse soil conditions such as are induced by liming certain soils. In Welsh experiments, the variety Superlative showed marked resistance. In Great Britain the purple-topped varieties are in general more prone to the disease than those with green tops [1]. In New Zealand, Vilmorin and Wilhelmsburger swede varieties appeared to be somewhat more resistant to brown heart than Superlative and Sensation [7]. The brassicas are more sensitive to boron deficiency than beet, and their seeds contain too little to enable the cotyledons and first true leaves to develop ; swedes have been found in England to suffer severely from brown heart in acid soils alongside beets and mangolds which showed no symptoms [1]. Pot culture experiments at Cambridge have shown that turnips deprived of boron suffer from severe marginal scorch of the outer leaves, twisting, elongation and narrowing of the inner leaves, and stunting and roughening of the root, which tends to rot.

Applications of borax even as low as 3 lb. per acre have proved beneficial in New Zealand, but a lesser rate than 10 lb. cannot be relied on, and in highly calcareous soils 20 lb. or even more may be necessary ; an increased yield of $5\frac{1}{2}$ tons per acre has been obtained by an application of 15 lb. without causing injury to ordinary crops in subsequent rotations [5]. If lime is required for the root crop, or is applied to combat club-root disease (*Plasmodiophora brassicae*), an application of boron at the same time may be advisable.

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Downy Mildew of Beet, *Peronospora schachtii* Fuckel

Though known in Europe as far back as 1852, downy mildew of sugar beet did not appear in England until 1921, in the counties of Lincoln and Cambridge. It is believed to have occurred on mangolds in 1913 [1], and again, in 1925, on a mangold crop grown for seed in east Kent [8]. The first record of the trouble in the United States was in 1911 [10], on sugar beet in California, in which area much research has since been carried out in relation to various aspects of this disease [1a, 6, 7], and further investigations have also been made in France [9]. The mildew is prevalent in most European countries and has also been reported in Egypt, Palestine, Japan, the Argentine, and various localities in America.

Early seedling infections often account for complete loss of plants in the root beds, while in later attacks disease on the foliage may so interfere with normal plant functions as to cause considerable reduction in the amount of carbohydrate stored in the roots, a feature which naturally affects the plants more adversely in early than late infections [3]. Still later infections, incident at the time when beets grown for seed are in flower, may, by reducing the number of flowers in the inflorescence, or by otherwise reducing their fertility, cause serious losses in the amount of seed produced.



FIG. 281.—Downy mildew of sugar beet (*Peronospora schachtii*). The mildewed leaves at the crown (photo by Dillon Weston)

Symptoms of trouble in the root beds are manifest by gaps in the drills, thus indicating destruction at early stages of germination. Plants attacked after making good growth show thickened and stunted stems, accompanied by the development of several thin secondary shoots growing out from the sides of the crown (Fig. 281). The lower leaves on the main stem of affected plants are smaller than normal, thickened and twisted, and covered on the under side with the buff-grey conidial fructifications of the mildew.

It is not known with certainty how primary infections of the seedlings in the field arise, whether from infected seed or from soil contaminated with the resting spores of the causal fungus. During infections of seedlings, under moist conditions, the cotyledons curl downwards and soon become covered over with conidia,

the young plants usually dying off, but cotyledonary infections, though easily effected under artificial conditions, are of somewhat rare occurrence in the field. Infections commonly occurring on older, expanded leaves consist of isolated or united spots of irregular shape, from 1 to 4 cm. in diameter; on the upper surface the spots are a paler green than the rest of the leaf and are coated on the corresponding under side with conidia, and if the weather has been dry for an appreciable period the spots are usually surrounded by a narrow ring of a pale red colour. Infected plants which continue their growth into the autumn may show, especially after heavy rainfall, an abnormal increase in the number of young leaves in the central rosette. These young leaves are smaller than usual, puckered and covered with mildew towards the base of the lamina and petiole, but their tips usually remain quite unaffected, flat, and of a normal green colour [9]. The central rosette, though heavily mildewed, may often be surrounded by older leaves which to all appearances are healthy, but in severe infections all the leaves may be killed [6]. In beets grown for seed, the disease causes a decided check to the elongation of the inflorescence axis on which, towards the base, the leaves are again thicker than normal, curled, and heavily mildewed, and the axillary floral shoots may either be suppressed, or remain dwarfed and distorted. Floral shoots which survive to blossom may also show infection in the form of swellings or blisters, particularly on the bracts and on the sepals of the flowers; in place of flowers there is often such an abnormal development and dense assemblage of small foliage leaves as to give the floral axis the appearance of a densely foliaged vegetative shoot. But even in advanced cases of infection of the inflorescence, it is not unusual to find apparently healthy 'seed balls' (fruits) among sterile flowers which are covered with mildew, and many fruits containing germinable seed may carry conidial fructifications on the dried, protective sepals [6].

Downy mildew of beet is caused by the Phycomycete, *Peronospora schachtii* [2]. The organism produces both conidia and oospores but the latter have not been observed in all localities where the disease is known [4, 8]. The mildewed appearance of the leaves is due to emergent conidiophores (Fig. 282) which pass out at the stomata singly, or in groups of about three, but many more may be seen on mildewed cotyledons. The conidiophores are straight for about three-fourths of their length before they branch out in characteristic and regular fashion to produce ultimately from 5 to 40, or even more, hook-shaped sterigmata each bearing a conidium, the length over-all ranging from 200 to 500 μ [9], sometimes up to 600 μ [6].

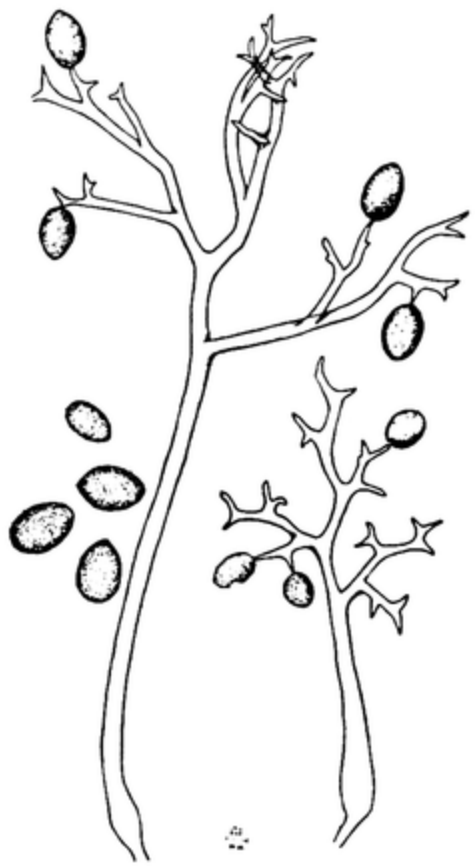


FIG. 282.—*Peronospora schachtii*. Two conidiophores and conidia (× 250) (after Salmon & Ware, Wye Reports)

The oval-shaped conidia measure from 20·3 to 28·1 by 17·5 to 24·3 μ [6], or show mean dimensions of 22 by 18 μ [9]. The multinucleate conidia germinate at comparatively low temperatures, 92 per cent. germination being obtained at 4° C., decreasing with higher temperatures of 10°, 21°, and 30°, to 75, 10, and 0 per cent. respectively [6], but an optimum temperature of 10° C. has also been observed [9], and slightly higher temperatures are required for sporulation [1a]. Although spore germination was reduced, conidia were not killed by freezing at −12° C. for 24 hours, and under natural conditions were enabled to survive periods of frosty weather [6]. Germination of conidia is mainly direct by germ-tube, but the formation of zoospores has also been observed [11]. The oospores are found mainly in the foliage leaves, but have also been seen in the floral organs, namely, sepals and pericarp of the seed clusters, and while their formation is apparently connected with maturity or death of the host, they may also arise in young leaves, and under artificial conditions have been produced in both cotyledons and young leaves within 30 days after inoculation with conidia [6]. The round, multinucleate oogonia are fertilised by paragynous antheridia; the oospores measure from 26·6 to 35·6 μ in diameter, and have a wall 1·2 to 3·4 μ thick. Some have succeeded in getting the oospores to germinate [11] but others have failed [6].

While the fungus becomes established mainly in the foliage leaves, it can also invade the entire plant, penetrating even into the vascular system of the crown and, to a lesser extent, the roots. Leaf penetration by germ-tubes is stomatal, and infection hyphae ranging from 4·4 to 11 μ in diameter [6, 9] ramify in the intercellular spaces to produce a somewhat coarse mycelium which enters the mesophyll cells here and there, to form large, branched, lobed haustoria (Fig. 82 G) [9]. The fungus concentrates much more in the spongy than in the palisade region of the leaf, so much so that, later, it becomes difficult to differentiate between these tissues of the leaf. Moreover, by hypertrophy, the leaves become considerably thickened, and meanwhile the affected parts of the lamina are of a much paler green than the normal parts, and the amount of carbohydrate produced in them is much less than in the healthy tissues.

The mycelium may be found in the leaf petioles, in the axis of the inflorescence, and in various organs of the flowers. While no sure connexion between mycelium in the older leaves and that in the crown has been established, continuity has, however, been detected between mycelium in the tissues of the crown and that found in the new leaves forming the rosette at the centre of the crown. Nevertheless, such new leaves in the rosette appear to become infected externally, from wind-borne conidia which settle and germinate on them chiefly along the tender petioles and succulent bases of the leaves. The probability is, therefore, that the crown becomes infected through the bases of the young leaves, and not from any mycelium that may have passed into it from older infected leaves. Again, while the first cauline leaves on the axis of the inflorescence often remain healthy, shoots and flowers in the axils of these leaves may be found severely infected. This is believed to be due to systemic infection, the fungus in the crown keeping pace with the elongation of the axis and the formation of the flowers, the mycelium being eventually found in the sepals, pericarp, and even in the stamens, at a time long before the flowers were open in the natural way for pollination and when external infection might also take place. Not only has the fungus

been found in the pericarp and sepals, but also in the integuments of young ovules, and in many cases oospores of the organism have been found in the sepals and pericarp, but in no instance has deep-seated infection of the nucellar and embryonic tissues been observed [6]. However, a contrary view is held, that floral infections, like those of the young leaves in the leafy rosette, are also of external origin from secondary infection by wind-borne conidia [9]. In any case, the undoubted observation of the presence of mycelium and oospores embedded in the coverings of the mature seed balls (beet ' seed ' is actually a fruit furnished with a thickened persistent perianth which is prickly and absorbent) is obviously of great importance in the possible survival of the fungus of beet mildew on the seed, whether infection originates from without from air-borne conidia or from within in a systemic manner from the crown.

Infection with conidia is effective over a wide range of temperature, from 5° to 20° C., the optimum being 8° C. [6]. Cotyledons and young leaves are highly susceptible, older leaves being more resistant. Infections in the field may appear on seedlings within a month of emergence and it is significant, in the case of a particular crop under observation, that all infections, with the exception of two, were caused by wind-borne conidia, the exceptions being considered primary infections from seed transmission or, perhaps, from the presence of oospores in the soil [6].

What form the fungus of downy mildew of beet may adopt for survival over the winter is not clearly known. In the case of beets stored for seed production there is ample evidence of the over-wintering of mycelium in the tissues of the crown, the mildew breaking out afresh when such beets are planted [4, 5]. We have seen that the fungus is capable of tolerating low winter temperatures, and there can be little doubt that by this method of beet culture perennial mycelium offers, at least, one way of establishing the fungus in the seed fields in the spring. The existence of oospores in the soil is another possible means of perennation, but these spores have not been observed in all places where the disease is common, and the conditions of the environment under which they are developed, and their germination made possible, are not known, and all attempts to induce infection of seedlings in soil deliberately contaminated with them have failed [6]. There still remains, therefore, the probability that the disease may be transmitted by seed. As already stated, evidence of the presence of mycelium in the pericarp of the seed ball and in the inner layers of the seed-coat has been established, with or without the formation of oospores. Possibly, from a few foci of infection set up by such infected seed, a number of primary infections may provide sufficient conidia to spread the disease to all parts of the crop by secondary infections.

As so many doubtful points have been raised in relation both to the survival of the fungus on the seed and the longevity and rôle of the oospores in decayed plant-remains or in the soil, it is obviously important to use seed only from disease-free plants, and not to plant a beet crop in land recently occupied by the same host. When transplanting, all plants showing any suspicion of the mildew on the leaves should be rejected and destroyed. Seed beds should not be planted near field crops of mangolds or beets, to avoid the possibility of transmission by conidia from one crop to the other. There is no definite evidence that any other member

of the beet family, except the wild beet (*Beta maritima*) found near the seashore, acts as a carrier host to this disease.

Out of a large number of experiments to test resistance to mildew [7, 9] only very few varieties of sugar beet showed any measure of resistance to downy mildew in the localities where these were performed. Spraying with Bordeaux mixture, or dusting with copper-lime, to check the disease in the root beds has been tried by some growers, but no definite evidence of the beneficial effects of these treatments is so far available.

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Black Leg of Sugar Beet, *Pythium de baryanum* Hesse ; *Phoma betae* Frank ; *Corticium solani* (Prill. & Delacr.) Bourd. & Galz. *Pythium* spp. ; *Pythium aphanidermatum* (Eds.) Fitzp. ; and *Aphanomyces levis* de Bary

' Black leg ' disease causes serious losses in fields of mangold and sugar beet during germination and early stages of growth ; older plants are generally safe from infection.

The disease has long been known to attack mangolds in Britain [28], and it appears also to have existed on beets in certain parts of England about 1895 or earlier, but it is not as serious a trouble of sugar beet in this country as in America [3]. In Britain, the three principal fungi associated with it are *Phoma betae*, *Pythium de baryanum* and other undetermined species of *Pythium*, and *Aphanomyces levis*, *Phoma* being the most important, but *Pythium* is reported to be the more common cause in south-west England [32]. In America, certain strains of *Corticium* (*Rhizoctonia*) *solani*, which are, apparently, different strains from those found in Europe, and another species of *Pythium*, *P. aphanidermatum*, are also named as causal organisms. In Germany, *Alternaria tenuis* is also cited [13a]. Most, if not all, of these organisms are considered to be only weak parasites, and there is reason to believe that certain factors pertaining to soil and climate, as well as other troubles of the sugar beet of a physiological nature arising probably from deficient nutrition, are actually the precursors of black leg disease and which render the weakened seedlings susceptible to attack by these various fungi [1, 37].

Early infection by any of these organisms may take place as soon as growth occurs outside the seed coat, or shortly before the young shoot emerges from the soil. On seedlings which succeed in breaking through the soil, the general symptoms consist, at first, of water-soaked spots on the young stem at or just below soil-level,

but as the lesions get bigger they turn brown, and later black. If infection penetrates as far as the vascular bundles the seedlings rapidly wilt and die; their roots are blackened and reduced to mere threads [5]. If the lesions go no further than the cortex, they usually heal up and the plants may survive [37]. Apparently it depends on the general vigour and rate of growth of the seedlings in comparison with the rapidity of penetration, whether invasion is held up in the cortex or is allowed to proceed into the vascular system.

The main structural and reproductive features of the fungi named above, and the symptoms caused by them separately in natural or artificial infection of beet, are outlined below, but as they produce so many symptoms in common it is often very difficult to ascribe certain phases of the disease to any one of them without resort to artificial culture [7].

Pythium de baryanum

While there can be little doubt that the effects of this fungus are mainly those of 'damping-off' of seedlings at soil-level, the same organism may also exact heavy toll of seedlings during pre-emergence [21], so that the latter form of this disease, which has long been associated with *Phoma betae*, is evidently not the work of that fungus alone. (Other species of *Pythium*, including *P. ultimum* [25a] attack beet and the latter is also known to cause a pre-emergent blight of tomato [23].) But in view of recent important work in America [5, 5a], corroborating much previous research in that country [2, 7, 10, 11, 12, 13, 29, 35] and in Germany [25, 30, 31], *Pythium* must rank first in importance, in relation to this disease, as a damping-off parasite. Damage to sugar beet in Iowa, caused by this fungus, is reported in certain seasons to be as high as 95 per cent. of the crop, and it is not uncommon to find fields in this area with stands of less than 50 per cent. nearly every year, and the trouble is sometimes so serious that entire fields have to be abandoned for growth of sugar beet owing to high mortality of seedlings [5]. This fungus, a member of the *Peronosporaceae*, is well known as a parasite attacking seedlings of cruciferous plants, but it is also associated with diseases of such diverse nature as a 'root rot' of conifers [17, 34] 'stem rot' of Pelargonium [4], 'fruit rot' of pepper [27], and in some areas it is responsible for poor stands of oat and barley [20].

Although its parasitic propensities are undoubted *Pythium* attains its maximum development and reproductive activity as a saprophyte. The mycelium is both intra- and extra-matrical in its relations with the host; the hyphae, 3 to 4 μ wide, are irregularly branched, at first continuous and crowded with dense protoplasm in which fat and glycogen are abundant; at a later period septation usually occurs and the time of formation of septa depends entirely on the supply of nutrition available, for if this is scanty the basal parts of the hyphae during their growth become emptied of protoplasm which passes on to the apical portion and septa are formed to cut off the full from the emptied parts. Cross-walls in the vegetative hyphae may thus arise at any time apart from their appearance at the development of the reproductive organs [6].

P. de baryanum forms two kinds of spores, namely sporangia (which may also function as conidia) and resting oospores. The spherical or slightly ovate sporangia, 10 to 35 μ in diameter, may arise in a terminal or intercalary position on the mycelium (Fig. 283).

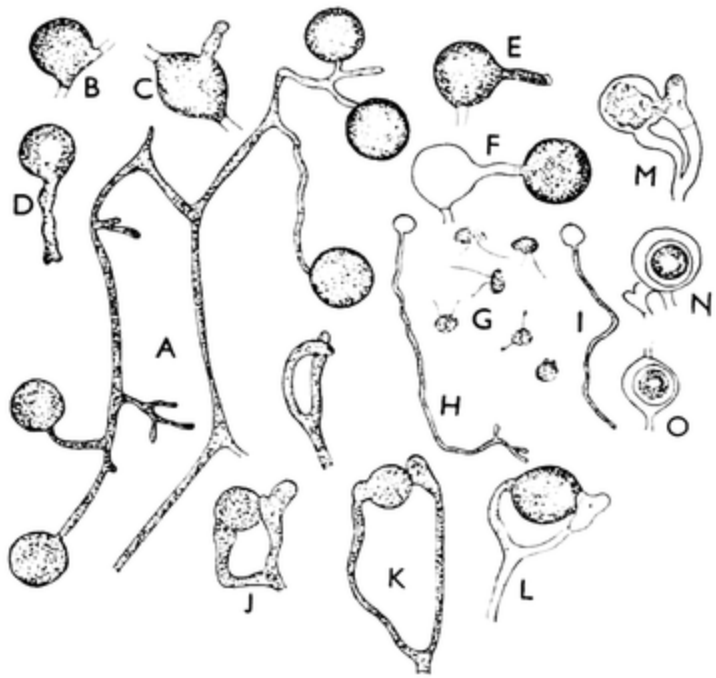


FIG. 283.—*Pythium de baryanum*. *A*, the coenocytic mycelium bearing terminal sporangia. *B*, an intercalary sporangium. *C*, the same, germinating. *D*, liberated sporangium germinating direct. *E*, early stage of indirect germination of sporangium, forming, as in *F*, a vesicle into which the contents are passed to form zoospores. *G*, the liberated zoospores. *H*, *I*, zoospore germination. *J*, *K*, *L*, the sexual organs, spherical oogonia with club-shaped antheridia. *M*, *N*, *O*, stages in the formation of the oospore (all × 250) (after Butler)

It is merely a question of environment whether these asexual spores behave as conidia or become sporangia, to germinate, in the former case directly by formation of germ-tubes, or in the latter, indirectly, by developing zoospores. The conversion into sporangia may be observed by bringing the mature spores into fresh, well-aerated water exposed to light. The contents of the sporangium are discharged into a protruding vesicle within which a number of kidney-shaped, biciliate zoospores are developed [6, 38]. After release, the motile zoospores come to rest, round off, develop a membrane and germinate to produce a branching mycelium. The conidia, as such, are capable of direct germination after many months' rest [6]. The sexual organs, oogonia and antheridia, usually appear at a later stage than conidia or sporangia but there is really no relation in time; they may arise either within or outside the substratum. In position on the mycelium, terminal or intercalary, and in the early stages of their formation, oogonia resemble sporangia, but have a smaller range of diameter, from 16·6 to 22·1 μ. Paragynous antheridia, of which one or more may invest an oogonium, may arise in various positions relative to the latter, either from the same hypha bearing the oogonium or from a neighbouring branch. Following fertilisation by a conjugation tube, each oogonium produces a single spherical oospore; oospores, with smooth walls varying from 1·5 to 2 μ in thickness, average from 13·16 to 17 μ in diameter. On the decay of the host the oospores pass into the soil where they are capable of wintering, and on germination may give rise directly to germ-tubes, or indirectly to zoospores, either of which, by coming into contact with the hypocotyl of germinating seedlings, bring about fresh infections.

The first sign of infection by *Pythium* is a glistening, water-soaked spot on the hypocotyl, about $\frac{1}{2}$ to 2 inches below the surface of the soil, according to the moisture-level and depth of planting. Penetrations are usually quite localised, either at the collar region, directly into the base of the hypocotyl, or into the upper parts of the roots close to the collar. The infections bore quickly through the outer layer of the hypocotyl or root and, entering between the cortical cells, cause the latter almost immediately to lose turgescence and, following upon shrinkage and softening of the hypocotyl, the seedlings collapse.

In mixed inoculations with *Pythium* and *Phoma* (which may be effected by introducing a culture of *Pythium* into sterilised soil, and planting in it *Phoma*-

infected seed) it is the general observation that if *Pythium* is abundant and active, the seedlings may be destroyed before *Phoma* makes any progress, so that *Pythium* appears to act more quickly than *Phoma* [12, 13, 30, 31].

Under conditions of uniform soil moisture *Pythium* is more active at comparatively high than at low temperatures. A strain of the fungus attacking cress in Wisconsin caused infection in over 80 per cent. of the seeds and seedlings, over a range of 20° to 30° C.; at lower temperatures the percentage was lower [22]. Moreover, when temperatures are high, growth of seedlings is so stimulated that the hypocotyls, drawn and etiolated, are rendered more sensitive to attack, and damping-off inevitably follows. Under conditions of low temperature, however, the fungus is checked and growth of the host is retarded, but the plants, though small, remain healthy. The fungus does not thrive in cold soils, and is most destructive in the fields when seeding has been done late and soil temperatures are still high [12]. In general, temperatures of 15° C. and below, are more unfavourable to damping-off than to seed germination and emergence, a conclusion which upholds the experience of growers of the benefit accruing from early planting of sugar beet [5]. A high moisture content of the soil is highly conducive to the incidence of damping-off, and the greatest losses occur on heavy soils having poor natural drainage [20]. A soil deficient in organic matter is usually more heavily contaminated with the fungus than a rich soil of a loose and friable texture [5].

Pythium de baryanum is eminently a soil-borne organism and is of common occurrence in field and garden soils, in which it survives in the form of oospores. The survival of the fungus in the soil suggests a method for its eradication by steam sterilisation, but on a big scale the method is obviously impracticable and the risk of re-contamination too great. Although direct treatment of the seed with fungicides is of doubtful value in reducing the activity of soil-borne organisms, yet such treatment often produces better stands than any from untreated seed, as was the case when beet seed was treated with 5 per cent. ethyl-mercuric-phosphate, (using 4 to 7 oz. of the phosphate to 100 lb. of seed), but this method is not advised in late plantings on soils of acid reaction. In general, a combination of seed treatment as above, with early sowing, helps to improve the stand in sugar beet [5]. In Virginia and Ohio a rotation with corn lessened damping-off, and improved sugar beet stands, whereas sweet clover or alfalfa aggravated the trouble [8]. In Minnesota, again, seedlings suffered more after planting of lucerne or sweet clover, and less after maize [26]. Treatment of beet ' seed ' with strong sulphuric acid, sufficient to decorticate them, was found to render the seed more readily germinable [17, 18].

Phoma betae

The relations of this fungus to sugar beet disease have been long established [12, 14, 15, 16, 25]. The organism is a member of the Sphaeropsidales (Fungi Imperfecti), the only fructifications being pycnidia. On the host, the round black pycnidia are present mostly on spots on leaves and petioles, and on lesions on the crown of the beet; they are not common on the fruits or ' seed clusters '.

The pycnidia are sub-epidermal, globose or lenticular, ostiolate, the pycnospores being extruded in a tendril. The fungus is readily cultivated and has long vitality on a

wide range of media. Pycnidia vary considerably in dimensions, from 125 to 635 μ in diameter ; the small hyaline pycnospores measure from 3·8 to 9·4 by 2·6 to 4·3 μ [12].

An ascigerous form *Pleospora betae*, discovered in Sweden on host debris left in the soil, has recently been claimed as being the perfect stage of this fungus. The hemispherical, black fruiting bodies, 230 to 340 μ in breadth, and 160 to 205 μ in height, arise on sub-epidermal stromata ; they contain ascospores, 19·5 to 25 by 8·5 to 10 μ, which are transversely and vertically septated [3a, 3b].

Phoma betae is usually carried by the seed, and the organism is said to be ever present on samples of commercial sugar-beet seed [24, 37]. It is now well established that *Phoma* cannot maintain itself in the soil for long unless it can live saprophytically on remains of beets in the field [31, 33], and under such conditions it is not improbable that it can start fresh infections when the same area is sown with sugar beet the following season [32]. The fungus is known to hibernate on the various coverings of the seed in the form of mycelium, but its presence actually within the seed coats has not been satisfactorily established, and whether infection from affected mother-beet into the seed-clusters takes place from without, or from within by systemic infection, is not definitely known. Though some have observed the pycnidia on seed-clusters, they are not common, and cannot in all cases account for the recognised high degree of infection usually harboured by seed of sugar beet. It is not improbable, however, that pycnospores from pycnidia on other parts of the host may be conveyed to seed-clusters, and finding there suitable conditions for germination, produce a resting mycelium. Under enclosed, moist conditions the latter may develop, and, in culture, two kinds of reproductive bodies, one, of the nature of thin-walled oidia, and the other, of hyaline round thick-walled chlamydospores, have been observed, both capable of producing mycelium in culture. Necrotic lesions on seed-beet stems have in some instances yielded such chlamydospores, from aerial mycelium [3b].

With the planting of infected seed, *Phoma* may start its attack as soon as the seeds begin to germinate, and the young germling may be destroyed forthwith before it emerges from the ground. But most commonly the first symptoms occur about the third day after the seedlings are through the ground, and may continue till the time they develop their third pair of leaves [12]. Early infection with *Phoma* is associated with a browning and blackening of the hypocotyl, the discoloured part being evident above soil-level before the seedling falls over. *Pythium* destroys more rapidly than *Phoma*, and while *Pythium* kills the cortex early and is soon deeply in the vascular tissues, *Phoma* though also highly destructive to the cortex does not, as a rule, enter the vascular region of the seedling. The general impression remains that there is a higher percentage of survivals amongst seedlings attacked by *Phoma* than by *Pythium*. Seedlings infected by *Phoma* despite loss of primary roots, may send out new roots and succeed in making good growth up to maturity, and yet may still carry infection in the tissues of the crown. Such beets may be placed in store, to all appearances healthy, but when planted out again for seed production may be the means of starting infection in the seed all over again [12].

When such infected beets from store (' stecklings ') are planted out, the parasite revives and travels from the crown to the leaves, on which, as well as on the petioles,

spots ultimately bearing pycnidia arise. The appearance of the spots is almost invariably followed by infection on the seed-clusters, at about the time they are ripening [37]. But, as already indicated, whether the infection reaches the clusters from within the parent beet systemically, or from without, by the splashing of pycnospores from pycnidia on leaf, petiole, or crown, is not clearly known, but the fact remains that the fungus which has found its way into the seed-clusters starts the cycle afresh when seed germination takes place [12]. Recent observations in Sweden have shown that necrotic lesions on the stems arise through contact with infected leaves in wet weather, while infection may also reach the stem direct from leaf through petiole; infection from over-wintered asci setting free their spores in the following summer is provided throughout the growing season in that country [31b].

Black-leg disease, traceable to this organism, is worse when the climatic conditions during germination are cold and wet [32]. In relation to soil reaction it is recorded in Switzerland that the fungus attacks the young plants only when the soil is alkaline [19]. In Sweden, environmental factors seemed to exert little influence, the disease, due wholly or in part to *Phoma*, being equally severe both in acid and alkaline soils [31b].

The seed-borne nature of *Phoma* disease indicates the importance of clean seed for planting. Hot-water treatment is not unattended with risk, for though the fungus can withstand as high a temperature as 60° C., the seed suffers injury [7, 12]. Various seed treatments with cupric and mercuric compounds are now recommended, disinfection being done preferably in small lots [24, 32]. Good results are reported with cresolsodium mercuric cyanide, applied at a strength of 3·125 per cent., 1 quart being used for 12½ lb. of seed; the treatment is carried out in a mixing machine, for 3 minutes, and the seed thereafter spread out on the floor to partially dry before sowing [37]. As the period of susceptibility to this organism is relatively brief in the seedling stage, a great measure of control can be exercised by attending to good cultural conditions so as to encourage vigorous germination. In good soil, with proper attention to drainage to ensure good aeration, germination is greatly stimulated and the seedlings pass quickly through the period the hypocotyl is susceptible to attack [12].

Rhizoctonia solani

The perfect stage of this fungus has not been found on the sugar beet. (The organism has already been described in connection with 'black scurf' of potato (p. 528).) Though a soil-borne fungus, symptoms of black leg attributed to *Rhizoctonia solani* are more like those caused by *Phoma* than by any species of *Pythium*. It differs, however, from both of these fungi in its capacity for attacking sugar beet over a much longer period of seedling growth, from the earliest stages of germination up to stages 4 to 5 weeks old [12]. Like *Phoma*, its action within the host is somewhat delayed, and while it causes a high percentage of disease, in comparison with both *Pythium* and *Phoma* it is slower in producing its effects and there is usually a high percentage of partially affected plants which make good recovery [7]. In America, where it is a very destructive crown rot of sugar beet, it is not uncommon to see entire fields of 50 to 100 acres practically ruined by this organism [10, 11, 12, 29, 35].

Penetration of the hypocotyl is followed by the appearance of dark-brown or chocolate-coloured lesions [5]. Growth is checked, and the tap root is often destroyed at the tip, but new roots which are developed above the decayed region frequently assist the plants to recover. Indications of attack by *Rhizoctonia* on older seedlings may sometimes be seen in an abnormal depth of green, or blue-green colour assumed by the foliage, and associated with this feature is a lemon-yellow discoloration of the stem [7]. In hot weather the fungus attacks the base of the leaves; there is no definite spotting of the lamina as in *Phoma* attacks, but the prostrated leaves on the ground finally wilt and turn brown. When the fungus has entered the crown, this part also turns brown and develops deep cracks on the surface, lined with mycelium emanating from the tissues of the crown.

Like *P. de baryanum*, *Rhizoctonia* is injurious to sugar beet at comparatively high temperatures, and partial or complete recovery may take place during periods of cool, dry weather [7, 12]. Clay soils and those deficient in organic matter favour the growth of this fungus [12].

Methods recommended for the control of black-leg disease due to this fungus are similar to those adopted for *Pythium*. *Rhizoctonia solani*, possessing very numerous strains, has a wide range of hosts and, in certain parts of the United States, clover, vetch, alfalfa, as well as potato are attacked (the strain attacking potato in Britain does not infect the sugar beet in this country), but it is not known whether the strains attacking these various hosts are also parasitic to sugar beet. It is obviously safer to avoid these hosts in rotations with beet; when corn is grown in these parts of America in the rotation, infection with this fungus is lessened [7].

Pythium aphanidermatum

In America, four fungi have been found to attack seedlings of sugar beet; they include the three above described, the fourth being a "fungus originally reported to be *Aphanomyces levis* but which has since been found to be new, and to which the name *P. aphanidermatum* has been given" [12, 13].

In its association with this disease of sugar beet, this species produces symptoms very similar to those caused by *P. de baryanum*, but it is a much more vigorous and destructive parasite than the latter, so much so that it is not usual for attacked seedlings even to break through the soil, and seedlings never recover from attack. It also causes disease in radish, cucumber, and melon [9].

In its method of asexual reproduction this organism differs strikingly from *P. de baryanum* and resembles more that of *Aphanomyces* (see below). The non-septate mycelium of branching hyphae, 2·8 to 7·3 μ wide, forms long tapering zoosporangia containing, not zoospores, but cells in which zoospores are formed. These cells, after liberation, function each as separate zoosporangia, discharging their contents into a vesicle in which numerous zoospores are finally developed. The released zoospores round up, increase in size, become walled, and germinate to produce mycelium. In the sexual phase, the oogonia are terminal on branches of the mycelium, spherical, 22 to 27 μ in diameter; antheridia are paragynous. The oospores 17 to 19 μ in diameter, produced singly in the oogonia, are spherical with a smooth or undulated wall, 1·5 to 2·5 μ thick. Germination of the oospores was obtained in water cultures containing young, sterilised

beet seedlings, and it is believed that they serve to tide the organism through periods of drought [13].

Methods of controlling this parasite in its association with sugar-beet disease are of the same order as prescribed for *P. de baryanum*.

Aphanomyces levis

This fungus was first reported in 1906 in parasitic association with *P. de baryanum* and *Phoma betae* to cause considerable damping-off of sugar beet in Germany [30]. Like *Pythium*, it is a soil-borne organism, and in its asexual reproduction develops long narrow zoosporangia similar to those of *P. aphanidermatum*, and the zoospores are said to lose their cilia before liberation. Sexual reproduction occurs as in *Pythium*, smooth-walled oospores being produced. The life-history and structure of this fungus are similar to those of *Aphanomyces euteiches*, described in this book in connection with a root rot of peas (p. 609).

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Heart Rot of Sugar Beet (*non-parasitic*)

Heart rot of sugar and other beets is a non-parasitic disease attributed to a lack of boron in the soil. Although the actual cause was not known, the trouble has long been prevalent in England on mangolds, and was very evident in this country in 1928 and 1929 on sugar-beet crops grown on alkaline soils [11]. It is still fairly common in some parts of Britain [10]. In Belgium, where extensive crops of sugar beet are grown, the disease causes losses up to 30 per cent. of the crop, and is more troublesome on scanty-leaved varieties distantly planted than on varieties with a luxuriant foliage [5, 6]. In Holland, a symptom described as ' vein rot ' believed to be due to boron deficiency may develop into the more serious heart rot unless checked by appropriate soil treatments [14]. The trouble is also well known in America [4, 8, 9, 13, 15].

Early symptoms of the malady always occur on the youngest leaves of the crown. These heart leaves, instead of growing compactly, spread outwards, and may show characteristic dark-brown scurfy patches chiefly on the inner, concave surfaces of the leaf stalks. This brown discoloration may extend to the midribs and spread into the lower lateral veins, while the stalks become brittle. The heart leaves wilt and turn yellow and the veins change in colour from white to yellow, and the leaves finally blacken and perish. If unfavourable conditions persist, the older outspread leaves may also decay. In Holland, local swellings on the midribs and lateral veins with longitudinal fissures in them have been observed, and sometimes the trouble here seemed to be confined to a few leaves only, the remaining leaves being normal [14]. In western Oregon [13] plants grown for seed showed a dwarfing of the inflorescence axis, with much distortion of growth, due to the death of some of the lateral floral shoots, the surviving shoots developing into a witches' broom type of growth [13]. In severe affections, blackening of the axis and death of the stem apices and flower buds may occur [2]. The bulbs and roots may also show a diseased condition. The tissues of the crown become discoloured brown or even black, shrink, and die. The discoloration may spread from the crown into the root and, if the latter is cut across, a brown stain is found both in the vascular bundles and the medullary rays. Except in severe attacks, the browning of the tissues is not very pronounced and is usually confined to the outer layers of the root, but in bad cases may spread into the inner tissues as well [11]. The histological effects in young seedlings of garden beet in Wisconsin subjected to deficiency of boron were first seen in the phloem cells of the root and hypocotyl, this tissue being filled with a dense substance, and there was also some hypertrophy of the cambial cells. The suggestion is made that boron deficiency may so affect the metabolic processes as to interfere with cell division and development, and thus hinder the important process of laying down adequate food reserves in the roots [8]. Similarly, table beets have been seen to react to boron deficiency by producing gummy deposits both in the intercellular spaces of the petiolar parenchyma and within the vessels of the petioles and laminar veins [9].

Numerous tests have shown that sugar beets remove considerable quantities

of boron from the soil [6]. While the application of the mineral has proved of undoubted value in the healthy growth of sugar beet and other crops, it should not be applied indiscriminately and should only be given when it is known that the soil is in need of it. To this end an analysis of the soil, especially in relation to its lime content, is advisable. There is abundant evidence that the availability of boron is closely connected with an alkaline condition of the soil, and it appears to be highly necessary that the operation of liming should be carried out at some other time in the rotation than immediately prior to planting of sugar beets [10]. It is customary to apply the mineral in the form of borax, at the rate of 21 lb. per acre, or 10 to 20 kg. per hectare. The application may be made as a top dressing, though it may be found advisable to mix the compound with sand or dry soil, or the crop may be sprayed with a solution of 10 to 20 kg. per 1000 litres per hectare of the crop [6, 7, 15].

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Speckled Yellows of Beet (*non-parasitic*)

This disease of sugar beets and mangolds is due to manganese deficiency. 'Speckled yellows' is fairly widespread in England, and has comparatively recently been reported on garden beet near Bristol and on sea-kale beet in Somerset [3]. In Holland it is responsible for considerable losses, in some years as much as 6 or 8 per cent. of the yield.

The young leaves of beets suffering from this disorder become chlorotic in the interveinal spaces; star-shaped yellow spots develop in these areas and are most visible in the outer leaves, where they may result in cavities in the leaf. The affected plants are markedly upright in habit and the edges of the leaves curl inwards, giving the leaf a triangular shape [1].

Speckled yellows is particularly troublesome on old alluvial soils, or heavily limed sandy soils, rich in organic matter. In some parts of England it is limited to a few districts on light sandy or gravelly soils or on light fen soils with a peaty subsoil [3, 5]. The trouble can be controlled by soil applications of manganese sulphate at the rate of 50 or 60 kg. per hectare. In England, increases in total sugar of 5 cwt. per acre, with an application of 50 lb., and of 8·1 cwt. with 150 lb. per acre, have been reported, the yield of tops being also increased.

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Beet Mosaic

This virus disease of sugar beet is found in all parts of Europe where the crop is grown, and is present also in several localities in the United States [8, 9]. Spinach, seakale-, fodder-, spinach-, and red beets, as well as mangolds are all subject to this virus. The wild beet (*Beta maritima*) of the seashore, and the white goosefoot (*Chenopodium album*) are also susceptible [5, 6, 16].

While in many areas the losses in yield and sugar content of the root crop is not usually very heavy, appreciable reductions may be experienced in the same areas, in the second-year crop grown for seed ; observations in North Colorado showed a difference of nearly 35 per cent. in the yield of seed, between plots planted with diseased and healthy roots [5].

The symptoms are highly variable during the growth of the plants. At least four types are distinguished [9] : (*a*) a mottled appearance of the leaves, due to the presence of light-greenish or yellowish-green areas over the whole leaf ; (*b*) yellowish areas confined to the regions near the veins, the interveinal areas being normal green ; (*c*) the interveinal areas light-green or yellowish whilst the regions near the veins are of a normal dark-green colour ; (*d*) the leaves of a yellowish colour, with dark-green areas of irregular size distributed over the surface. In addition to these very variable symptoms [16] there may be a certain amount of puckering or crinkling of the leaves, and a characteristic feature is a bending back of the older leaves near the tips, which is frequently followed by a curling of the margins before the leaves die off at the tips. In severe infections growth is retarded, the leaves may be distorted and rolled inwards, and the plants present a stunted and deformed appearance.

Synonyms for the virus (*Beta virus 2* Smith) of beet mosaic are : *Beet yellow virus* (Prill. & Delacr.) ; *Beet mosaic virus* (Lind) ; *Sugar beet mosaic virus* (Robbins 1921, Hoggan 1933) ; *Sugar beet virus 2* (J. Johnson's classification) ; and *Spinach beet mosaic virus* (Böning 1927). The virus may be transmitted by sap inoculation ; it is not soil- or seed-borne. The insect vectors responsible for its spread are *Myzus persicae*, *Aphis rumicis*, and *Macrosiphum gei*. It does not persist for long in the vector, so that it becomes necessary to visit a fresh source of the virus. The ' incubation period ' is reported to be from 8 to 16 days, and the virus is harboured over the winter in weeds, and in beets stored for seed production [6].

The histopathological effects of the virus in the leaves may be seen in a less marked distinction between palisade and spongy mesophyll than is found in normal leaves, with a reduction in the size of the intercellular spaces.

Sugar-beet mosaic may be kept under control if means are taken to eradicate the sources of infection where the insect vectors flourish. All infected beets or mangolds should be removed and weeds kept under control. A root crop grown

near a seed crop usually suffers severely owing to aphis infestation of the latter. Loss of seed may be reduced by early planting. Liberal manuring and avoidance of phosphoric acid deficiency should ensure healthier crops [1].

Beet Yellows

This virus disease of sugar beet, though the virus was not identified until 1940, has probably been known in commercial crops in Britain over a long period [9, 10, 20]. It is common throughout Europe and exists also in the United States [2, 14, 17, 18]. Losses in the Belgian crops due to this ' virus yellows ' were estimated in 1939 to be 46,422,700 fr., an increase of over 16 million fr. over the losses experienced three years previously ; and if the losses due to the same disease on fodder beets are included, a total of 60 million fr. is estimated as an average loss every year in this area [4]. There is a significant reduction in the average weight of the leaves and roots of affected plants, as well as in the sugar content of the roots [12]. Other plants susceptible to the virus of beet yellows are spinach, mangold, spinach beet, seakale beet, *Chenopodium album* and *C. purpureum* [13].

The disease may appear in the field in patches of diverse size, in which practically all the the plants may be infected, or affected plants may be found at random over the crop, or in groups of a few together. This irregular distribution of diseased plants in the open may probably be accounted for if these segregated groups of diseased beets are actually the first infections in the crop. Such initial infections have probably followed upon the visitation of the winged forms of the insect vectors which migrated from one or more sources of infection in the vicinity. With a comparatively few foci of infections thus established, infections thereafter may become more or less general throughout the crop. The chief source of infection is the seed crop, in the second year of growth, and the large number of the winged migrants produced on it carry the virus to the root crops in the near vicinity. It is further known that the infecting principle of ' beet yellows ' is harboured through the winter in weeds, and in the roots of seed bearers and wild beets [12]. The spread of the virus in the root crops is probably by the walking forms of the aphides, though the winged forms found towards the end of the season may be responsible for conveying the virus again into the ' steckling ' seed beets, and so infection is ensured from year to year [20]. The virus is not transmitted through the seed or by sap inoculation. It has been ascertained in Belgium [15] that silos and feed-lots where beet remains may often be found in a sprouting condition, afford good feeding grounds for the vector *Aphis rumicis* before migrating to the fields. The peach aphis *Myzus persicae* is also known to be a very efficient vector, and it does not appear that an ' incubation period ' is necessary, for half an hour on a healthy beet was found a sufficient sojourn for the production of infection, though the infectivity of the vector was increased greatly by the length of feeding time on the infected source and on the plant infected. In Holland, *Macrosiphum solanifolii* is also said to be a vector of the virus of sugar beet yellows [14].

Symptoms of the disease in the crop do not usually appear until about mid-

season. These become apparent on the older leaves, the young heart leaves being little affected. The symptoms are, however, highly variable, differences being apparent on leaves of different sizes, or in the severity of the symptoms, and varying according to the dates of infection. Early infections in June and July are usually characterised by severe stunting and necrosis, whilst August infections show these features to a lesser degree. As the plants grow to maturity towards September or soon after, there is barely any stunting or necrosis and the symptoms are merely localised. It is probable that the characteristic attacks of sugar-beet yellows occur in August and September, in well-grown crops. While later infections are not uncommon, they seem to have little effect upon the general growth of the plants, and a colourful feature presented by the foliage of these late-infected though still well-nourished and well-grown beets, simulating a ' false ripening ', is in marked contrast to the appearance of the unaffected beets which remain green until harvest [20].

The discolorations developing in affected leaves vary from pale-watery or greenish-yellow to a rich orange colour, or red in some varieties of sugar beet. These chlorotic areas of the leaves are also thickened and brittle, and crackle when broken. They also have a waxy or dry texture and the plants rustle as one walks through the crop. The discoloration in a leaf may start at any point, in early infection beginning at the tip, spreading along the interveinal areas, but sometimes covering the veins as well ; or, the mottling, starting at the margin, may be confined to a half of the leaf. Early leaf infections nearly always result in necrosis and frequently afford entrance to fungal organisms. Chlorosis all the way down the leaf is followed by necrosis, and in dull or cold weather the latter may so outpace the former that the typical yellow areas are masked over, a condition not unlike the symptoms presented by sugar beet suffering from potash deficiency. With a spell of bad weather succeeding good growing conditions, or when plant growth is checked in some other way, spread of necrosis is so rapid that all the infected leaves die and the plants perish [20].

It appears that the leaves of sugar beet affected with ' yellows ' are more prone to become rusted with *Uromyces betae* than the healthy leaves. Affected leaves contain a higher percentage of the sugars hexose, sucrose, and maltose, than the healthy leaves, and are also said to transpire less freely, by virtue presumably of the stomata being less widely open than in normal leaves [17]. Though the diseased leaves manufacture their starch less rapidly than healthy leaves, they come to contain a higher percentage of it, owing to its accumulation, and not to an enhanced photosynthetic activity [13]. Its retention in the leaves is a consequence of a disturbance in its translocation due apparently, at least in part, to a gummosis of the phloem [12, 17, 18]. The symptoms of sugar beet yellows are said to be favoured by strong light and dry conditions [12]; they are less severe in low light intensities, and do not usually become apparent in the field in dull weather, and do not develop in the glasshouse during the winter months, though these conditions in no wise interfere with the growth of the plants [20]. Experiments in Holland showed that the symptoms were more pronounced at moderate (17° C.) than at comparatively high (30° C.) temperatures ; in a soil temperature of 25° C. the disease was less in evidence than at 12° C., due probably

to a stronger development of the foliage at the higher temperature. Differences in humidity relations do not appear to influence the course of the disease. Furthermore, in a series of experiments in Belgium no correlation could be found between the physical properties of the soil and the incidence or severity of beet yellows; an increase in the nitrogen supply appeared to mask the symptoms of the disease [13].

A system of manuring, with nitrogenous fertilisers, to balance the potash and phosphorus supplies, should be observed to increase vigour of growth. On a well-grown crop it appears that the aphides find greater difficulty in establishing themselves than on small immature plants [20]. Early sowing of non-bolting varieties of beet is preferable to late sowing, which with poor cultural conditions and proximity of the seed crops to the root crops, encourage aphis infestation. Every effort should be made to eradicate all sources where the insect vectors may multiply [7a, 12]. Groundkeeper beets and infected debris from mangold clamps are frequent sources of over-wintering infection and should be cleared away. As the infected seed crop is the most serious menace to the health of the root crop, stecklings for seed production should be obtained from an aphis-free area and planted as far as possible from root and seed crops.

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Chapter XIII

DISEASES OF PULSE CROPS

Rust of Broad Bean, *Uromyces fabae* (Pers.) de Bary

THIS autoecious rust occurs on peas, broad beans, and vetches. Whitish spots which soon turn brown appear first on the leaves and later on the stems. They bear the reproductive spores. The uredospores and teleutospores are developed somewhat late in the season; spermagonia and aecidia are very infrequent but have been found on the leaves of the broad bean [3]. The rust has the effect of causing partial defoliation but usually the injury is slight.

The uredosori are developed on both sides of the leaf as well as on the stem and petioles. They are arranged in small circles, and present a powdery, light-brown appearance (Fig. 284). The uredospores are round to ovate, spiny, with 3 or 4 germ-pores, and measure from 20 to 30 by 18 to 26 μ (Fig. 285 c). The teleutosori also occur on the leaves but mostly on the stem and are of a dark-brown, almost black colour. The teleutospores are sub-globose, ovate, or elliptical, with the apex rounded or flattened and the



FIG. 284.—Rust of broad bean (*Uromyces fabae*). The sori on the leaflets

wall greatly thickened at this point. They are smooth, brown, and measure from 25 to 38 by 18 to 27 μ. The stalk is persistent on the fallen spore and is pale-yellowish brown, thick, and up to 90 μ long (Fig. 285 D).

The aecidia and spermagonia (Fig. 285 A, B, F) develop on all green parts of the plant, chiefly on the under surface of the leaves. They appear in yellow spots, singly or in round or elongated clusters. The peridium is short, whitish, cup-shaped, and is furnished with an indented reflexed margin. The aecidospores are round to angular, or elliptical, yellow, with fine warts, and measure from 14 to 22 μ in diameter.

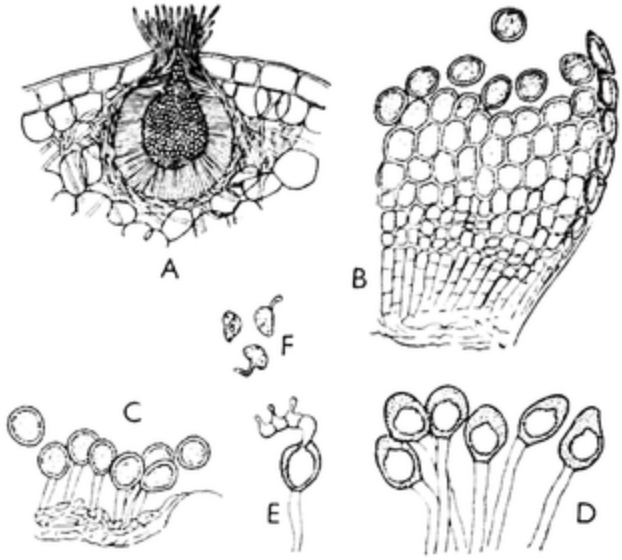


FIG. 285.—*Uromyces fabae*. *A*, spermagonium (× 50). *B*, part of an aecidium (× 215). *C*, part of uredosorus (× 215). *D*, teleutospores (× 215). *E*, germination of a teleutospore, and *F*, of sporidia (× 200) (*A*, *F*, after de Barry; *E*, after Plowright)

The optimum temperature for germination of the uredospores ranges between 16° and 22·5° C. and the highest production of these spores on the broad bean occurred at 14° to 24° C. [2]. In localities where the aecidia are not common or absent the uredospores are believed to survive the winter on the shaws, to bring about fresh infections in the next season. The dead shaws should, therefore, be collected and burned. Peas are also attacked by another species of Uromyces, *U. pisi*, which is a heteroecious fungus, its aecidial stage being found on the spurge *Euphorbia cyparissias*, but this rust is rare in Britain.

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Chocolate Spot Disease of Broad Bean, *Botrytis cinerea* Fr.; *B. fabae* Sard.

Brown spots on the leaves of the broad bean are of common occurrence and may be caused by various organisms. The 'chocolate spot' disease is attributed to two species of *Botrytis*, *B. cinerea* [10, 11], and *B. fabae* [9]. The latter appears to be the more virulent, but has only recently been reported in Britain [6a], though it is fairly widely distributed elsewhere. The disease appears to have been first discovered in England, in 1849, by Berkeley [1], but he did not associate it with any particular organism. In 1920, 1926, and 1935 widespread epidemics of this disease occurred in Britain, that of 1920 being responsible for losses of from 40 to 50 per cent. of the bean crop in southern England, and losses as high as 75 per cent. were experienced in particular areas [11]. The disease has also been recorded in Australia [4, 5], Spain [9], Portugal [11], Japan [3], Cyprus [6], Argentina [3a], and parts of lower Egypt [2a].

Chocolate spot is far more common on winter-sown than on spring-sown beans, and first signs of the disease may be seen as early as December but usually from March onwards, in the form of small brown spots of variable pattern, on one or both sides of the leaf, but chiefly on the upper side. The spots may either be superficial or may extend through the leaf from one epidermis to the other. Brown lesions occur, too, on the leaf petioles and on the stems, in the form of streaks of variable length having a reddish margin if caused by *B. fabae*, and brown spots on the pods may work their way through the wall of the pod to the testa and seed. The trouble is checked in dry weather, the lesions remaining as mere spots or streaks, but if there is prolonged rainfall during spring the disease takes on a more severe aggressive turn, and spots on the leaves may coalesce so as to present the appearance of a blight which may be followed by partial defoliation, and in severe infection whole plants, blossom included, turn black and die (Fig. 286).

The disease has been variously attributed by different authors to bacteria [7, 8], virus [3, 4], frost injury, aphis infestation [4], nutritional deficiency, and to various fungal organisms [6, 9, 11, 12]. Whilst it is comparatively easy to isolate bacteria from the chocolate-coloured lesions, it has been definitely established that they follow in the wake of the two species of *Botrytis* which cause this disease, and while some authors ascribe the trouble to one or the other species, it is probable that there exist many forms of both species capable of producing chocolate-coloured spots on leaves of the broad bean. Leaf spots induced by aphis punctures are more of a reddish tinge than chocolate colour, and confined usually to the upper side of the leaf, and they are blotchy and more diffuse than the true chocolate spots due to *Botrytis*. Brown spots may also be caused on the leaves of broad bean by the fungi *Cercospora fabae* and *Ascochyta fabae* [12], but these attack the plant in the spring and early summer, and they confine their attacks mostly to the lower leaves near the ground. Spots caused by *Cercospora* are large, dark brown, and zonate, and the conidia which develop on them form a velvety layer, while in the other case of attack by *Ascochyta*, the black pycnidia which develop at the centre of the spots are sufficiently distinctive of that organism.



FIG. 286.—Chocolate spot of broad bean (*Botrytis cinerea*). Showing the 'non-aggressive' and slight, 'aggressive' forms of the disease (after Wilson, *Ann. App. Biol.*)

There are good reasons for accepting *Botrytis cinerea* as the conidial stage of a 'perfect' stage in the genus *Sclerotinia* in the apothecial group, the Discomycetes, and the relations between the two forms, viz. the *Botrytis* stage, and the *Sclerotinia*, apothecial stage, have already been discussed in Chapter I (p. 37, see also Figs. 40, 58, 77 D). The apothecia are, however, rarely formed, but sclerotia are

plentiful and they produce the *Botrytis*-form on germination. The fungus is easily grown in artificial culture, and good growth may be obtained on steamed bean leaves, the fungus producing abundant spores [11]. On malt-agar, the *Botrytis*-' form ' isolated in Cyprus [6], produced a sparse growth of aerial mycelium and conidiophores, but black sclerotia were plentiful ; the oval conidia are described as developing on minute sterigmata at the swollen apices of branched conidiophores, and forming clusters, either in a terminal or intercalary position ; the conidial dimensions of the Cyprus fungus are 13 to 20 by 9 to 18 μ and those of the sclerotia in culture, 0·5 to 3·0 by 0·3 to 2·0 by 0·8 mm. In view of the similarity of the climate it is believed that the ' form ' isolated in Cyprus [6] is very similar or identical with a ' form ' found in Spain [9] named *B. fabae*, the dimensions for the conidia of the Spanish ' form ' being 15·2 to 24·3 by 10·9 to 18·0 μ, and for sclerotia 1·0 to 3·6 by 0·9 to 2·2 by 0·4 to 2·0 mm. *B. cinerea* is probably the most ubiquitous of all fungi, possessing numerous strains ; some virulent, others, more or less weak parasites, and practically all strains are capable of thriving as saprophytes on plant debris such as decayed leaves and rotted fruit. The organism in mycelial form appears to be capable of hibernating within the fallen bean leaflets on the ground, but as the fungus is so common, other sources for the supply of conidia are probably extensive, but it is not known whether all strains of the organism can attack the bean plant. Conidia of *Botrytis* are said to be present on leaves of broad bean at all times, even of healthy plants in weather unsuited for infection, and are evidently capable of existence in the face of adverse conditions over long periods.

In artificial infection, when conidia are deposited in drops of water on a leaf of broad bean, small spots of an iron-grey colour begin to appear in about 48 hours, and in wet weather entire leaflets may be destroyed in 3 or 4 days if the spots are close together. Older or moribund leaves respond to infection quicker than young leaves but, all the same, the strain or strains of *Botrytis* concerned are virulent parasites of the bean plant [11]. A distinction is made between two types of the disease, namely, a ' non-aggressive ' mild form characterised by the death of localised areas of tissues, and an ' aggressive ' form causing blackening and death of a part or the whole of the shoot system, the more severe form following upon heavier infection under conditions favourable to the growth of the fungus (Fig. 286). Early infections of the mild form soon change colour from iron-grey to brown except for a small area at the centre of the spot which remains black. The conidia germinating on the leaf produce appressoria on the germ-tubes before penetration of the cuticle is effected, but unless infection is heavy the invading fungus usually enters no further than the epidermis below the point of penetration. In the severe, aggressive type of infection, however, following upon a heavy deposit of conidia on the leaves, the invading hyphae pass into the intercellular spaces of the mesophyll tissue and may extend from one surface of the leaf to the other, causing the cell walls to swell before a collapse of the cells takes place, and meanwhile the dead tissue has gradually turned brown, and the development of the characteristic chocolate colour in the spots is believed to be due to the conversion by the fungus of the colourless substance tyrosin present in the cells of the leaf into the brown melanin [11].

The incidence of chocolate-spot disease is greatly influenced by temperature, and while the minimum degree for infection lies around 1° C., and the maximum close to 30° C., the optimum is about 20° C. Thus heavy infection may be

obtained in 8 to 12 hours at 20° C., while at 5° C. the same amount of infection is only obtained after 3 or 4 days. As in many other diseases, relative humidity of the atmosphere and temperature are closely interdependent, and while in a dry season the absence of the disease may be attributed to lack of moisture, the amount of disease even in a wet year is comparatively small if temperatures remain high. A spell of dry weather offers a decided check to infection; thus in 1941, in the south of England where extensive damage was again witnessed on field beans, in some areas the crop recovered so well following sunny weather that one-half to two-thirds of the estimated yield was realised [12]. At the start infections with *Botrytis* demand a high degree of humidity in the form of a surface film of water, and for the sustained aggressive type of infection the atmosphere must remain practically at saturation point before the tissues are killed. A heavy water-logged acid soil is also conducive to the disease. In soils deficient in potash, attacks may be controlled by the application of kainit, or muriate of potash, the former at the rate of 6 cwt. per acre, the latter at 1½ cwt. per acre. Spraying with Bordeaux mixture (10 : 10 : 100) gave good results on a small scale. Seed dressing is recommended, and infected debris, an important source of infection, should be burnt. Spring-sown beans are less susceptible than winter-sown beans to infection, but it appears that they are more liable to aphis infestation [11].

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Halo Blight of Dwarf and Runner Beans, *Pseudomonas phaseolicola* (Burkh.) Dowson

Several bacterial blights of dwarf and runner beans (*Phaseolus vulgaris*) have long been known in the United States and attributed by various authors to a number of closely related but distinct organisms [4, 11, 12, 28]. In 1924, however, some crops of bean plants showed, in addition to the usual dwarfing and wilting of a part or the whole of a plant affected with bacterial blight, certain features on the leaves which had hitherto not been detected. These new symptoms of disease consisted of one or more spots of variable size but surrounded by a peculiar light-coloured area forming a characteristic 'halo' [4, 5]. There is abundant evidence that the organism isolated from the halo spots and named *Pseudomonas phaseolicola* is distinct from the other bacteria known to attack beans, and so the new disease became known as 'halo blight' [4, 5, 7, 15, 23].

In 1928 this disease was again very destructive in America, causing great losses in Montana, Wyoming, and Colorado [10]. It is also present in Europe, especially in northern Holland [26] and Germany [2, 3] where it is known in both countries as ' streak ' or ' grease-spot ' of beans [14, 16, 25]; its occurrence is also reported from France, Spain, and Switzerland. In Britain, bacterial disease of dwarf bean was not known before 1930, when an outbreak reported on the varieties Canadian Wonder, Early Prolific, and Masterpiece turned out to be halo blight, and since that date the trouble has been somewhat on the increase on dwarf beans, but not to the same extent on runner beans [17]. In Australia, during the season 1930–31, halo blight caused grave concern to seed growers in Victoria [1], and it has been reported also from other parts of Australia and from New Zealand [20, 21].

The disease is characterised by a wilting of a part or sometimes of the whole plant, the leaves collapse, turn brown, and cling to the plant, or, if the leaflets have fallen, the leaf petioles usually remain erect, and in severe attacks the pods wither and fail to produce seeds [4]. The chief damage done by the disease in England, except in wet seasons, is in checking the plants and reducing the yield [19]. The symptoms are somewhat variable at one season or another, especially in the type of lesions on the leaves. Three kinds of leaf spotting are distinguished: two of these are believed to arise from outside or local, stomatal infections, and the third is due to systemic infection. The halo spot is a local infection showing up as a brownish area of dry texture, with a chlorotic border, the halo around the dead necrotic part; the extent of the halo may vary from a half to one inch in width. The halo, however, is not a constant symptom, although it gives the disease its name, for it appears only during the early, cooler parts of the season; when the temperature rises, as during July and August, the halo is not evident and another type of lesion appears. These later-formed spots, also arising from local stomatal infections, are smaller and more numerous than the halo spots; they are angular in outline, and instead of each spot being furnished with its own halo, the whole lamina presents a more or less pallid appearance. Sometimes spots appear on the leaves which are neither angular nor haloed and may, instead, be of indefinite shape but furnished with a narrow yellowish border. These differences in the nature of the spots are believed to arise from different ways of infection, the yellow-bordered type arising from systemic infection, and the halo and angular types as already stated, from stomatal infection, from without [5]. The spot-form with yellow margin is, no doubt, the



FIG. 287.—Halo blight of French bean (*Pseudomonas phaseolicola*). On the French bean (*Phaseolus vulgaris*). *A*, the compound leaf showing typical haloed spots. *B–D*, leaflets showing clear haloes. (*A*, *B*, photos by Ogilvie, by permission of Long Ashton Res. Station. *C*, *D*, by Foister & Noble)

type described by European writers as suggestive of a mosaic pattern on the leaves [25].

While the more evident symptoms of the disease are those on the leaves (Fig. 287), characteristic lesions may also be seen on the stems, pulvini, leaf petioles, pods, seeds, and on seedlings. On the stem the lesions take the form of elongated streaks furnished with a narrow reddish border [15]. Similar lesions may also appear on the stems of the seedlings, first as water-soaked areas which later turn dark green, and finally, as the spots dry out, developing a reddish margin like the lesions on the mature stem. These lesions on the stems of seedlings are usually of local, external origin (but may also be systemic, as described below) arising from stomatal infections. Since the stems of young seedlings are furnished with stomata, these early infections from without are believed to be brought about from the dissemination of the bacteria in the surface water of irrigation [24]. But when the infection of seedlings is systemic, the bacteria within the tissues of the stem, accompanied by copious slime formation, may break out to form long streaky lesions or deep fissures through which the bacteria exude in white milky masses to collect on the surface of the stem. On the pods the spots are more or less circular, but may often run together to form groups of irregular shape; like those on the leaves they appear water-soaked at first, but turn a reddish brown as they dry out. The bacterial ooze which exudes from the pod lesions often dries into a thin silvery crust in dry weather but is easily washed off, and no doubt, during rainy periods, the bacteria from the pods, or from any other infected parts of the plant, are splashed on to neighbouring plants to set up secondary infections. Pod lesions may also exist as more or less continuous streaks lying within one or both sutures or grooves of the pod. While sutural infections are, in general, of systemic origin, it is conceivable that secondary infection of the sutures may take place when moisture, trickling down the leaf petiole, carries with it bacteria from the lesions on the leaves, and thence along the sutures of the pod where infection may again take place through the stomata present in the pericarp of the young pod. The most important infection is, of course, that of the seed. If the seeds get badly infected they remain small and wrinkled; on white varieties of seed, infection may cause the seeds to turn completely yellow, but slight affections consist merely of maize-yellow to cream-coloured spots on the testa; on coloured varieties of beans, however, it is not easy to detect slight seed infections, and when such seeds, unsuspected of carrying the disease, are collected as healthy, they are the principal means of spreading the disease.

Infection of the seeds may be brought about in two ways. Firstly, from an external source, the bacteria may pass from the lesions already present on the surface of the pod, through the pericarp into the seeds in contact with it; and secondly, as already stated, seed infection may be systemic. By either way of infection the bacteria collect mostly under the seed coat, forming raised blisters, around and sometimes also between the cotyledons, the tissues of which are finally entered by the bacteria. If there is an external lesion in the ventral suture of the pod, the bacteria may find their way into the seed by penetrating the seed stalk, or the organisms may actually pass into the seed through the micropyle. While some authors [14, 28] state that the organisms are not found within the

cotyledons before the seeds start to germinate, others have seen them in the resting seed to a depth of four to five cells in the tissues of the cotyledons [21]. Thus, the germs are carried in the seed, in the tissues of the testa, on and between the cotyledons, and in the tissues of the latter organs as well.

There is no definite evidence that the organism causing halo blight of beans is capable of persisting for any length of time on plant remains or in the soil [5, 18], and it is fairly well established that the disease is carried by the seed [5, 12, 14, 18]. While it is true that infected seeds produce infected plants, the disease may be contracted every year from the debris of a previous crop of diseased beans ploughed into the soil, but only when such material is unusually heavily infected and the ground is planted with beans again almost immediately [1, 26a].

With the planting of germ-laden seeds, systemic infection begins. Only seeds which are slightly infected will germinate, and the first signs of disease are usually manifest when the seedlings are about 9 to 12 inches high [28]. In the field, the first signs of trouble are seen by the presence of bare patches, and in these areas it is found that some seedlings were killed before emergence, and others, soon after putting forth their cotyledons. On the flat surfaces of the latter may frequently be seen lesions identical in shape and position, indicating that the lesions originated from the presence of bacteria between the apposed cotyledons whilst in the seed. When one or both cotyledons are only slightly affected, the bacteria may not travel through the tissues of the cotyledons quickly enough to enter the stem before the cotyledons are ordinarily thrown off by the absciss layer ; in these cases the seedlings have every chance of growing into healthy plants, if they escape secondary infections. But when cotyledonary infection travels as far as the junction of the cotyledons with the hypocotyl, invasion of the stem tissues of the seedling is inevitable [28].

Within the tissues of the cotyledons the organisms multiply enormously during germination, increasing their accommodation within these organs and, later on, in the plant, by dissolving the middle lamellae, so that large pockets of bacteria are produced ; numerous cells of the cotyledons also become filled with bacteria [28]. In the young shoot the bacteria travel for the most part upward, and do not appear to enter any of the tissues of the plant situated below the cotyledonary node. The stem of the young seedling is invaded either by way of the vascular strands or by means of the intercellular spaces between the parenchyma. It is evident that the bacteria are often present to a great extent in the vascular tissues, but it is not clear how the organisms are enabled to enter lignified vessels, or how they migrate from one lignified tract into another. In close vicinity to the vascular bundles, however, the germs are seen to occupy large lysigenous cavities, and the probability is that, at first, they collect in the intercellular spaces of the parenchyma, and that the first elements of the xylem to be occupied are those of the protoxylem close to the germ-filled pockets. This occurs either by a solvent action exercised by the bacteria on the thin walls of the protoxylem cells, or these cells may become occupied by the bacteria when the thin walls become torn, as so often happens, when the protoxylem elements elongate rapidly in the early stages of growth. Once within the stem there is no doubt that the organisms are capable of causing great destruction of various tissues, for large groups of cells may be replaced

by bacterial pockets and slime which, through absorption of water, may bring about the displacement and rupture of the xylem tissues and so allow the bacteria to enter the lignified tracts. The first indication of the arrival of the bacteria in the leaves, from the stem, is revealed by the appearance in the younger leaves of a slight transparency over the smaller veins which later show a reddish discoloration, and microscopic examination of the leaf shows the bacteria to be present both in the lignified tracheids as well as between the cells of the parenchyma adjacent to them [28]. It is possible, therefore, that from the rapid multiplication of, and pressure set up by the bacterial masses within the stem, the organisms, sooner or later, may be extruded through the stomata, or, if infection is very severe, the pressure of the exudate within may be so great as to split the epidermis of the young stem on the surface of which the bacterial exudate collects in the form of milky, mucilaginous streaks. In the young bean seedling, even at an early stage of germination, the bacteria may be found within the xylem tracts and in lysigenous cavities in the neighbourhood of the bundles. Cell destruction and occupation of the water channels may be so extensive during germination that the transpiration stream is seriously interfered with, and the young seedling wilts or dies. Whether the wilting is due to actual plugging of the vascular elements with bacteria [5], or to the infiltration in advance of a toxic substance secreted by them [28], is not known.

In the same way as it has been mentioned above that the bacteria can break out to the surface of leaf or stem by passing through the intercellular spaces or disintegrated tissue, so also may they find exit from the interior of the pod to the surface by way of stomata in the wall of the pod. Here, too, the germs fill the substomatal cavities, occupying the tissues of the pericarp and intercellular spaces, and finally causing a disintegration of the pod tissues. All stages of pod infection may be met with ; even when only a slight amount of infection may be in evidence on the outside the lining of the pod may be covered with masses of bacteria embedded in slime, and surrounding in many cases every individual seed [26]. Seed gaps in the pod indicate early ovular infection and destruction of ovules.

The planting of seed which has become infected by micropylar penetration alone appears to present similar features to the systemic type of infection and is believed to be as widespread as penetration by way of the testa. But micropylar infection initiates a different method of attack from the systemic way and really falls within the category of secondary or external infections. When the micropyle-infected seed begins to grow, it is clear that the young emergent hypocotyl, as it pushes its way out through the diseased region of the seed coat, has every opportunity of picking up bacteria from the micropyle as well as from the germ-laden testa. The tissues of the newly emerging hypocotyl are, however, not easily entered by the bacteria, because the young epidermal cells of this region are close together, and not until this organ has begun to elongate and to develop stomata in its epidermis are the organisms enabled to enter it ; as the stomata open, the adherent bacteria enter the hypocotyl and so initiate infection in this region of the seedling.

Secondary or local infections are usually stomatal, and it does not appear that wounds are necessary prior to an attack. Dew, splashing rain, contact of healthy

with infected individuals, and possibly insect agency, are responsible for the spread of infection from primary lesions in the field. In local infections, when the bacteria collect over the stomata, the organisms, having penetrated into the substomatal cavities and intercellular spaces of the leaf, seek out the small veinlets in various parts of the leaf, and here, too, as in the case of early systemic invasion of the hypocotyl, entry of the bacteria into the vascular system of the leaf is believed to take place first into the thin-walled tracheidal elements of the veinlets and thence into the xylem of the larger veins and midrib, finally travelling down the petiole and pulvinus into the stem of the plant. In secondary infections of the stem, thus begun from the leaf or from direct entrance of the germs from the exterior by way of the cauline stomata (as above described when the hypocotyl gets infected during germination), it appears that invasion of the stem tissues is largely confined to the cortical parenchyma ; only in cases of very severe infection, resulting in much displacement and rupture of tissues, are the bacteria enabled to find their way into lignified vessels of the secondary xylem [28].

The bacterium causing halo blight has been named *Pseudomonas medicaginis* var. *phaseolicola*, but recently the name has been abbreviated to *Ps. phaseolicola* which has the advantage of being shorter and distinctive ; it is a fairly large, rod-shaped organism, occurring singly, in pairs, or in chains ; long filaments are frequently formed ; non-sporing ; motile by a single polar flagellum or 1 to 3 flagella [14] ; dimensions, according to various authors, are 1·35 to 3·6 by 0·9 to 1·2 μ [5], 1·5 to 3·75 by 0·75 to 1·5 μ [6], 1·5 to 3·0 by 0·5 to 1·25 μ [8] ; gram-negative ; not acid-fast ; facultative anaerobic. In culture, on nutrient agar, the slow-growing white to creamy colonies are concentrically ringed, with undulate edges, but the colonies may be rough or smooth and the organism seems to be unstable so far as this character is concerned [15] ; on gelatine media the colonies are slow-growing, circular, raised and wrinkled. It produces an abundant green fluorescent pigment in Uschinsky's solution [19]. The temperature relations for growth are, opt. 25° to 30° C. ; max. 36° to 37° C. ; min. about 0° C. The thermal death point is between 49° and 50° C. *Bacterium puerariae* (Hedges) is the same organism [11].

On plants inoculated at 24° and 28° C. the first symptoms of disease appeared in about 6 to 10 days, but the effect of temperature on the incubation period was, on the whole, very slight. It is interesting to note that the characteristic halo surrounding the infected spots occurred only at the lower temperatures, 12°, 16°, and 20° C., and at these comparatively low temperatures the number of lesions per leaf was small in comparison with the greater number of spots without halo formed at higher temperatures [9]. While it appears that halo blight can occur over a wide range of temperatures, the actual number of lesions per unit area may be far greater at higher temperatures ; from these numerous lesions secondary infections are set up, and when the temperature drops an epidemic of the blight may follow. High humidities are stated by some to be a necessary preliminary to an outbreak of halo blight [1, 12], but others observed that low humidities had no apparent effect on the development of the characteristic halo symptom, and even at high temperatures, when the foliage wilted during periods of low humidities, the severity of infection was as great as with high humidities [9]. The general view would appear to be, however, that provided there is a 24-hour

period of relatively high humidity at the time of infection, it is immaterial what the amount of moisture may be in later periods for the progress of the disease.

In Britain [17, 18, 19] the most popular varieties of dwarf bean are more or less susceptible to halo blight, and the trouble is fairly common on Canadian Wonder, Masterpiece, and Early Prolific; Abundant, Unrivalled, Incomparable, Magpie, Superlative, Ne Plus Ultra, and Black Wonder are somewhat resistant. In later trials at Long Ashton, Peerless also proved to be resistant, and The Prince, a valuable variety on account of its earliness, is also resistant [19]. Since the bacteria are in intimate contact with the seed, it is pointed out that there is a very small margin of safety for the control of the disease by seed treatment with chemicals, and in Australia such methods have failed [21]. A selection of the susceptible Canadian Wonder, named Burnley Selection, and a variety called Pale Dun were very resistant in Victoria, and Feltham's Prolific also maintained its resistance except under greenhouse inoculations. Several investigators advise the practice of roguing, if carried out soon after the appearance of the halo stage [1, 18, 22]. In Australia and New Zealand it seems that seed treatment with fungicides has not proved a success [1]. Other investigators in Europe, however, recommend steeping the seed for 15 to 30 minutes in water heated to 52° or 55° C. following a preliminary soaking for 12 hours in tap water [2, 13, 14]. Immersion of the seed in a 0·25 solution of uspulun, or in 0·05 to 0·1 per cent. germisan, for fairly long periods of 12 to 16 hours, has also given good results [1, 13].

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Anthracnose of French Bean, *Colletotrichum lindemuthianum* (Sacc. & Magn.) Bri. & Cav.

This disease is known wherever French beans are cultivated. It was first discovered in Germany, in 1875, and appeared in England five years later [4]. It is not very common in Britain [21, 22] but may be severe in cool, wet summers, [15a] and is fairly widespread throughout Europe, America, Australia, New Zealand, Africa, and India [1, 2, 3, 5, 6, 9, 10, 11, 12, 19, 20, 30]. Anthracnose occurs most commonly on the French bean (*Phaseolus vulgaris*) but has also been found on the scarlet runner (*P. multiflorus*), and to a lesser extent on other pulse crops such as Rangoon bean (*P. lunatus*), cowpea, val (*Dolichos*), etc., in India [9], and on the broad bean (*Vicia faba*) in China [31]. But whether the disease on these hosts (and on a wide range of exotic plants on which symptoms have been described suggesting close affinity with anthracnose of the French bean) is due to the same fungus or to different races of it, or to distinct but related species, is not yet clearly established [28].

The term ' anthracnose ' is descriptive of the appearance of the symptoms in the form of ' burnt black ' lesions which may appear on all parts of the plant except the roots. This disease may attack the cotyledons and leaves of seedlings, the stem, the bracts of the inflorescence, stalks and sepals of the flowers, but produces its most striking effects on the pods (Fig. 288); the foliage leaves are not usually badly affected. The characteristic symptoms consist of black, rather sunken spots, surrounded by reddish or yellow, slightly raised margins. The spots begin as small, round, brownish or purplish specks which become darker in colour as they enlarge to about a $\frac{1}{4}$ inch in diameter. As the infected tissues become dry and collapse, a depression develops at the centre of the lesion which under moist conditions becomes pink and oily in appearance due to the liberation of a great quantity of spores. Spots may unite together, especially on the stem where they may form lesions several inches long, and owing to cracking and rotting of the tissues young stems may collapse to the ground. The spots are very conspicuous on the pods and are of somewhat similar appearance to those on the stems. When the pods are opened the lesions on them are often found to have penetrated right through to the seed coat into the seed. On the foliage leaves the lesions are mostly on the veins and on the lower side of the leaf stalk on which they are similar to those on the stem; sometimes the petiole may be so badly affected that it



FIG. 288.—Anthracnose of French bean (*Colletotrichum lindemuthianum*). The lesions on the pods. Note the minute black spots, the acervuli, within the lesions (photo by Foister & Noble)

fails to support the leaf, and at times the lamina may show on the upper side a number of angular, elongated areas which may sometimes become torn, but a whole lamina is seldom killed [3].

Anthracnose of dwarf bean is caused by the fungus *Colletotrichum lindemuthianum*, first described in 1878 under the name *Gloeosporium lindemuthianum*, but because of the presence of bristles or setae in the fructifications, was transferred to the former genus in which it still remains, as a member of the Fungi Imperfecti. But the setae, never numerous, are often entirely absent, and the organism has been variously referred to the genus *Colletotrichum* or *Gloeosporium*, according to the presence or absence of the bristles, respectively. The evidence with regard to the existence of a perfect ascigerous stage for this fungus in the genus *Glomerella* is incomplete and, so far, this species causing anthracnose of bean has not produced a perfect stage. Numerous physiologic races of the organism exist [5, 6, 15, 23, 26, 27, 29] and certain strains have produced sclerotia in culture [6]. The conidial fructifications or acervuli are developed on stromatic layers between cuticle and epidermis (Fig. 289). From the surface cells of a stroma, short, densely crowded conidiophores arise which abstrict the conidia singly, and the surface of the spot where the cuticle has been broken through soon becomes covered with pink masses of spores held together by mucilage. The conidiophores are cylindrical, hyaline, unbranched, non-septate, and measure from 45 to 55 μ in length. The conidia are hyaline singly, but pale pink in the mass, unicellular, and vary in shape from cylindrical to elongate-oval, sometimes slightly curved, with rounded ends; they measure from 15 to 19 by 3·5 to 5·5 μ. Most commonly, as above stated, the circumference of the stroma may be provided with stiff bristles or setae, dark brown in colour, unbranched and septate, arising singly from the marginal cells of the stroma; they may also be found here and there among the conidiophores; the setae range from 30 to 90 μ in length, and may vary from a few to as many as 20 within an acervulus. The acervuli are formed first near the centre of a lesion, and may be followed by more as the spot becomes extended, so that in a single lesion as many as fifty or more acervuli may be present.



FIG. 289.—*Colletotrichum lindemuthianum*. *A*, *B*, sections of acervuli showing setae and spores (×245). *C*, conidia (×480). *D*, germination of conidia; one has formed a septum, two others have formed appressoria (after Edgerton)

The fungus may be cultivated on bean agar, or on sterilised bean pods, at temperatures from 20° to 25° C., but on other media it appears to lose its powers of sporing unless a very small fragment of fresh bean leaf or pod is added, after which growth or the germination of the spores is stimulated to a remarkable degree, though loss of virulence and pathogenicity appear to vary considerably with different strains of the fungus [8]. In culture, the mycelium is white at first but darkens in a few days; sporulation generally is very sparse on agar media without the addition, as above stated, of a suitable growth substance. The fungus is capable of tolerating as low temperatures as −15° to −20° C. for several days [18], but is sensitive to high temperatures; the optimum and maximum for growth lie between 22° and 23° C. and 30° and 31° C. respectively, though values of 18° to 22° C. and 33° to 35° C. for these points respectively, and as high a temperature as 50° C. for the survival of the mycelium, are also recorded [11, 26]. The

optimum temperature for sporulation is near 15° C. with the minimum and maximum at 4° and 38° C. respectively [26]. Germination of the spores takes place more rapidly at temperatures higher than the optimum for growth, but not normally above 27·5° C., the critical temperature occurring between 32° and 35° C. [15].

Cool moist weather during the summer affords the best conditions for the prevalence of anthracnose [21], and high atmospheric humidity is essential, not only for the success of initial infections but also for the dissemination of the spores in the field [14, 26]. In dry seasons the seed harvest is relatively free from disease. The fungus is capable of wintering on bean shaws, and of fructifying on them in the spring, and survives in the seed in the form of a dormant mycelium, either in the testa or within the cells of the cotyledons [3]. Sporing acervuli may sometimes be found on the surface of lesions on the seed, and even between the apposed surfaces of the cotyledons [26]. Few spores are apparently able to survive the winter in the soil, and the evidence, as a whole, indicates that the parasite is usually carried over from one season to the next in a mycelial condition in the seed.

Carefully conducted experiments have proved beyond all question that seeds taken from diseased pods into which the fungus has penetrated so as to infect the seeds, give rise to diseased seedlings. The fungus may be found in the seed coat, in the fleshy cotyledons, and even in the plumule. If infected seeds are kept dry the fungus makes no growth, but if the correct conditions are obtained at planting, the mycelium is stimulated to growth. There is, however, little invasion of the tissues of the growing shoot, and the fungus, confined largely to the surface tissues, soon breaks out and actually sporulates on the young stem and the cotyledons. Some of these early-formed spores may be washed into the soil only to be splashed up again in raindrops, and this appears to be the chief method of carrying infection to neighbouring plants, wind dispersal being of little account. Other spores may be picked up by any of the older leaves that may happen to touch them. Spread from field to field is slight and is usually due to carelessness of workers, and experience has taught the danger of working in the crop on wet days since the spores can be carried about on clothes and implements. All later-developed parts of the plant may be infected in turn from the primary seedling infections.

The spores become attached to the cuticle by virtue of the mucilaginous coat, and penetration following the formation of an appressorium is direct through the cuticle. A finely branched mycelium eventually becomes established in the superficial cells of the organ affected (such as the outer cortex of the stem), the cells becoming occupied by the invading mycelium and suffering little harm, and with hardly any discoloration of the tissues evident from the outside. About 4 or 5 days after infection, however, the invaded tissues collapse and die, and discoloration is soon apparent in the injured area. In the latter the fungus proceeds to aggregate towards the surface, concentrating chiefly at one or more places under the epidermis where the stromata finally become established to develop the acervular fructifications.

Control over anthracnose of the bean is mainly directed towards the elimination of diseased seed. Attempts to do this by selecting seeds which show no sign of spotting has not been very successful, for it has been found that many seeds may

be infected without any external markings sufficient to enable them to be readily detected. On the other hand, diseased pods can always be easily recognised by the spots on the pericarp, and if all spotted pods are discarded and seed taken only from the clean, the resulting crop should be free from primary infections. Seed treatment with fungicides is not advocated, since the parasite is usually too deep-seated, and the good effects reported in some cases following seed disinfection seem to be nullified by the reduced germinability of the treated seed. Treatment may be given with 0·25 or 0·5 per cent. solutions, for 15 minutes, of either 'uspulun', 'ceresan', or 'fusariol', or the dry applications may be made at 2 per cent. for uspulun or ceresan and 4 per cent. strength for fusariol [25]. Bordeaux spraying has proved effective on a small scale, but when applied in the field it has been found difficult to cover all parts of the plant, and the general effects were not sufficient to be profitable. Burning all infected plants or ploughing them deeply into the soil will help in preventing the fungus being carried to the next crop.

While all grades of resistance to this disease have been found among different varieties of French beans, no variety has yet been produced which has proved to be immune from it. Breeding experiments conducted chiefly in Germany [6a, 23, 26, 27, 29] and America [7, 16, 17], in the latter, by crossing the susceptible White Marrow bean with the resistant Well's Kidney Red, etc., have yielded promising results, and further trials on the production of a white seed variety resulted in the choice of White Imperial on the score of its high resistance to anthracnose; in addition to its vigorous growth, this variety, when crossed with Robust, a high yielder, gave even better quality seed than White Imperial, and proved highly resistant to certain strains of the parasite [24]. In New South Wales, a selection from a crop of Canadian Wonder, called Tweed Wonder, proved to be resistant to all strains of the fungus in that locality [1]. In Britain [21, 22], the varieties Princess, Hundred for One, Selected Canadian Wonder, Foremost, Magpie, Leicester Wonder, Abundance, Emperor William have been observed to be practically unaffected, while Lightning, Unrivalled, and Best of All were seen to be only slightly susceptible. In a given locality, a variety may preserve its resistance for some time if the local strains of the parasite are not able to attack it; but other strains may be introduced, through seedsmen for instance, and the variety fails. On the whole, therefore, no method gives such good results as the use of clean seed [9].

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Foot Rot of French Bean, *Fusarium solani* (Mart.) Sacc. var. *martii* (Appel & Wr.) Wr. *f. 3* Snyder

'Foot rot' or 'dry root rot' affects all commercial varieties of the French bean. The disease was first reported in England in 1929 [4], but was known in America, where it causes more serious losses, long before that date [1].

There is little evidence of the trouble in the field before the plants are about 5 to 6 weeks old above the ground, and there is not much difference between affected and healthy individuals, except for a slight check to growth. The rot, starting at the tips of the main and lower lateral roots, causes a gradual drying of the tissues and killing them as the parts near the surface of the soil are reached. There is, however, little if any invasion of the stem, and by development of surface, adventitious roots, diseased plants often make good recovery. Frequently the symptoms may not be seen until the first pods have made their appearance, when the lowermost leaves of affected plants begin to turn yellow and wither at the edges, and the later-formed pods may become shrivelled before they fill out [4]. But a yellowing and dropping of the leaves is not a constant feature, and diseased plants usually come to maturity earlier than the healthy because of the drying effect the disease has on the tissues, due to a reduced rooting system. The worst effects are seen on the roots. The primary roots may be covered completely with a reddish discoloration, or this may take the form of streaks which may extend even above soil-level. Later, the discoloured roots turn brown and develop fissures, sometimes extending as deep as the cortex. There is, however, considerable replacement of injured roots, and above the latter new roots develop, but these, as they pass out through the cortex, also become infected, and other roots may then develop, still higher up, near the surface. The surface roots frequently form a dense mass of fibrous rootlets and may persist with varying degrees of success, but in bad attacks when infection extends to the 'foot', or base of the stem, even these roots may be destroyed [1, 8].

Foot rot is caused by a form of *Fusarium solani*, as above indicated (see p. 621). The fungus produces three types of spores, namely macroconidia, microconidia (rare), and chlamydospores, mostly in pseudopionnotes of a yellow colour like buttery layers. In dry weather the conidia are rarely produced, even on the roots, but develop in profusion during wet periods. The macroconidia are curved, of uniform width, and may be from 3- to 5-septate, mostly 3-septate, the latter measuring 44·5 by 5·1 μ (mean), and all are usually pedicillate. The chlamydospores are terminal or intercalary, arising singly or in short chains, and measure on an average 11·6 μ in diameter ; they are formed in the host tissues, mostly in the cortex of the finer rootlets, fewer in the larger laterals and tap root, but they have not been observed to germinate. In culture the aerial mycelium is scanty and white, but varies considerably in depth of colour in the culture medium, being sometimes green, or pale olive-buff, or a cinnamon green, according to the type of medium and especially the degree of aeration [1].

The organism probably survives in the soil in the form of spores or mycelium, and as it has been known to exist for ten years in the soil, even remaining active for many years in the absence of dwarf beans [6], there is little doubt that it is capable of thriving under saprophytic conditions. It appears to be unaffected by the hydrogen-ion concentration of the soil [2]. The spores are produced readily on old (but not on growing) bean roots and shaws and may thus be returned to the soil if host debris is thrown to the manure heap. There is no evidence that the disease is carried by the seed, but infection takes place readily when seed is planted in contaminated soil [1, 4, 8].

The exact manner of root infection is not known, but apparently may either be direct or through wounds [3]. The fungus travels almost exclusively in the intercellular spaces of the cortex in which, after disintegration of the tissues, strands of mycelium resembling thin rhizomorphs may be seen, and these strands may also be seen on the exterior of the roots. The fungus rarely extends for any distance above soil-level, though at times may reach the lower parts of the stem. In the latter, the mycelium is often found inside the cells, such parts being stained distinctly red, and while the fungus is concentrated largely in the cortex, in the topmost roots hyphae may also occasionally penetrate into the vascular system in which case there is usually much deposit of gummy substance in the tissue [4].

Practically all varieties of French beans and scarlet runner beans are susceptible to foot rot, though variable degrees of resistance are shown by Flageolet Victoria (Magnum Bonum), Saxony, Incomparable, Flageolet St. Andrew, and Dwarf Sharpe's Goliath [4, 5]. Even among seedlings of the same variety, however, marked differences in susceptibility have been observed. Other species or strains of *Fusarium* may also attack French beans but the pathogen here described has proved to be the most virulent [4, 7].

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Root Rot of Pea, *Aphanomyces euteiches* Drechsler

This type of root rot is undoubtedly the most serious of all diseases that affect the pea plant. It is not so common in Britain as in the United States where it is prevalent in every pea-growing district. It attacks the roots and the base of the stem at all stages of growth. Other hosts include the narrow-leaved vetch, alfalfa, sweet clover, sweet pea, broad bean, and other leguminous plants [17, 24, 28, 42].

This disease occurs with great regularity on land where peas have been grown in repeated succession. In early stages of attack it is impossible to detect the presence of this root rot in the field, for, apart from a slight check to the growth of the young plants, there is no other external symptom, such as spots on the foliage, and it is necessary to pull up trial plants to determine the presence of the trouble. An affected specimen shows a decay of some of the roots and a blackening of a part of the stem just above the roots ; but these features are not distinctive enough to separate this disease from one caused by a species of *Fusarium* (see p. 620) and actually one cannot decide with certainty until the diseased tissues are examined microscopically for evidence of the sexual organs and oospores characteristic of the fungus causing this disease [17].

In this type of root rot the cortical tissues covering the roots become softened and the disease extends from the roots into the stem above ground, though only for a short distance, but by the time this part of the stem has become infected the leaves have turned a pale-yellow colour all over. Invasion of the basal part of the stem is usually followed by a sudden wilting, and with the death of the cortical tissues covering the roots and basal part of the stem, these affected regions become thoroughly blackened [17]. As the disease makes slow progress from root to stem, there is always a decided check to the growth of the shoot, and the foliage dies from below, upwards ; but as a general rule the majority of affected plants are enabled to carry on their growth, though much weakened, until they produce pods and seeds most of which, however, are small and of inferior quality.

Pea plants affected by root rot, even in advanced disease, have a remarkable power of recovery. This often happens when environmental conditions change in favour of the host, as would occur, for instance, when wet situations are improved by adequate drainage, and despite the loss of a considerable amount of rooting system through disease, drier conditions allow the plants to develop new roots from the base of the stem.

Aphanomyces euteiches, the organism causing this root rot, belongs to an interesting group of fungi, the Saprolegniales, few members of which are known to cause plant disease. The group consists largely of aquatic fungi some of which are parasitic on fishes, and others infest low algal forms, such as Spirogyra, Zygnema, desmids and diatoms. The fungus can be grown on various media, e.g. cornmeal or prune agar [16]. Mycelial growth is very slow and sparse ; it is feeble at low temperatures of 8° to 10° C. ; the optimum is about 25° C., the maximum about 34° C. The mycelium is hyaline and non-septate, about 4 to 10 μ in thickness ; it branches moderately, in characteristic fashion, by producing at right angles to the main hyphae short stubby branches which make but little

growth themselves. It is a feature of the fungus to pass through its vegetative phase very quickly before entering upon the reproductive stage, and the same rapidity of development also takes place within the host, for only a few days of incubation at ordinary temperatures are needed before resting oospores are produced. A different strain of this parasite isolated from diseased peas in Tasmania produced coils of hyphae, " like the hair-spring of a watch ", and on agar media containing peptone the strain behaved peculiarly, for only after the omission of this substance from the culture were the sexual organs of the organism developed [9]. Oogonia and antheridia are developed in abundance in the tissues of diseased plants. They are formed, with the ultimate resting spores, the oospores, within the tissues of the decayed cortex of all underground parts of the plant, stem and roots. In culture, oogonia and antheridia develop from distinctive hyphae ; the oogonia are formed in great number, each on a short stalk cell usually at the end of one of the stubby branches of the wider mycelium ; in the Tasmanian strain the oogonia were found enmeshed in the hair-spring mycelial coils. The oogonia are thin-walled, sub-globose, densely granular with vacuolated contents ; after fertilisation the oogonial wall gets thicker ; there is only one oospore developed within an oogonium. The antheridia arise from a less stout hypha as a number of short, curving branches ; one to five antheridial branches may invest an oogonium, and all may put forth fertilising tubes, but details of fertilisation are not known (Fig. 290). The oospores are sub-spherical, 18 to 25 μ wide, thick-walled, and capable of germinating in a direct manner by producing one or more germ-tubes, or indirectly by producing a long, tapering, slender hypha which develops into a sporangium. Within the latter the cytoplasm which is passed into it from the oospore breaks up into several portions, which are released through a small orifice formed at the tip of the sporangium, each portion rounding itself off and becoming encysted to form a spore, but all the spores remain more or less loosely attached to the sporangial tip for some time. When they separate (about 15 are formed in a sporangium), each spore releases through a minute opening in its wall, a motile zoospore. But the development of zoospores, a process which is so characteristic of other truly aquatic members of the Saprolegniales, does not take place in this particular species under natural conditions and their formation has only been observed in culture. It is rather remarkable that zoospores, abundantly produced in artificial culture (such as pea decoction) are not formed when

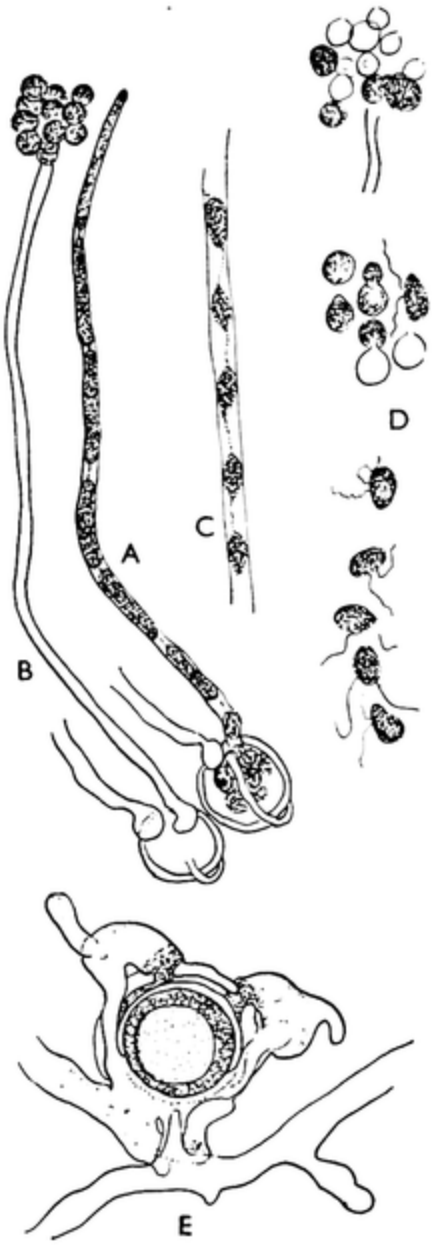


FIG. 290.—*Aphanomyces euteiches*, causing root rot of peas. *A*, *B*, the filamentous sporangia produced from germinating oospores. *A*, 30 sec. before discharge. *B*, the zoospores collecting at the top of the sporangium. *C*, hyphal contents segmenting to form zoospores. *D*, stages in the encystment of zoospores, and their liberation. *E*, an oogonium with a branched antheridium ; all ×310 (after Drechsler, *J. Agric. Res.*)

infected host tissue, well supplied with mycelium is placed in water ; on the contrary, a renewed development of oospores takes place within the rotted tissue.

The great ease with which oospores are produced under natural conditions leaves little doubt that they are the chief means of survival of the organism in the soil. There are records of the persistence of the disease in the soil over intervals of from two to ten years [9, 17, 25]. It is not known, however, in what form the fungus attacks the host, whether from mycelium in the soil or from oospores. But greenhouse inoculations can be performed with success by introducing a culture of oospores into the soil, and though such an experiment may fail at first, a second planting of peas usually becomes well infected. There is no clear evidence that watering the soil with a suspension of zoospores results in infection, though in sand cultures this method has been reported to give infection [17].

From a study of seedling infection, the parasite, applied in the form of mycelium to the young roots, penetrates the hypocotyl by putting forth one or more hyphae which bore through the epidermis at regions between the epidermal cells, and though the basal part of the young stem at a point higher up than the hypocotyl may sometimes be penetrated, it is not usual for the fungus to invade the stem except by travelling up from within the roots. The non-septate mycelium develops almost exclusively in the cortex of the root and basal part of the stem, causing, as above stated, a soft decay of this tissue, resulting in shrinkage and blackening of the root and the part of the stem below ground. A diseased seedling pulled up at this stage of infection usually breaks off near soil-level, carrying with it much of the stringy core of the stele of both tap and lateral roots [17]. The vegetative phase of the fungus within the host, as in artificial culture, is very brief, and the reproductive organs, followed by the oospores, make their appearance as soon as the cortical tissues begin to show signs of collapse.

Root rot of peas is greatly encouraged by a high water content of the soil. It is most prevalent at points near saturation, and is absent or greatly retarded within a range of 40 to 20 per cent. soil moisture. The greatest losses occur when the soil is warm and wet during infection, when the soil temperature is 14° C. or above, for several days, and if there is a spell of drought before the plants reach maturity, large areas of the crop may be completely ruined [12, 13, 17, 22, 41].

The fertility of soil given over to constant pea cultivation tends to become more and more exhausted every year. The main deficiency is attributed to lack of humus and calcium [9]. As a plant of somewhat rapid growth, the pea demands from the soil nutrients in a readily available form, and numerous authors give good reports of the application of nitrate of soda, ammonium sulphate, and muriate of potash in retarding the effects of root rot [14, 40, 41].

As root rot of peas is carried in the soil, not by seed, the disease is controlled mainly by crop rotation [17, 42], and as the fungus is reported to be of long duration in the soil, from two to ten years, intervals between pea crops must be judged according to severity of attack. In an average case of infection a 3-year rotation was found to have delayed but not entirely prevented a recurrence of the disease [8, 25]. Good drainage and good cultivation, to keep the soil in a fairly dry condition, are essential. Since peas grow well at comparatively low temperatures, early planting is recommended so that the plants can be well forward before the soil

gets warm enough to encourage the parasite to action. Some state, however, that early peas, in general, are more susceptible to root rot than late-maturing varieties[37], but no variety is claimed to be truly resistant to this disease [7], even the roots of resistant varieties being affected in the cortex though not in the epicotyl and vascular cylinder [18]. The more susceptible varieties are Alaska, Winner, Green Admiral, Perfection, Horsford, and Advancer, while Horal and Rice's No. 330 are resistant.

Leaf, Stem, and Pod Spot of the Pea, *Ascochyta pisi* Libert

This disease is carried over from year to year by the sowing of infected seeds; it does not usually affect the rooting system nor the part of the stem below ground but has been known to do so in some areas [11a]. It is essentially a leaf, stem, and pod spotting disease, and as the seeds can become infected through the wall of the pod, it is often accountable for heavy reduction in yield.

Leaf and pod spot is common in all pea-growing areas but is not very prevalent in Britain, although at times, capable of causing considerable losses [30]. It has also been recorded from the United States, Ireland, eastern Canada, Denmark, Germany, Holland, India, Australia, and the southern Ukraine.

On the average, healthy plants are about three inches taller than diseased



FIG. 291.—Leaf, stem, and pod spot of pea (*Ascochyta pisi*). Pods showing the minute black pycnidia at the centre of the lesions (photo by Ogilvie, by permission of Long Ashton Res. Station)

specimens, and the leaves, stem, stipules, pods, and seeds may be affected with this disease. On leaves and pods diseased areas are round, up to 9 mm. in diameter, tan to brown, and definitely sunken into the leaf or pod (Fig. 291); each spot is furnished with a black and thickened edge surrounding a paler, tan centre [29]; on stems and leaf petioles the spots are more elongated and around the tan-coloured spots there is a dark brown margin, but lesions on the stems are not usually abundant [20]; if present, they penetrate deeply and in bad cases may girdle the stem, causing the shoot above the lesion to wither (Fig. 292). As the attack on the stem, when it occurs, begins near the ground, this condition may result in complete loss of the plant. When young seedlings about to emerge from the ground are attacked, the tips of the shoots often suffer severely and many young plants may thus be destroyed [30]. Symptoms of disease on the seeds are not much different from those caused by the other seed-affecting parasites described below. Affected seeds are not easily recognised in a dry condition, but when moistened they generally show one, or a few ill-defined dirty-yellow to greyish-brown, or black, wrinkled spots on that side which had been in contact with the lesion on the pod; and sometimes the discoloration penetrates through the testa to the cotyledons. The embryo may also be involved and germination greatly reduced or even prevented [33]. While spots on the seeds are somewhat less conspicuous in this disease than in the others described below it is impossible to associate any type of seed spot with any particular organism from examination of the seed lesion alone.



FIG. 292.—Severe lesions on the stem of the pea, caused by *Ascochyta pisi* (photo by Foister & Noble)

This disease is caused by *Ascochyta pisi*, a member of the Fungi Imperfecti. The two diseases described next are caused by two fungi very closely related to this one, namely *Mycosphaerella pinodes* (a perfect form having an *Ascochyta* pycnidial stage) and *Ascochyta pinodella*, and while all these three fungi have many features in common, the diseases caused by them are distinctive. Thus, as indicated, *A. pisi* does not usually attack the underground parts of the plant, and is a leaf, stem, and pod spotting fungus, whereas the other two cause a severe foot rot as well. Still another foot rot of peas, included in this series, is caused by a variant form of *Fusarium solani*, which like *Aphanomyces euteiches* above described, causes no 'spotting' of the above-ground parts of the host at all. From a consideration of these five fungi and the diseases caused by them, it is clear that we are dealing with a problem which is really a 'symptom complex', presenting great difficulties in allocating to each organism its characteristic features of disease [30]. *A. pisi* has also been shown to be pathogenic to apples and tomato fruits in Germany [45].

Ascochyta pisi has only one kind of fructification, the pycnidium, and is classified in the *Hyalodidymae* of the Fungi Imperfecti. Pycnidia may be present on all lesions wherever they occur. They are tan brown, round and flattened, immersed in the necrotic tissues ; they are fairly numerous and arranged in a group at the centre of the spot, or concentrically in circular zones especially on the mature pods. Pycnidia range from 75 to 225 by 75 to 205 μ (average, 141 by 124 μ) [34] ; the hyaline pycnospores, pink or carrot-red in the mass, are exuded at the ostiole in a mucilaginous matrix which assists in their expulsion by swelling when wetted, and fixes them in a crust around the ostiole when the pycnidium is dry. Collected from moist pycnidia, the spores measure from 10·0 to 18·0 by 2·5 to 5·5 μ (average, 12·9 by 3·3 μ) [34], or 14·1 by 4·1 μ [20], according to different authors. The pycnospores are oblong, obtuse at the ends, 1-septate (rarely 2 or 3 septa) ; each cell has a conspicuous oil globule [20].

A. pisi, as well as the related species mentioned above, are easily cultured on oat, pea, yeast, or potato agar ; the morphological differences between them are seen to better advantage on oat agar, and least on potato agar [20]. *A. pisi* has an optimum temperature for growth between 15° and 23° C., but the amount of mycelium formed is very scanty ; the most suitable reaction is about the neutral point, 6·8 to 7·0 *p*H [34]. Sporulation occurs over a range of 18° to 23° C., and pycnidia are formed in great number, exuding red spore masses over the medium [4, 20]. The fungus is able to utilise soluble starch, glucose, saccharose, but not cellulose ; ammonium salts are superior to nitrates as a source of nitrogen ; lack of magnesium is said to check growth almost completely, and development is poor without phosphorus and sulphur ; in the total absence of all forms of nitrogen pycnidial formation is inhibited [43].

A. pisi is recorded to maintain vitality in culture for 4 years [27], and on pea seeds for at least 6 years [3]. The pycnospores embedded in mucilage are not adapted for wide dispersal, and they appear to be largely disseminated from lesions on the host by splashing rain, or perhaps further afield by insects, but outside the plants in close proximity there is usually no great extension of the disease to distant plots, which probably accounts for the common occurrence of the disease in patches of the crop.

The percentage of infection from planting of diseased seeds, under conditions favourable to the fungus, may range from 50 to 62 per cent. of plants produced [30]. Recent observations in the Bristol area showed that if the seeds do not carry more than 20 per cent. infection there is little loss in the stand [14a]. Although this disease is not carried in the soil, successful infections may be obtained if old pods bearing lesions are scattered under healthy seeds ; 2 to 50 per cent. of pods harvested from particular plants thus infected were found to harbour infected seeds.

If the plants survive to produce pods, the spots on the pods develop with great vigour in wet weather, and the fungus may often be found to have penetrated into the cavity of the pod to attack the testa, and in severe infection the cotyledons as well [6]. Every stage of seed infection may be found, from tiny aborted seeds which infection had probably prevented from developing, to others which matured successfully and bearing mere brown blemishes on the surfaces of the testa. Seeds, apparently clean when the pods are freshly opened, may yet be infected, and the brown discoloration may develop if the seeds are exposed to damp air for some days ; in 4 or 5 days a white mycelial growth

develops on the brown spots, followed some 10 days later by the characteristic pycnidia [5].

Prior to germination of the seed, the fungus is confined for the most part to the seed coat, though in severe infections, as we have seen, it may also be found in the cotyledons. Plants growing from seed infected with this organism are rarely prevented from emerging from the ground, and the primary lesions occur usually on the leaves and stem at the base of the plant, and may sometimes be seen on the first leaves of the emergent plumule. Secondary infections take place by the splashing of spores in raindrops from these early lesions, and the greatest infection of leaves and pods occurs during wet weather.

The spores, germinating on moist leaf or pod, put forth one or more germ-tubes each, and the young hyphae are frequently seen to anastomose before the cuticle is penetrated ; they do not appear to enter at the stomata. The mycelium travels below the cuticle for a while before it wedges apart the epidermal cells by dissolving the middle lamella [27]. The hyaline mycelium is found in and between the cells of the invaded tissue, and the period of incubation before the appearance of spots on the leaves is usually about six to eight days. At this early stage, lesions are light green in colour and somewhat sunken into the epidermis ; two or three days later the spots change into a tan colour, and have a definite brown margin [20]. Artificial infections may also be successfully performed by smearing healthy seeds with a spore culture. Under natural conditions the disease appears to be carried over almost exclusively by infected seed, and the growing of healthy seed in soil contaminated by the organism produces no appreciable amount of infection except in cases of deliberate heavy infestation as pointed out above when heavily infected pods were placed in contact with the seed.

Since this disease is seed borne, attempts have been made to control it by disinfecting the seed. Such a treatment, however, cannot be successful if infection has reached the cotyledons, nor can it be adopted by growers who make a practice of bacterial inoculation with nitrogen-fixing organisms for crop improvement. If seed disinfection is decided upon, it is recorded that applications of organic mercury dust are superior to any wet treatment since the latter has been found to reduce germination and stand [21]. Good results are reported by applying the dust at the rate of 2 oz. per bushel of seed, rotated in a drum for at least 5 hours, but such treatment of diseased seed does not give as high a percentage of stand as untreated healthy seed. Additional benefit is said to accrue from mercury seed treatment of varieties of peas which are wrinkled, over those which have a smooth testa ; what significance can be attached to this observation is not clear unless the rough surface retains more of the fungicide, but the probability is that wrinkled varieties have an inherent resistant property not possessed by smooth-skinned peas, to this disease.

Early varieties of peas are reported to show a lower percentage of infection than late varieties, due probably to earlier harvesting, before conditions of higher temperatures favourable to the fungus set in. There is no claim to complete resistance to this disease for any variety of pea. In the United States the following are slightly susceptible to *A. pisi* and *M. pinodes* (below) : Admiral — ' 17·78 ', Advancer, Badger, Badger —' 20·140 ', Special, Champion of England, Horsford,

and Perfection. But a marked difference in susceptibility of certain varieties is reported for different isolates of *A. pisi*, and strains of the organism from England and Canada were less injurious than other strains obtained from seed harvested in New York and Wisconsin [20]. In the southern Ukraine five strains of *A. pisi* reacted differently in their pathogenicity not only to the pea but to other members of the *Leguminosae* [3].

Leaf, Stem, and Pod Spot with Foot Rot of the Pea, *Mycosphaerella pinodes* (Berk. & Blox.) Vestergr., & *Ascochyta pinodella* L. K. Jones

As an organism causing lesions on the leaves and pods of the pea, *Mycosphaerella pinodes* bears considerable similarity to *A. pisi*, and indeed was, for a long time, thought to be its perfect stage [39a], but is now accepted as a distinctive species [26]. Unlike *A. pisi*, this fungus is also capable of attacking the basal part of the stem underground severely, so as to cause a foot rot and is therefore more destructive than *A. pisi* (Fig. 293 A).

A. pisi and *M. pinodes* commonly occur together on samples of seed in most pea-growing areas. The two organisms are reported to be common on peas in England [30]; but latterly only *A. pisi* has been found on commercial samples of seed used in Britain [14a].

Spots on the foliage leaves (Fig. 293 B), stems, and pods (Fig. 293 C) caused by *M. pinodes* are brown to purplish; they are not so definitely delimited from the green of the leaf as those caused by *A. pisi*, but like them, as they spread with increased wetness of the leaf, tend to become circular in shape. On the stems, early lesions are black to purple, and take the form of streaks which are more evident at the nodal regions than elsewhere, though they may later enlarge into brown to purplish irregular areas over the entire stem for a length of some 6 to 10 inches above the highest insertion of the roots.

The part of the underground stem attacked by this fungus is just above the insertion of the cotyledons (epicotyl) and the affection may also extend a little below into the hypocotyl and pass imperceptibly into the tap root, but the roots are rarely invaded. In the neighbourhood of the epicotyl the whole circumference of the seedling becomes a continuous dark brown lesion which may extend as far as the base of the tap root, and in advanced disease this region may become completely girdled by a dense blackening of the tissues, and the shoot may thus become severed from the roots. The term 'foot rot' is, therefore, an apt title for this type of disease which destroys the basal part of the stem underground.

The pycnidia of *M. pinodes* are difficult to differentiate from those of *A. pisi*; they are, however, darker in colour, distinctly black when mature, and instead of being aggregated towards the centre of the lesion as in that fungus, they are relatively sparse and strewn over the whole lesion but occur more densely near the edge. Moreover, the spore exudate at the ostiole is of a light-buff to flesh colour, not red, and is not nearly so copious as from pycnidia of *A. pisi*. In practically every case of inoculation with the pycnospores of *M. pinodes* the perfect ascigerous fructifications come up easily, and the fungus is



FIG. 293.—Leaf, stem, pod spot, and foot rot of pea, all caused by *Mycosphaerella pinodes*. *A*, five plants showing the foot-rot condition. *B*, the leaf-spot condition. *C*, the pod spot showing lesions on the pod wall and along the sutures (photos by Ogilvie, by permission of Long Ashton Res. Station)

therefore immediately distinctive from the related types described here, which do not possess perfect forms. The pycnidia are minute, sub-globose, from 75 to 175 by 60 to 160 μ, averaging 134 by 128 μ [34]; pycnospores are hyaline, oblong with rounded ends, straight to curved, slightly constricted at the septum, and altogether approximate very closely to those of *A. pisi*; they measure from 8·0 to 18·0 by 2·5 to 5·5 μ, averaging 13·6 by 3·8 μ [34], and are mostly 1-septate, with numerous oil globules [20, 34]. The perithecia of the ascigerous stage are found on leaves and stems in abundance after prolonged wet weather and begin to appear on the necrotic lesions in about 13 to 21 days from infection [20, 23]; they are also developed in culture [34]. Perithecia are round, brownish in colour, 100 to 300 μ in diameter, and sunken in the host tissue save for a short neck portion; asci range from 60 to 80 by 12 to 16 μ, thickened at the apex and at maturity provided with a pore; ascospores, 8, hyaline, ovate-elliptical, 1-septate with constriction, range from 12 to 15 by 6 to 8 μ [39a]. In culture on oatmeal agar, at a similar range of temperature and hydrogen-ion concentration as for *A. pisi*, the amount of aerial mycelium made by *M. pinodes* is relatively more than shown by *A. pisi*; at first it is white, but soon turns grey to dark grey with age. It sporulates much more rapidly than *A. pisi*, and at a slightly higher optimum temperature, between 20° and 25° C., but the pycnospores of the two fungi germinate alike by the production of 1 to 3 germ-tubes [34]; in cultures the dimensions of the ascospores are slightly greater than given above; thus, on oatmeal agar, 18 to 19·8 by 7·2 to 9·9 μ [20]. Chlamydospores and pycnidia appear on pea-straw agar [36a].

Pycnidia and perithecia are capable of surviving the winter on old pea stems [15]. While the pycnospores, as in the case of *A. pisi*, are disseminated largely by splashing of rain to plants in the immediate neighbourhood, the ascospores on the other hand, by being ejected into the air and borne by wind have a wider range of infection. In illustration of the comparative range of action of pycnospores and ascospores, an experiment is recorded where individual plots inoculated with *A. pisi*, *M. pinodes*, and *A. pinodella* (see below), situated 5 feet apart, when examined a month after inoculation showed no spread of infection by any of the three organisms beyond their own plots, but as soon as the ascospores of *M. pinodes* were produced and active, infection from this fungus was general on all the plots [20]; the optimum temperature for growth lies between 20° and 28° C. [11a].

Inoculations, as with *A. pisi*, may be performed by spraying the plants with a spore suspension, or the seeds may be smeared over with a culture and planted in sterilised soil. The latter method of inoculation when performed with *M. pinodes* is far more damaging to the seedlings than is the case with *A. pisi*, and while in that instance the great majority of seedlings manage to emerge from the soil, in this method of inoculation with *M. pinodes* all seedlings perish in the soil. Clearly, *M. pinodes* in its capacity as a severe foot-rot organism causes much more damage than the leaf and pod spotting *A. pisi*.

For the control of this leaf, pod-spot, and foot-rot trouble, the same treatment is recommended as outlined for that caused by *A. pisi*, and there appears to be a marked reduction in the amount of foot rot by seed treatment with organic mercury compounds. In the recommendation of the varieties Rice's No. '330', Horal, and Blackeye Marrowfat, as being more resistant than others to foot rot, it is interesting to note that these varieties have a thicker cuticle over the vulnerable basal part of the stem than have susceptible kinds over the same region, but whether this property is inherent requires confirmation.

Ascochyta pinodella L. K. Jones

This organism was once thought to be a varietal ' micro-form ' of *Mycosphaerella pinodes* [26] but is now considered to be a distinct species [20]. Only pycnidia are known. Together with the two foregoing types, this fungus occurs over the same geographical range as already indicated for those types. In the United States it is more prevalent in the drier regions of the west, and often in a few of the eastern parts as well, but in these places occurs only on the one host, the pea [38]; in Alabama, in 1937, it was reported to occur on winter peas and vetches [35]. In other localities numerous different strains of *A. pinodella* attack, besides the pea, lupin, broad bean, lentil, vetch, and soya bean, but all strains of the organism are more pathogenic to the pea than to any other host, and least of all to soya bean [28, 29, 34].

Like *M. pinodes* this organism attacks leaves, stem, and pods, and causes foot rot. In its virulence as a foot rot fungus it is about equal with *M. pinodes*, though sometimes reported to cause greater damage in certain localities [11a].

In contrast with attacks due to *M. pinodes* on the leaves, spots caused by *A. pinodella* are not so numerous and tend to be more regularly circular and zonate; on the pods the spots are more distinctive, being very small, brown to black, and scattered in characteristic fashion *obliquely* to the long axis of the pod. On the stem and basal parts underground the lesions vary in size considerably, are purplish to dark brown, and resemble closely those caused by *M. pinodes*.

Pycnidia of *A. pinodella* are scattered at random over the lesions and are very indistinct, being depressed into the tissue. They are globose, ostiolate, light to dark brown, and range from 79 to 182 μ in diameter [20]; the pycnospores are distinctive, being *unicellular*, rarely 1-septate like those of *M. pinodes* or *A. pisi*, and they form practically no exudate at the ostiole; they are oval to oblong, 5 to 12 by 1·5 to 4·5 μ; if 1-septate spores occur, they are from 5 to 13 by 2 to 4·5 μ [20, 34]. Pycnidia occur on root lesions and on the seed coat [10]; they occur, with chlamydospores on pea-straw agar [36a].

In culture on oatmeal agar of alkaline reaction, or *p*H 7·0, over a temperature range of 18° to 23° C., a fair quantity of whitish grey, aerial mycelium is formed within four to seven days. Here again, as under natural conditions, the pycnidia form hardly any exudate of spores at the ostioles. Spore germination is rapid, at an optimum temperature between 20° and 30° C., and the unicellular spores produce only single germ-tubes.

This fungus invades the epicotyl of the seedling in a distinctive way; instead of creating a general brown discoloration encircling the epicotyl as produced by *M. pinodes*, there is first formed a dark-brown *triangular* lesion, and from it infection spreads up and down through the cortical tissues, even down into the tap root, but again, as in *M. pinodes* it is not usual to get much root infection [10]. With advanced disease, however, the lesions at the epicotyl caused by these two fungi cannot be distinguished, for in both cases the epicotyl becomes completely girdled. Furthermore, when the vascular system is invaded it is not long before the roots become severed from the shoot. In general, *A. pinodella* appears to be more destructive to the tissues of the basal parts of the stem than *M. pinodes*, for while slight attacks by either fungus may extend no further than the endodermis

in the region infected, in bad cases, prior to girdling, the stem shrinks so much after an attack by *A. pinodella* that there is practically nothing left at the 'foot' except strands of sclerenchyma [10].

A. pinodella like *M. pinodes* is carried on the seed and good results are reported to follow the same treatment of seed dusting. No variety of pea is known to be immune from this disease, and with regard to relative resistance of certain varieties, the information available appears to apply to all three types, *A. pisi*, *M. pinodes*, and *A. pinodella*.

Foot Rot of Peas, *Fusarium solani* (Mart.) Sacc. var. *martii* (Appel & Wr.) Wr., *f. 2*, Snyder

This type of foot rot, which may be called a 'fusariosis', is common in all pea-growing areas in Britain, United States, Canada, France, Holland, Germany, and Denmark [2, 16, 30]. It appears to have been first described in Holland under the name 'St. John's Disease' [11, 44]. In Britain, this particular disease of peas is associated with a varietal form of the fungus *Fusarium solani* in conjunction with the eelworm *Heterodera schachtii*, and the separate effects of fungus and eelworm on the host appear to be difficult to estimate. There is, however, no doubt about the pathogenicity of the fungus in producing foot rot (Fig. 294) by inoculation with a pure culture, but the characteristic symptoms which are associated with the death of the plants following natural infection are not exactly identical [31, 32].

This disease breaks out in scattered areas over the crop, but there are no spots on the leaves or the stem, as in the three types described above. In this respect it resembles the root rot caused by *Aphanomyces euteiches* and, like it, is also accompanied by a general yellow discoloration of the leaves. Presumably from disturbance set up by the presence of the fungus in the basal parts of the stem, the leaves turn yellow all over; and while more than one fungal organism may be responsible for this interference, the species of *Fusarium* above named is believed to be the one mainly associated with foot rot of peas in Britain and Holland. In both countries the disease has been found in high- and low-lying areas, in Holland chiefly on clay soils, but in Britain on many types of soil, light and heavy [30, 31, 32, 44].



FIG. 294.—Fusarium foot root of pea (*Fusarium solani* var. *martii f. 2*) (photo by Ogilvie, by permission of Long Ashton Res. Station)

Here, too, as in the types above described, the parasite attacks its host near the point of attachment

of the cotyledons to the axis. At the point of entry, as in the case of *A. pinodella* the early lesion is triangular in shape, but that caused by this *Fusarium* is a darker, reddish-brown colour, not brown, and later this region becomes blackened as far as the top-most roots. Moreover, the lesions are not sunken into the affected part until a much later stage of decay, and when eventually the vascular stele becomes involved in infection this disease is distinctive in that the stelar strands take on a bright orange-red colour which may extend as far as the first node ; furthermore, the dark lesions may often be seen at the base of the tap root as well as the bases of lateral roots [16].

With regard to the title of this organism, Snyder [36] states, on the ground that spore measurements have not been shown to be sufficiently distinctive from a taxonomic standpoint, that the species of *Fusarium* variously designated *F. martii* vars. *minus*, *viride*, *pisi*, and *phaseoli* are all of the same morphological type, namely *Fusarium solani* var. *martii*. According, therefore, to their distinctive physiological behaviour on their respective hosts, he proposed for each organism a suffix to this name, thus, for the one attacking the pea, the addition of '*forma 2*' ; for the fungus on the runner bean, '*forma 3*' (p. 607), so that the present fungus is styled *Fusarium solani* var. *martii f. 2*, which is synonymous with *F. martii* var. *pisi*, under which name it has appeared in most of the literature [16, 32, 36].

It is remarkable that this species of *Fusarium* has not been observed to form any kind of spore or fructification on the host plant [16]. It is, however, easily grown in culture forming mycelium and spores ; the mycelium is short, white or greyish, sparse in quantity especially at sporulation ; old cultures are coloured green or blue from the mass effect of the diffuse sporing surface, and the general colour of the medium is vinaceous. The spores, in sporodochia, are typically curved, mostly 3-septate, of nearly uniform diameter, and range from 27 to 40 by 4·5 to 5 μ ; small microconidia are also formed but not abundantly ; chlamydospores may be developed in the mycelium, as intercalary or terminal cells, or in chains, or within the spores ; sclerotia are rare but have been found to develop on rice media. Under natural conditions the organism survives in the form of mycelium in the soil ; it is not carried by seed. Its extension in the soil, however, is relatively slow, not more than 2 or 3 feet in a period of two years [16].

The degree of moisture in the soil does not appear to be a critical factor in relation to the progress of this disease. Though a wet soil is undoubtedly favourable to the decay of the infected host, it does not help the fungus itself under all conditions. In Wisconsin, however, attacks by this organism and also by *A. euteiches* were mainly restricted to rainy seasons and wet soils, clays, and heavy silt loams, but in Britain, after a period of heavy rainfall (as in April and May 1932) an attack by this *Fusarium* with the help of the eelworm reached epidemic proportions independently of water-logged conditions [30].

Soil temperature plays a more important part than the degree of moisture in the incidence of this disease. If soil humidity is constant, the first sign of disease after inoculation occurs in about 20 days when the temperature is 27° C. This consists of a general wilting and is followed by the characteristic symptoms of a rotting of the cortex, and a reddish-brown discoloration of the vascular strands in the foot region, which may extend for some distance above the soil into the stem. With continued incubation plants may wilt at lower temperatures, thus after 35 days this took place at 24° C., but below this temperature affected plants, though practically girdled by cortical lesions, managed to survive, and in them the fungus

had not penetrated as far as the vascular stele, so that they were enabled to grow forward by virtue of new roots developed above the lesion. The optimum soil temperature for the development of the disease appears to lie between 24° and 33° C., the latter being the upper limit at which the pea plant can normally thrive; plants exposed to infection below this range contract only superficial or shallow cortical bruises from which they can usually recover, and at a comparatively low temperature of about 15° C. they suffer no injury [16].

This type of foot rot, carried by soil, is best controlled by suitable crop rotations. Since the serious phase of vascular invasion of the plant does not usually take place after the early stages of germination have been passed through successfully, maintenance of vigour is important, and the drilling of fertilisers with the seed substantially reduces the losses from foot rot on heavy soil [7].

There do not appear to be any varieties of peas immune from this foot rot. The varieties Green Admiral, Yellow Admiral, Horal, and Rice's No. 330 are resistant in Canada but not in Britain [31].

The main differences between these five diseases of the pea, and the causative organisms are :

Aphanomyces euteiches	*Ascochyta pisi*	*Mycosphaerella pinodes*	*Ascochyta pinodella*	*Fusarium solani* var. *m. f.* 2
Causes a root rot	Leaf, stem, and pod spot. Not usually a root or foot rot	Leaf, stem, and pod spot and a foot rot	Same as *M. pinodes*	Foot rot
No external symptoms above ground except general yellow discoloration and wilting	Spots on leaves and pods tan to brown, round, sunken, with black, thickened edges surrounding paler tan centre. On stems and petioles long, tan lesions	Spots on leaves, stems, and pods brown to purplish, not so definitely delimited as of *A. pisi*. Stem spots mostly on nodes in black to purplish streaks	Spots on leaves, not distinctive from *M. pinodes*, but spots on pods smaller brown black arranged *obliquely*. Stem lesions same as *M. pinodes*	Same as *A. euteiches*
Oogonia, antheridia, and oospores	Pycnidia only, in a group at centre of lesion	Pycnidia and perithecia. Pycnidia scattered but denser near edge of lesion	Pycnidia only, scattered over lesion, very indistinct, sunken into the lesion	Only mycelium, in nature; spores only in culture
Oospores	Spore-tendrils red; spores, 10–18 × 2·5–5·5 μ; mostly 1-septate	Spore-tendrils light buff or flesh-coloured; spores, 8–18 × 2·5–5·5 μ, mostly 1-septate	No tendrils, spores, 4·5–13 × 1·5–4 μ, *unicellular*, rarely 2-celled	Spores curved, 3-septate, 27–40 × 4·5–5 μ

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Pea Mosaic

This virus disease of the garden pea is common throughout Britain, Europe generally, and the United States, and has been reported also from New Zealand (4, 12, 17, 22). It has a wide host range, which includes sweet pea (*Lathyrus odoratus*), crimson clover (*Trifolium incarnatum*), red clover (*T. pratense*), white sweet clover (*Melilotus alba*), alsike clover (*T. hybrida*), broad bean (*Vicia*



FIG. 295.—Pea mosaic (photo by Foister & Noble)

faba), blue lupin, common vetch (*V. sativa*), and others [4, 11, 15]. In some years yield is appreciably reduced, losses in Australia in 1935–6 being 47·7 per cent. [5], and seedlings of the variety Greenfeast suffered a loss of 17·4 per cent., due to mosaic, though later infections had little effect on the yield [13].

The first symptom of the disease on garden peas is a clearing of the veins, that is, the appearance of light-coloured areas along the veins of the young leaves. This network of mottling is later replaced by a more general mottling in which the light areas often occur between the veins. Affected plants are stunted and pale, with small, occasionally distorted leaves (Fig. 295). They flower later than healthy plants, their pods are fewer, smaller and scantily filled, and ripen later than usual. On several varieties the symptoms are variable. Thus a general chlorosis tends to develop on Alaska and Telephone while distinct mottling is observed on Alderman, World's Record, and Market Surprise. On broad beans, first symptoms, consisting of a spotting of the leaves or of a clearing of the veins, give place later to a conspicuous mottling [15]. Infection of red and crimson clovers is characteristic; the former under field conditions showing paleness and stunting with dwarfed leaves exhibiting a definite mosaic pattern; the latter by a brilliant mottling of the leaves with severe stunting [12, 15]. Red clover is an important 'indicator' host for the present virus, for other viruses of the pea are not transmissible to red clover (*T. pratense*). On the lupin the disease is known as 'sore shin'; it is characterised by a slight stunting, with the top of the plant bending over and the appearance of a light brown streak extending the whole length of the stem within the curved side.

Synonyms for the virus (*Pisum virus 2* Doolittle & Jones) of pea mosaic are: *Pea mosaic virus* (Chamberlain 1936, 1937); *Garden pea mosaic virus* (Dickson 1921); *Common pea mosaic virus* (Pierce 1935); *Pea virus 3* (Pierce 1935, Murphy & Pierce 1937); *Pea mosaic virus 1* (Zaumeyer & Wade); *Pea virus 2* (Osborn) 1937; *Pisum virus 2* (K. M. Smith); *Lupin sore shin virus* (Neill *et al.* 1934, *fide* Chamberlain 1935). Different strains of the virus exist [1, 9]. Three, somewhat distinctive types, of pea mosaic are recorded in America [19], which all differ from the one described here, in that the viruses are not transmissible to red clover. The present disease is also different from 'enation pea

mosaic ', known in England and the United States [1], caused by another virus. The virus of pea mosaic is sap-transmissible [4]. It is spread through the agency of the aphides *Macrosiphum pisi*, *Myzus persicae*, and *Aphis rumicis* [3, 4, 15]. No ' incubation ' period appears to be necessary for the infective principle, but the capacity for infection is early lost after feeding on healthy plants. Infection takes place within 15 minutes of insect visitation, even by single alate or apterous vectors, but infection is heavier the greater the number of the vectors [15]; others have found little infection to take place by the flying type of aphids [13]. The virus is not seed borne [11, 12].

Numerous varieties of peas are reported to be more or less resistant to pea mosaic in America [11]. Selection, and breeding experiments in New Zealand, showed the varieties Lord Chancellor and Little Marvel to be immune or highly resistant, with promise of still better results towards immunity [5, 6].

For the control of pea mosaic, the crop should be grown as far away as possible from other susceptible plants, notably red clover, in which the virus is known to over-winter, whence it may be spread to the pea crop by aphides in spring and summer [3, 6, 22].

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Marsh Spot of Pea

(*non-parasitic*)

' Marsh spot ', a deficiency disease, derives its name from the fact that it was first observed chiefly in peas from crops grown on low-lying marshland near the sea [17]. It has, however, been found in many places remote from the sea and on soils known not to be reclaimed marshland. The trouble is fairly common in Europe, especially in Holland, and has probably existed in Britain as far back as 1845 [17]. Symptoms similar to those of marsh spot have also been observed on broad beans in the field, and on runner beans [2, 14, 17]. In some districts it

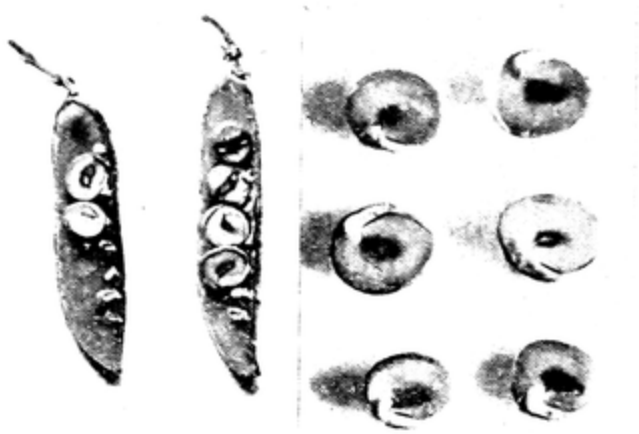


FIG. 296.—Marsh spot of pea (photo by Foister & Noble)

causes considerable financial loss, some areas being quite unable to raise the crop [4]; in other affected areas losses are reported to be negligible [11].

The disorder affects the seeds of the culinary pea, and though it has been encountered in processed canned peas, so far it is not serious in the canning industry. Marsh spot is not caused by any organism, and the trouble may develop in a crop grown from perfectly healthy, as well as from affected, seeds from the same stock [4, 21]. Though the cause of the trouble has not been satisfactorily explained, the general trend of opinion is that it is due mainly to a deficiency of available manganese in the soil [3, 8, 12, 13, 15, 18].

Symptoms of marsh spot are not discernible until the peas are fully developed in their pods. Healthy and diseased seeds may lie together in the same pod, and pods containing healthy seeds or diseased seeds may grow on the same plant. Except in severe attacks affected seeds show no unusual symptoms from the outside and the testa has to be removed and the cotyledons separated before the typical symptoms are viewed. These consist of one or more discoloured spots on the inner surface of each cotyledon, usually at the centre, and hardly ever extending over the entire inner surface (Fig. 296). At first these spots have a water-soaked appearance and may be superficial or involve a fair depth of the cotyledonary tissues. Eventually one or more pockets filled with shrunken cells of a rusty red or greyish colour are formed below the spots [2]. Owing to the shrinkage of the dead cells, a cavity arises below a spot and the skin of the cotyledon may remain stretched taut over it like a drum [17]. Sometimes the tip of the plumule is also affected and similarly discoloured.

When the lesions are confined to the cotyledons germination of the affected seeds is not prevented, but if the plumule is also involved in the necrosis normal germination is interfered with. The plumule may perhaps grow for a little while in a distorted manner but soon becomes exhausted if the growing point is necrosed. Affected seedlings may, with varying degrees of success, produce young branches in the axils of the cotyledons, replacing the plumule which remains as a withered stump. Plumular injury is not often met with unless the cotyledons are also affected [11]. Apparently, some varieties of peas, e.g. Harrison's Glory, when heavily affected, produce injured plumules, and the lesions on the cotyledons are unusually deep.[5]

Water-culture experiments have shown that seedlings grow normally for the first five weeks or so, but afterwards those receiving no manganese die. The foliage of seedlings deprived of manganese develop a severe mottling of the young leaves and brown lesions may be seen on the internodes and near the growing tip of the stem, all growth coming to an end two or three weeks before the flowers

are produced on plants receiving normal treatment. The addition of manganese to the cultures, at the rate of 500 mg. per litre, produced vigorous growth, the plants flowering freely and yielding a heavy crop of seeds [19]. In the field it has been observed that seeds in the later-formed pods of individual plants were more severely affected than those formed earlier, this being probably due to the larger amount of manganese still present in the latter [3]. More affected seeds are usually found among the large peas of a sample than among the smaller ones [2, 4]. There is no proof that marsh spot can be transmitted by seed [11].

There is abundant evidence that the content of soluble manganese is low in soils which produce symptoms of marsh spot in peas. While small amounts may be sufficient to meet the early requirements of seedlings, they may not be adequate to enable them to reach fruition with abundant seed production [19]. Experiments have shown that peas exhibit normal growth and production on soils with more than 0·3 mg. per cent. of manganese [8a]. It is, however, not an easy matter to determine in advance what soils are liable to cause marsh spot disease. Thus, on the Lincoln silt soils containing small quantities of readily soluble manganese the disease occurs where the amount is particularly low. On the Romney Marsh soils, which contain considerable amounts of manganese oxides, marsh spot has not been found at *p*H values below 7·0. In the field, soluble manganese may be produced from the oxides in various ways: by reducing processes, by the fermentation of crop remains, by the application of farmyard manure, and acidifying fertilisers such as sulphate of ammonia [8] (*vide* 'grey speck' of oats, p. 420). It has further been established that the mineral is more available to the plant in acid than in alkaline soils, but even in the former the pathological symptoms develop in the absence of manganese, indicating that soil reaction is not the only factor concerned [3]. In general, observations agree that most soils which are slightly to moderately acid contain sufficient manganese for normal plant growth [11]. While there is little doubt that the availability of manganese in the soil is affected by liming, the presence of lime in the soil is not a primary factor which may favour marsh spot, and the trouble is apparently not aggravated by a deficiency of potash or phosphates in the soil [4].

The incidence of marsh spot has also been considered in relation to the presence of a 'water-table' in the soil [1, 4]. In a deep, well-drained area the plants are probably provided with all the minerals they need for full development. In poorly drained soils, however, it has been found that the trouble was prevalent in all the soil series in the field when the water-table was closest to the surface. Marsh spot was more severe on the heavy soils than on the light, one reason probably being the difference in the conducting power for drawing water possessed by soils of different texture. In this connection, however, important differences have been observed and it is apparently not safe to conclude that water-table and soil texture by themselves aggravate marsh spot although they may be contributory [18].

A micro-analysis of the content of seed affected with marsh spot has shown the presence of manganese in the peripheral tissue of the cotyledons, the embryo (plumule and radicle), and the seed coat. The distribution of manganese is not, however, equable throughout all parts of the seed. The percentage is higher

in the tissues at the centre of the cotyledons than in the testa, and higher again in the plumule and radicle than in the outer layers of the cotyledons. Accordingly, the suggestion is made that the migration of food contents from the central necrotic tissues in the cotyledons may partly account for the difference in manganese content of healthy and diseased peas [2]. The cytoplasmic contents early disappear from the necrosed cells, and the starch grains may disappear entirely [17] or be left in a dry condition in the dead tissues [7]. There is no development of a cork layer between the necrosed and unaffected tissues in the cotyledons, and in the browned plumules necrosed areas have been seen in some cases to extend as far as the apical bud, though microscopic tests revealed the presence of suberin in the diseased walls in this region [7]; but a brown deposit on the cell walls and in the intercellular spaces of the affected organs is thought by others to be merely a brown gummy deposit [17].

There is little doubt that varietal susceptibility to marsh spot exists among different kinds of peas. As already stated, there is some evidence in the field that the larger-seeded, heavy varieties are more severely affected than the lighter ones, but this has not been borne out in culture experiments [3, 4]. The trouble has been observed to be worse in the later main crop than in the early-maturing varieties, with small seeds [6, 11]. Moreover, roundness of seed was also associated with earliness of maturity and resistance to it [6].

The varieties Union Jack, Earliest of All, Early White Seedling, First and Best, King of Serpette, Serpette, William the Conqueror, are usually free from marsh spot, while Peerless, Prestige, Sutton's V.C., Onward, and Giant Stride have proved susceptible in the areas where they were tested [6].

The application of potassic manures, without the addition of nitrogenous fertilisers, has been shown to reduce the amount of marsh spot, and a balanced manurial treatment, or high nitrifiable nitrogen tended to increase the trouble [2, 8a]. Others have failed to confirm the efficacy of potassium, under all conditions; and there is apparently no correlation between previous croppings of an area and inducement to the development of marsh spot [4]. Several investigators have found beneficial results to follow the spraying of the crop with a weak (0·25 to 1 per cent.) solution of manganese sulphate, and of the application to the soil of heavier dressings, at the rate of 1 to 2 cwt. per acre [10, 15, 16]. Manganese-deficient sand cultures at Long Ashton showed that while peas showed the usual symptoms of marsh-spot, broad and runner beans were more resistant; and dwarf and runner beans, while not free from leaf symptoms, remained more resistant [9].

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Chapter XIV

DISEASES OF VEGETABLES

Leaf Spot of Celery, *Septoria apii* Chester & *Septoria apii-graveolentis* Dorogin

This is the most serious disease of celery in the British Isles [1, 2, 9, 10, 25, 26, 28, 35], and occurs also in many other countries. In America it is termed ' late blight ' to distinguish it from the ' early blight ' due to *Cercospora apii*. Two forms are found, a ' large spot ' and a ' small spot ' type, once regarded as due to varieties of the same fungus *Septoria apii* [20], but now held to be caused by distinct though closely related species, *S. apii* [7, 8] and *S. apii-graveolentis* [14] respectively. There is no evidence that these two types of disease ever change from one to the other; in the field each is usually found alone, though combined attacks have often been seen in the Argentine [18]; and in cultural and pathogenic studies each of the two parasites appears to preserve its identity indefinitely. Another closely related fungus, *S. petroselini*, occurs on parsley but the diseases on the two crops are not inter-transmissible [2].



Fig. 297.—Leaf spot of celery (*Septoria apii* and *Septoria apii-graveolentis*). Leaf showing the small spot type of the disease (caused by the latter organism). Insets, top, the entire fruit; below, the two one-seeded, half fruits showing the small black pycnidia (photos, Lft. 241, by permission of Minis. Agric.)

S. apii-graveolentis is found chiefly on the expanded green parts of the leaf but may fairly often be seen also in lesser amount on the blanched edible leaf stalks (Fig. 298), where *S. apii* rarely occurs [11, 14]. Plants heavily spotted by the former may be only half their normal size and owing to reduction in assimilatory activity their leaf stalks are thin, dwarfed and unpalatable. The small spot type (Fig. 297) is much the more severe and it alone is responsible for the epidemic outbreaks that have been reported in England when the spotted form changes to a blight that may destroy the crop [35]. The mature spots vary from 0·5 to 2 mm. in diameter and are without sharply defined margins, though the edges may be raised; adjacent spots often



FIG. 298.—Leaf spot of celery. Leaf stalk severely affected; the minute black pycnidia are on the surface of the lesions. Inset, left, upper surface; right, under surface showing three spots on young leaflets (after Salmon & Ware, Wye Reports)

fuse. Pycnidia are usually visible as numerous small black dots on and outside the spots (Fig. 299 A), sometimes appearing in great numbers on both surfaces of leaves and stalks showing no other obvious signs of infection [27].

S. apii is less common and the large irregular spots, 3 to 10 mm. in diameter, that it produces almost entirely on the leaf blades are usually widely spaced, bear only a few scattered pycnidia or none, and are less frequently followed by extensive secondary infections than the small spot type. The mature spot is demarcated from the sound tissues by a reddish-brown rim, outside which pycnidia do not occur nor have they been seen on the stalks. Both types begin as small chlorotic flecks, those due to *S. apii* turning brown in the centre earlier than those of *S. apii-graveolentis*, the incubation period in which is some two days longer than that of *S. apii* [11]. The latter causes relatively little injury either in America or in Britain, where it has never been reported in epidemic form or to result in serious depreciation of the crop [35].

The mycelium of both species in the leaf is composed of brown, sparingly septate, intercellular hyphae, from 1 to 5 μ across, which extend beyond the spot in *S. apii-graveolentis* but do not reach to its margin in *S. apii*; the latter therefore kills the cells in advance of its growth. The pycnidia occur on both leaf surfaces. Those of *S. apii* are sparse, globose, 58 to 95 μ in diameter, and restricted to the centre of the spots; they contain straight or slightly curved pycnospores, measuring about 11 to 43 by 1 to 2·5 μ, with up to 4 septa. Those of *S. apii-graveolentis* are crowded, sub-globose, from 60 to 150 μ in diameter, and not restricted to the spots; their spores are slightly flexuous, blunt, measure 17 to 61 by 1·5 to 3 μ (Fig. 299 B, D), and may have as many as 7 septa (though in both species 3 is the commonest number and in both the septa are visible with difficulty) [11, 18]. Pycnidia also occur deeply embedded in the wall of the two-seeded

fruit, which, when ripe, splits into two halves, each firmly retaining its seed, so that the celery ' seed ' of commerce consists really of one-seeded half fruits (Fig. 297, insets).

On spore germination the number of septa may increase or the spore may break up into segments, each able to give a germ-tube. Secondary non-septate spores may be borne on the pycnospores. In culture, growth is best at comparatively

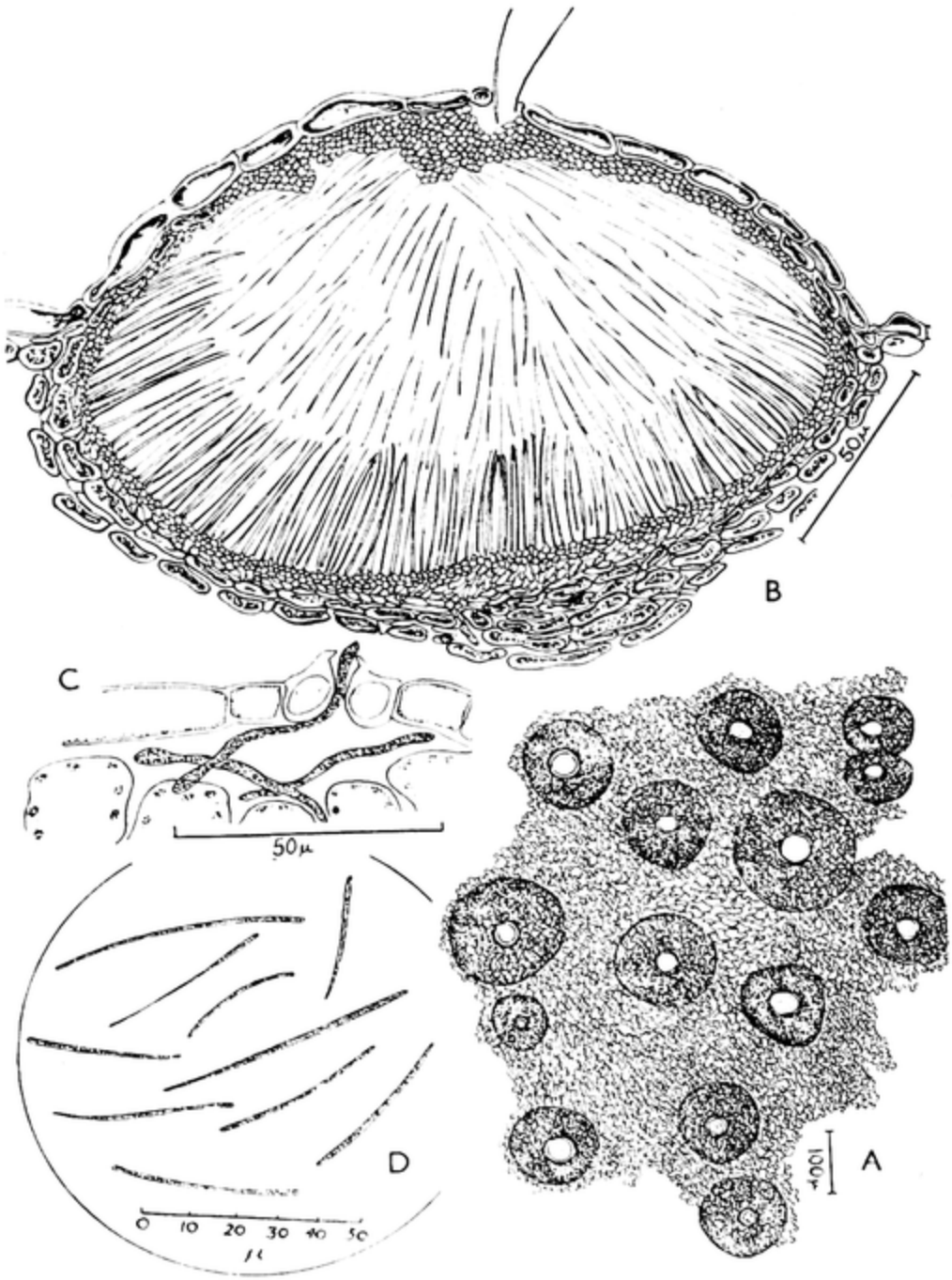


FIG. 299.—*Septoria apii-graveolentis*. *A*, surface view of portion of leaf affected with the small spot type of celery spot, showing the small sub-epidermal pycnidia and ostioles. *B*, portion of leaf section showing a pycnidium ; note the extremely short conidiophores cutting off the long, needle-shaped conidia. *C*, stomatal penetration at infection. *D*, the conidia showing septa. (*B*, from a slide by Macfarlane)

low temperatures, ceasing about 27° C. in *S. apii* and 25° C. in *S. apii-graveolentis*. Infection occurs through the stomata (Fig. 299 C) or more commonly directly through the epidermis. The earlier (primary) attacks on the cotyledons and young leaves come from spores extruded from pycnidia embedded in the pericarp of the 'seed'. The best conditions for germination of the latter and those for extrusion of spores, and for their germination and the infection of the young leaves, are quite similar, so that when the cotyledons emerge from infected seed they become contaminated by contact with the long spore tendrils given out in moist weather from the pycnidia; this is especially likely to happen under the conditions of frame cultivation. Spores also reach the soil at this period, or later from the shed pericarp shells, and may be disseminated to the young seedlings by splashing. Primary spots are found on the seedlings from 9 to 12 days after germination, but it does not appear that mature pycnidia ordinarily develop while the plant is in the seedling stage, that is, before transplanting, so that secondary spread to the aerial parts is not seen in the seed bed. After transplanting, secondary infection of the aerial parts occurs from the pycnidia on the primary lesions, dissemination of the spores being both by rain-splashing and by contact. Infection in the seed bed can, however, occur below the soil, for it has been established that the roots can be attacked from germinating spores or mycelium in the soil and that infection can pass from root to root [3]. Over-wintering in the soil has not been found to occur, but conidia may continue to develop on decayed celery tissues from the previous crop and spores from dead leaves may retain vitality for nearly a year [11, 16]. In the deeply immersed pycnidia in the seed coat, viability has been preserved under good storage conditions for several years, but as a rule seed more than two years old can be regarded as fairly safe [6, 32].

The disease causes most injury when conditions favour the germination and growth of the fungus but retard growth of the host, as for instance during dry spells with cool dewy nights [3, 34, 35]. As already mentioned (see Chapter V, p. 175), in a general way anything that favours the growth of the host increases susceptibility to infection as judged by the number and size of the spots [36]; moisture and the application of nitrogenous or 'complete' fertilisers [37] may greatly increase the spotting, but they may also increase the growth of the host by so much that the net injury to the crop is lessened. In both the late and early blights of celery there is a lower percentage of total nitrogen in diseased than in healthy tissues [13].

Control of celery leaf spot is based primarily on the exclusion of infection from the seed. Scarcely any sample of untreated commercial celery seed is free from *Septoria* which may sometimes be found on over 80 per cent. of the seeds. In New York State it was estimated that one pycnidium from a seed sample released 3,675 spores [21]. Seed completely free from *Septoria* was produced in England in 1928 by intensive spraying of the plants kept as seed bearers, with 4 : 6 : 40 Bordeaux mixture applied twice in each of the months May, June, and July, and once each in August and September, the last four applications being made when the plants were flowering and setting seed [35]. This was in a year when leaf spot was severe, and usually four applications (before transplanting, at 6 inches, at 15 inches, and at maturity) will yield clean seed. 1 per cent. Bordeaux

mixture has also been successfully employed in Germany [15]. In the seed bed and in the field 5 : 5 : 50 Bordeaux mixture gave the best results in Ohio, over four years [38]. Though growers in Britain are reluctant to use copper-containing fungicides on the plants intended for consumption, both because of the trouble and because they are eaten raw, this objection does not extend to the plants grown for seed ; elsewhere it is sometimes the practice to spray the growing crop with Bordeaux, or Burgundy mixture [24], or to dust it with 20 : 80 copper-lime dust [38], weekly applications of which are given in Alberta [32].

Seed disinfection by ordinary methods gives uncertain results owing to the deeply immersed position of the pycnidia in the pericarp. Copper sulphate, organic mercurials and the like have, however, been recommended [4, 5, 17, 19, 31]. Better results have been obtained by immersing the seed for 3 hours in very dilute formaldehyde (1 in 300 to 400, or in 2 per cent. for 30 minutes [29]) which, if anything, stimulated germination, combined with disinfection of the soil in the frames with 2 per cent. formaldehyde [33]. Still better results are claimed for treatment of the seed with hot water ; 10 minutes at 48° C. has proved effective in Germany [16], 30 minutes at 118° F. in the United States [12], and 10 minutes at 136° F. in New Zealand [23]. Immersion in hot water should be followed by a dip of 2 minutes in cold water. The last-mentioned high-temperature bath is reported to reduce the germination of new seed by 10 per cent. and of two-year-old seed by 50 per cent., so that a preliminary test on a small sample is recommended.

Crop rotation is advisable if there is risk of a carry-over in the debris from the last crop [30]. There is some evidence that the spores of both early and late blights of celery may be carried on workers' clothing [22]. Watering from above is dangerous on account of splashing, once pycnidia appear. No commercial variety of celery is reported to have any pronounced degree of resistance to the disease [18, 36].

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White Blister of Cabbage, *Cystopus candidus* (Pers. ex Chev.) Lév.

This disease is common on cruciferous plants, which include turnip, cabbage cauliflower, radish, mustard, cress, rape, and many weeds [1, 2]. It is of little economic importance but is often reported to cause a good deal of damage to cabbage and its varieties. The causal parasite *Cystopus candidus* (Peronosporales) is found to be frequently associated in its attacks, with a near relative *Peronospora parasitica* (described below) which causes the downy mildew disease, and numerous instances are known of considerable injury from combined attacks by these two fungi.

All parts of the plant except the roots may show the characteristic white chalky pustules of this disease (Fig. 300). They are of variable size and shape on the leaves, stem, and inflorescence. Marked swelling and distortion of the attacked parts frequently result, especially in the inflorescence which may become enormously thickened, up to twelve or fifteen times the normal thickness ; floral organs become swollen, fleshy, and develop a greenish or a violet colour, and persist instead of being thrown off in the normal manner ; petals may become like sepals and the stamens leaf-like or occasionally like the carpels. The latter remain imperfectly closed while the ovules are usually atrophied, as are also the pollen grains, and thus sterility is caused. The pustules of the parasite are usually developed on the swollen parts ; the reproductive organs, oogonia and antheridia, and finally the oospores are generally found inside the swellings.



FIG. 300.—White blister of cabbage, broccoli, etc. (*Cystopus candidus*). Portion of cabbage leaf showing the floury pustules of sporangia (photo by Ogilvie, by permission of Long Ashton Res. Station). Inset, top, on leaf of broccoli (photo by Foister & Noble) (see also Figs. 52, 75)

The effects of the parasite on the host tissues are profound [3]. In hypertrophied leaves the differentiation between palisade

and spongy tissue is lost, all the cells of the mesophyll being twice or three times the ordinary size. In thickened stems the increase is due to a modification of the cortex into a tissue of large thin-walled cells with few intercellular spaces, sometimes by the development of new layers through cell division. In some hosts (cress) the cells of the endodermis and pith also multiply by division. The sclerenchyma between and over the bundles is altered to a thin-walled parenchyma like that of the cortex. The cambium of the bundles retains its activity longer than the normal and the new elements are somewhat longer, and bands of parenchyma also occur in the xylem. The epidermis increases both as regards size and number of its cells and the number of stomata is augmented. In the thin-walled parenchyma, especially in the outer layers, chlorophyll and starch are copious, and in addition there is often a reddish-violet pigment in the cell sap in the neighbourhood of the conidial beds and in places where oogonia are developed. Starch may also be found in the cambium, endodermis, pith, and other parts in which it is usually absent ; in the branches of the inflorescence the same general changes are found. The general tendency throughout is to a lessening of the differentiation of tissues particularly such as are secondary. Further, there is a similar tendency to reduce the differentiation of organs. Internally the general result is the development of a nutrient tissue of a more or less constant type. This tissue is rich in starch and up to a certain point the accumulation of food supplies is more than sufficient to counterbalance their consumption by the parasite. Later on, however, usually when spore production starts, the parasite consumes more than the host can manufacture. The result is the depletion of the nutrient cells and their consequent collapse and death. This is marked by the drying-up and turning brown of the infected parts and sometimes by complete disintegration of the soft tissues.

The hyphae are unseptate and the mycelium is intercellular (Fig. 52). Numerous very small, spherical haustoria are sent into the nutrient cells, often a large number into each cell. Branches from the internal mycelium collect below the epidermis into which numerous haustoria are sent. From these hyphae the sporangial beds arise. The sporangiophores or conidiophores arise close together under the epidermis, which is raised up into a blister ; they are very thick-walled and measure about 35 to 40 by 15 to 17 μ long. The overlying epidermal cells swell and rupture along the softened middle lamellae, exposing the sporangial bed. The sporangia or conidia are formed in basipetal chains ; the first-formed spore is not capable of germination, its function being probably to aid in the alteration of the epidermis and the final break-though. The chains of sporangia break up into their individual elements by the solution of the jointed neck (disjunctor) of callose between the sporangia. The latter are round, hyaline, multinucleate, with uniform thin walls, and measure from 12 to 18 μ in diameter. A sporangium may give rise to 4 to 8 biciliate zoospores. After coming to rest the latter develop a cell wall and form a germ-tube which is capable of infecting the host through the stomata. Distinct physiologic races of the fungus exist on various hosts [5, 8, 9].

The oogonia and antheridia are formed in abundance in some hosts and in some localities ; in others they are rarely found [4, 10]. They occur chiefly in the hypertrophied parts of stems and inflorescences (Fig. 52). The oogonia are spherical, about 50 to 60 μ in diameter, and may be found accompanied by a single clavate and much smaller antheridium. The oospore is provided with a thickened wall which turns brown, and is provided with low, blunt ridges. The mature oospores measure from 40 to 55 μ in diameter. They

survive in the soil and germinate after a period of rest of several months. Each spore may produce, within an extruded vesicle, up to 100 or more zoospores exactly like those formed in the sporangia. Primary infections are no doubt due to infection by zoospores from germinating oospores left in the soil or in plant debris from a previous crop.

Germination of the asexual sporangia occurs over a range of temperature from 1° to 18° C. They do not immediately on their attachment to a new host develop zoospores unless they are partially dried, and experiments have shown that the sporangia must suffer a loss of water content of about 30 per cent. of their maximum content before they can germinate. Under natural conditions, as on the leaf, this loss of water is directly related to the drying of the host tissues, and is, therefore, dependent on intake of water and rate of transpiration. This occurs irrespective of the age of the sporangia [7]. Cotyledons and young leaves are easily infected at the stomata, and in the substomatal cavities the germ-tubes form vesicles and with the appearance of intercellular mycelium haustoria soon become established in the tissues [6].

Treatment is on the same lines as indicated below for downy mildew [3].

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3. Butler, E. J. : 1918. *Fungi and Disease in Plants*, Calcutta.
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5. Hiura, M. : 1930. *Jap. J. Bot.* v, 1.
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7. Napper, M. E. : 1933. *J. Pomology*, xi, 81.
8. Togashi, K., and Shibasaki, Y. : 1934. *Bull. Imp. Coll. Agric. & For. Japan*, 18.
9. — and Sugano, Y. : 1931. *Jap. J. Bot.* v, 82.
10. Wagner, H. : 1895. *Ann. Bot.* x, 295.

Downy Mildew of Cabbage, *Peronospora parasitica* (Fr.) Tul.

Downy mildew attacks many plants of the cruciferous family, including most of the cultivated species. It is frequently found on cabbage, cauliflower, brussels sprouts, on young seedlings as well as on moribund leaves of grown plants, and on the plants in storage [8]. The causal fungus, *Peronospora parasitica*, produces on the host much the same effect as that caused by *Cystopus candidus* but not to the same extent. Owing to the frequent coexistence of the two fungi, it is not easy to separate their effects, but *Peronospora* produces a greater deformity in the stem; *Cystopus*, in the flowers. There is never any trace of the violet discoloration produced so often by *Cystopus* [3].

The fungus is visible as a thin, greyish-white, downy growth occurring in scattered patches on the under surface of the leaves in cabbage (Fig. 301), cauliflower, and turnip,

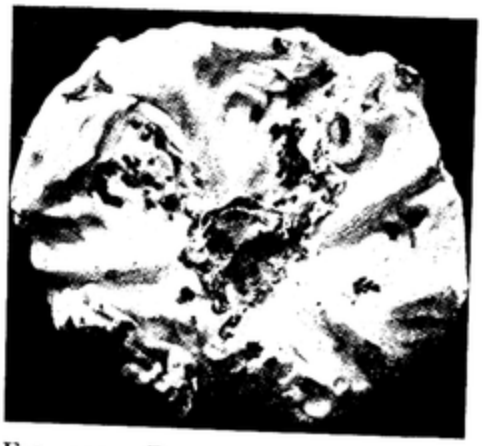


FIG. 301.—Downy mildew of cabbage, etc. (*Peronospora parasitica*) (photo by Thung)

X 2

and in the radish may often be seen on the leaves, stem, and inflorescence. The upper surface of the leaf is marked by white spots corresponding to the downy growth on the lower surface. In bad attacks the spots may be so crowded that the leaf dries up, shrivels, and tears easily. In seedlings the whole under surface may be covered, and according to favourable moisture and temperature relations and light intensity at germination, the oospores of the fungus may be found in abundance in the cotyledons while these spores may only be sparsely developed in the affected leaves [6]. Occasionally the roots are attacked ; the tissues blacken and rot near the surface, oospores are formed within the tissues, and conidiophores may be developed if the affected roots are exposed to the air.

The internal changes differ from those caused by *Cystopus* in many respects. Thus the palisade cells of the leaf are not changed and in the stem the cortex is less hypertrophied, the pith more. But, in general, the effect on the cells seems to be more destructive than in attacks by *Cystopus*. The mycelium is again exclusively between the cells ; the haustoria are large, elongated, club-shaped, twisted, and often branched, and may often be so crowded as to give the appearance of an intracellular mycelium (Fig. 83).

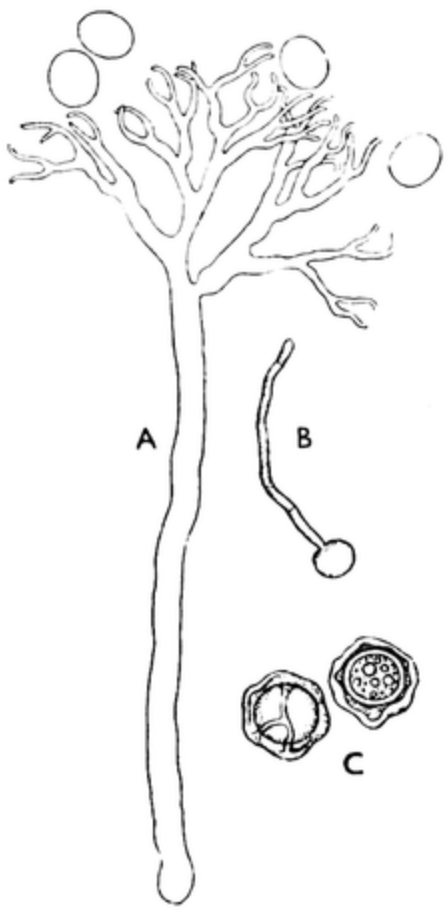


FIG. 302.—*Peronospora parasitica*. *A*, conidiophore and conidia from shepherd's purse (× 285). *B*, germination of conidium. *C*, oospores (*A*, from Butler's *Fungi and Disease in Plants* ; *B*, after Duggar ; *C*, after Berlese) (See also Fig. 83)

The numerous erect, branched conidiophores emerge through the stomata on the under surface of the leaf and those of the stem and inflorescence. The conidiophores arise directly from the mycelium and are often twisted on themselves at the base, several usually coming through each opening (Fig. 302) ; they are from 200 to 300 μ long, and bear conidia only at the branched tips, in strong contrast to the spore beds of Cystopus formed under the epidermis ; towards the top they bifurcate six or eight times, the final branches being slender, pointed, and terminating in a single conidium. The conidia are broadly oval, quite colourless 24 to 27 by 15 to 20 μ in diameter. They germinate direct by a lateral germ-tube, not by forming zoospores as in *Cystopus*, and infection occurs both by direct penetration of the epidermis and through the stomata.

Oospores are borne in the interior of the host tissues at a later period than the conidia, or sometimes (especially in hypertrophied parts) alone. They resemble those of *Cystopus* but are furnished eventually with a thicker wall which becomes swollen into crest-like folds, and is pale yellow. The oospores are globose, yellowish-brown, 30 to 40 μ in diameter ; germination again is by a germ-tube.

Infection, as for *Cystopus*, occurs most easily in parts composed of young tissues. When infected, all or most of the leaves may bear conidiophores,

while some parts, especially the stem, may show no external change though the mycelium is found to be present in their tissues, having entered them while still in a young condition [3].

P. parasitica appears to be specialised on some of its hosts, e.g. *Brassicae* and wallflowers [5], and it has been observed to be common on one host and absent from another in the vicinity. Single-spore cultures revealed slight differences of a physiological nature between numerous strains, some of which also showed differences in sexual compatibility [1].

The fungus probably survives from season to season in the form of oospores released into the soil, and in plant debris; in the turnip the fungus is stated to over-winter as a resting mycelium within the roots [4]. Little appears to be known about the effect of fertilisers in relation to the incidence of downy mildew except that cauliflowers suffering from potash deficiency are more liable to the disease [7]. Experiments have shown that the time of year at which infection takes place is more important than any effect of fertilisers on the plants. Light appears to have an important influence on the green parts of plants in relation to susceptibility. The fungus was found to be capable of attacking chlorotic cotyledons and chlorotic parts of cabbages as well as green seedlings in spring and autumn, but during the winter, green seedlings were attacked with difficulty or not at all, even at the same temperature. It appears, therefore, that a special equilibrium of food substances and the condition of the chlorophyll in the host cells are factors which influence successful attacks by the parasite [2].

The treatment consists of the removal of all cruciferous weeds capable of harbouring parasite. At the end of the season all crop refuse should be destroyed to get rid of the oospores, and rotation with non-cruciferous crops should be practised [1].

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2. — 1935. *Inst. voor Phytopath. Meded.* 72, Wagen.
3. Butler, E. J.: 1918. *Fungi and Disease in Plants*, Calcutta.
4. Gardner, M. W.: 1920. *Phytopath.* x, 321.
5. Gaümann, E.: 1918. *Beih. Bot. Centralb.* xxv, 395.
6. Le Beau, F. J., and Pinckard, J. A.: 1942. *Phytopath.* xxxii, 648.
7. Quanjer, H. M.: 1928. *Tijdschr. PlZiekt.* xxxiv, 254.
8. Thung, T. H.: 1926. *Ibid.* xxxii, 161.

Ring Spot of Cabbage, *Mycosphaerella brassicicola* (Duby) Oudem.

'Ring spot' disease is common on the leaves of cabbages, cauliflowers, and other crucifers, but occurs in Britain chiefly on broccoli. While it causes little harm to the plants in general, severe spotting of the outer leaves and stem may cause some amount of loss of foliage, and if the season is continuously wet, increase of disease from outer to inner leaves usually results in retarded growth [1, 2, 6, 7]. The disease is caused by *Mycosphaerella brassicicola* (Pyrenomycetes) [3].

All parts of the host, except the cotyledons and youngest leaves of the crown, are liable to attack, and include the stem, the more or less mature leaves, flower stalks, sepals, fruits, and seeds, but it is the outer leaves that suffer most. Leaf



FIG. 303.—Ring spot of cabbage (*Mycosphaerella brassicicola*) (photo by Foister & Noble)

spots may be few and large, up to an inch in diameter, or small, numerous, and scattered, and in severe infections the entire leaf may be involved and killed. Early symptoms on the leaves (Fig. 303) consist of small, dark-purple spots, visible from both sides, which, as they increase in area, develop different tints of discoloration in a concentric manner ('ring spot') so that the older spots have a brown centre of dead tissue surrounded by successive zones of grey and yellow brown, and sometimes the surrounding lamina may vary in colour from normal green to yellow ochre, or brown, the colour of the dead leaf [7]. The concentric rings in the spots are further enhanced when the fungus adopts the same arrangement for the production of the dark-coloured pycnidial and perithecial fructifications which may occur on both surfaces of the spots but more abundantly on the upper side. Lesions which appear on the under side of the midrib are roughly rectangular, often $\frac{1}{2}$ inch long or more and $\frac{1}{4}$ inch wide, and are of a dark neutral-grey colour. Similar lesions may also occur on the flower stalks and on the stem, on which they extend for an inch or more and may sometimes girdle it entirely. Lesions on the floral parts usually consist of small spots on the sepals, which later may become covered with pycnidia, and of tiny spots on the fruits which develop in such number as to coalesce and cover entire fruits which become shrivelled and dry. Fruit lesions may extend through the pericarp into the seed, rarely, however, penetrating further than the testa, but as the fruits perish the pericarp gets covered with pycnidia and perithecia.

Pycnidia usually appear on the leaf spots while the leaves are still green; they are sub-epidermal, dark, globose, 75 to 100 μ in diameter; pycnospores are unicellular, bacilliform, straight or slightly curved, thin-walled, hyaline, and measure from 3 to 7·5 by 1 to 2·75 μ [7]; the pycnospores ooze out through the ostioles in a pink, gelatinous thread [4]. Perithecia may occur at the same time as pycnidia, or more commonly later, in the autumn; they are flask-shaped or pyriform, 90 to 125 by 80 to 112 μ; asci are cylindrical-oblong, 8-spored, 40 to 50 by 8 to 12 μ; ascospores (Fig. 304) are bicellular, oblong or slightly fusiform, hyaline, straight or slightly curved, constricted at the septum, measuring from 15 to 25 by 3·5 to 5·5 μ [5, 7]. High humidities and low temperatures are conducive to the development of perithecia, and ascospore-discharge takes place over a wide range of temperature from 0° to 25° C.[7].

The rôle of the pycnospores in the spread of the disease is uncertain and the perithecia which may be found during the autumn, winter and spring, appear to

be solely responsible for the renewal of infections [7]. Discharge of ascospores takes place under humid conditions and the spores are disseminated by splashing rain or conveyed on wet clothes of workers [7]. Infection of the leaves is stomatal, and a closely septated branching mycelium, 2 to 4·5 μ wide, at first hyaline but later darkening, occupies the cells and intercellular spaces of the leaf. The mycelium extends from one epidermis to the other, and in about three and four weeks after infection, pycnidia and perithecia appear, respectively, from globose masses of cells laid down in the sub-stomatal cavities, at both upper and lower surfaces of the leaf. Infection is favoured by cool, moist weather; the optimum temperature for ascospore germination is between 15° and 22° C.; infections and good mycelial growth can take place at 8·8° C., but the disease declines rapidly at higher temperatures, up to 24° C.[7].

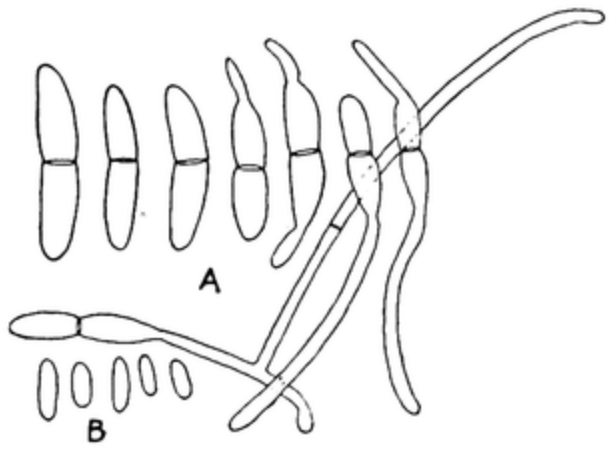


FIG. 304.—*Mycosphaerella brassicicola*. *A*, ascospores and their germination (× 1500). *B*, pycnospores (× 750) (after Weimar, *J. Agric. Res.*)

Despite possible seed infection, as stated above, when the fruits become infected, there is no clear evidence that this disease is seed borne, but as a precaution seed may be dipped for 10 minutes in water at 55° or for 30 minutes at 50° C. Spread of the disease may be largely checked by removing the outer and older affected tissues, and by burning all plant debris, and suitable rotation with non-cruciferous crops is advisable [1, 7].

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4. Osmun, A. V., and Anderson, P. J.: 1915. *Phytopath.* v, 260.
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Light Leaf Spot of Cabbage, *Gloeosporium concentricum* (Fr.) Berk. & Br.

This disease of cabbage, cauliflower, and broccoli is fairly common throughout Britain. It was first discovered in 1822, near Edinburgh [5, 6], and was again noted in 1850, in Northamptonshire [2]. It occurs also in Australia [1, 7] and has recently been reported in Portugal [4] (Fig. 305).

The most characteristic symptoms take the form of white spots on both surfaces of the outer leaves, especially on those which have already turned yellow in the normal way of senescence. The spots are arranged in a series of concentric circles, but each group of spots forming a circle is quite distinct from an adjacent circle, though sometimes circles may overlap and fuse together. On the cauliflower, the disease not only affects the fleshy midrib and lamina but the leaf



FIG. 305.—Light leaf spot of broccoli (*Gloeosporium concentricum*). The lesions on parts of the upper surface (left) and on the under surface (right) of the leaf. Inset, close-up of a lesion showing the white sporing areas (photos by Foister & Noble)

petioles and inflorescence or curd as well [1, 7]. Later, a conspicuous feature is the pure, snow-white, fluffy appearance of the spots, but with continued growth the spots gradually turn yellow and finally black so that eventually a series of concentric black circles is formed. Younger leaves may be attacked in turn from the outer ones especially during periods of prolonged wet weather during the season. Ordinarily, however, there is never any serious injury to the solid heart of the cabbage, or curd of the cauliflower.

The individual spots in the concentric groups are very small, being frequently less than 1 mm. across, and each spot usually develops a single acervulus (Fig. 306). The latter is laid down between the cuticle and epidermis and when from pressure of spores the cuticle is finally broken through, the spores escape to the surface in the form of a fine gelatinous tendril issuing through a tiny opening in the cuticular roof of the acervulus. Similar features accompany the formation of spores on the cauliflower on which the sporing lesions developing in abundance on midribs and petioles turn brown or black almost as soon as the spores have broken through the cuticle, so that the snow-whiteness of the spots, a typical feature during sporulation on the cabbage, appears to be of shorter duration on the cauliflower. On the larger veins and petioles of the cauliflower leaf, the lesions form narrow linear scars with upraised margins, and as they get older become sunken at the centre before being finally delimited from the surrounding tissue by the formation of cork.

Leaf spot is caused by a fungus which has been variously placed in the genera *Cylindrosporium* and *Gloeosporium*, the present designation being *Gloeosporium concentricum* [2, 3, 8, 9]. The acervulus (Fig. 306), between cuticle and epidermis, arises on a



FIG. 306.—*Gloeosporium concentricum*. Section of cauliflower leaf showing a portion of an acervulus; broken cuticle at the top, and epidermis below (× 364) (after Halsey, *Proc. Roy. Soc. Vict.*)

thin but well-defined stroma from which mycelium passing between the epidermal cells extends for variable distances into the intercellular spaces of the mesophyll of the leaf, or cortex of the petiole, as the case may be. The stroma gives rise to a close palisade of very short, branched conidiophores from the tips of which conidia are abstricted in succession. With the rupture of the overlying cuticle the acervuli become slightly erumpent and the spores ooze out in tendril fashion on to the leaf surface. The conidia are cylindrical with rounded ends, unicellular and generally biguttulate; according to different authors spore dimensions are variable; thus, from 10·4 to 13·1 by 2·5 to 2·8 μ [7]; 8·5 to 15 by 2·5 to 5·5 μ [9]; 6 to 14 by 2 to 4·5 μ [4]. The spores germinate easily in water, preferably under neutral reaction [7], to form a single terminal or sub-terminal germ-tube [9]. Growth in culture on Dox's agar, at an optimum temperature between 18° and 20° C., produced puckered, cream-coloured colonies of rather slow growth, covered with spores; at higher temperatures of 22° to 25° C., spherical black bodies resembling sclerotia, 140 to 270 μ in diameter, appeared [4]; others found such dark-coloured bodies in culture to be pycnidia containing a hymenial layer of conidiophores bearing conidia [7], while similar sclerotial bodies have also been found with no internal organisation for the production of spores [9].

Inoculation of young cauliflower plants by spraying them with a spore suspension showed the period of incubation to be about 10 days [7], the period on the cabbage varying from 13 to 15 days [4]. Penetration appears to be more readily effected at the lower than the upper surface of the leaf, and takes place direct through the cuticle, even the walls of the guard cells being penetrated and stomatal entry seems to be entirely precluded [7].

Although leaf spot may assume serious proportions in wet seasons, once the plants are in good heart there is little to fear from occasional rain. If spots appear on the outer leaves the latter should be removed and destroyed. On infected land a change of crop from brassicas is recommended for one or two seasons.

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5. Greville, R. K.: 1823. *Sc. Crypt. Fl.* i, No. 27.
6. — 1824. *Flora Edinensis*, 471.
7. Halsey, F. J.: 1934. *Proc. Roy. Soc. Vict.* xlvii, 96.
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9. Thomson, J. R.: 1936. *Ibid.* xx, 123.

Black Ring Spot of Cabbage

This virus disease was first observed in Britain in 1934, on cabbage, sprouts, and other brassicas near Cambridge, and has since been recorded chiefly from districts in the south and east of England and south Wales [3]; the first occurrence in Scotland was in 1939 [1]. The disease is widely distributed on numerous hosts, viz. stocks (*Matthiola incana* & *M. incana* var. *annua*), wallflower (on which it causes a ' breaking ' of the flowers, especially of the blood-red variety), rockcress, sweet rocket (*Hesperis matronalis*), and others. A disease known as ' black ring ' of cabbage and other crucifers, in America, is believed to be similar to ' ring spot ' in Britain, which in addition to the hosts already named attacks rhubarb, *Cheno-*

podium album, *C. murale*, spinach, *Stellaria media*, swede, turnip, watercress, honesty (*Lunaria annua*), Turkish and white burley tobacco, and *Nicotiana glutinosa* [7]. Another disease observed in Wisconsin, in 1937, called 'ring necrosis', resembles 'ring spot' in several respects, but the properties of its causal virus, its host range, and symptoms are different [2].

The symptoms of black ring spot in the field occur on full-grown cabbage and sprouts. The older leaves in particular are covered with necrotic rings uniformly distributed over the surface. The rings are almost black in colour and deeply sunken in the tissue. In smaller plants the necrosis may take the form of circular or irregular lesions. Inoculations made into healthy cabbage seedlings with sap expressed from diseased plants of *Nicotiana glutinosa* (a species highly susceptible to the virus) produced symptoms of ring spot in 20 days; as a rule, necrosis developed on the inoculated leaves, and systemic infection showed in the younger leaves as a mosaic mottle which gave a marbled effect, but there was no preliminary clearing of the veins, such as appears with another virus attacking the cabbage, namely that of cabbage mosaic. Necrotic rings developed later in the young seedlings and, occasionally, chlorotic rings as well [5]. On cauliflower and broccoli diffuse systemic mottling develops, following inoculation of the seedlings. Small, pale-green, roughly circular areas stand out in marked contrast to the normal dark-green colour of the leaf. In the field, old infected broccoli repeat the same feature of necrotic rings as was shown in affected cabbages in the field [6]. Similar observations were made on inoculated plants in California affected with 'black ring'; in this area the disease was prone to appear during cool weather [7].

Synonyms for the virus (*Brassica virus 1* Smith) of this spot disease are: *Wallflower mosaic virus* (Smith 1935); *Cabbage ring spot virus* (Smith 1935); *Cabbage black spot virus* (Tompkins 1935, 1937); *Cabbage mosaic virus* (Larson & Walker 1939, in part). The virus is sap-transmissible and is spread by *Myzus persicae* and, in California, by *Brevicoryne brassicae* which breed on the host [6, 7]. The virus is not persistent in the vectors which must make frequent visits to fresh sources to maintain infection. There is no evidence of transmission by seed.

As the disease occurs on a wide range of cruciferous plants and on various weeds, the chief means of control lies in the eradication, as far as possible, of all sources where the insect vectors hibernate. This is not an easy task since the vectors are known to multiply freely on all kinds of cruciferous debris, particularly of cabbage.

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Powdery Mildew of Cucumber and Melon, *Erysiphe cichoracearum* DC.

Powdery mildew caused by *Erysiphe cichoracearum* (Ascomycetes) is very common on plants belonging to the cucumber family. The fungus also attacks

a large number of hosts widely divergent in classification, which include potato seedlings [13, 16a], lettuce [19], cineraria [8], sunflower [16], tobacco seedlings [10], mango trees [17], and others. It occurs commonly in Britain on cucumbers and vegetable marrows grown in frames and greenhouses [11, 13], and is reported in the United States to cause considerable losses on cantaloupe melons [9].

On cucumber and vegetable marrow under excessively moist conditions the mildew begins to appear as small white areas on the leaves and stems but not on the fruit. Unless checked, the spots cover entire leaves on which the characteristic white powdery conidial fructifications of the fungus soon appear. The conidia are easily conveyed to fresh leaves by wind currents, or when the plants are splashed during careless watering. On cantaloupe melons the stem and leaves and basal parts of the crown may often be covered with these white fructifications, especially on parts overshaded by the leaves, close to the soil, and on the under side of the lowermost leaves. From such parts the mildew spreads over to the upper leaf surfaces and to the younger leaves and stems, and also to adjacent healthy plants [9]. In general, the effect of the mildew by covering stems and leaves with its mycelial web is to check growth and induce chlorosis, and the fruit suffers by reduction in size and quality.

E. cichoracearum includes numerous physiologic races which differ widely in virulence and in morphological features, especially in the size of the conidia [1]. It is interesting to note that infection experiments conducted with this fungus on a variety of hosts resistant to it showed the unusual feature of repeated attempts being made at penetration of the host. After the first penetration-process from the conidial germ-tube had been checked by the development of a papilla in the epidermal wall, a second, and sometimes a third process would arise from the germ-tube only to be stopped again at the same stage of attempted entry [3]. The fungus forms a tangled web of mycelium over the surface of stems and leaves, and rather large spherical haustoria are developed in the epidermal cells (Fig. 307 F). The superficial mycelium gives rise to a great profusion of erect conidiophores bearing conidia which vary considerably in dimensions according to their race (Fig. 307 A); thus, in one race they are from 24·4 to 35·39 by 12·55 to 20·53 μ [1], and in another from 33·8 to 63·8 by 18·8 to 31·9 μ [19]. Perithecia are of rare occurrence, due possibly to the great majority of infections being caused by strains sexually alike, the fungus being probably heterothallic [20]; thus, perithecia have been found only on certain parts of diseased leaves where probably sexually compatible strains of the fungus had established union; they have, however, been reported from localities of cold climate, and on cucumber plants in the open [14].

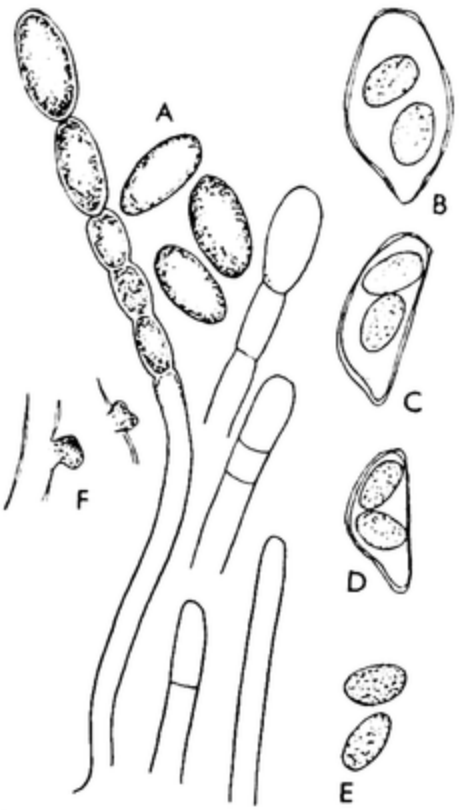


FIG. 307.—*Erysiphe cichoracearum*, causing mildew of cucumber, etc. *A*, stages in development of conidiophore and conidia. *B*, *C*, *D*, asci and ascospores. *E*, ascospores. *F*, haustoria (after Salmon, Torrey Bot. Club)

Like the conidia, the globose perithecia and their spores vary widely in dimensions, from 80 to 140 μ in diameter, containing about 10 to 15 ellipsoid, more or less stalked asci (Fig. 307 B, C, D), 58 to 90 by 30 to 50 μ, furnished with 2, rarely 3, oval or sub-cylindrical ascospores, 20 to 28 by 12 to 20 μ [15a]; from another race the dimensions were 124 to 169 μ, 64 to 86 by 34 to 49 μ, and 30 to 45 by 15 to 26 μ, for perithecia, asci, and ascospores respectively [19]. Powdery mildew of cucurbits in the Volga region of Russia showed the presence, in addition to this species, of a varietal form of the organism of the hop mildew, *Sphaerotheca humuli*, namely, the form *fuliginea* [15].

New races of *E. cichoracearum* are believed to arise by hybridisation, for cantaloupe melons hitherto resistant in the same locality were later attacked [7]. It is interesting to note that the perithecia of this fungus found on Jerusalem artichokes, in New Jersey, were infested with another fungus, *Cicinnobolus cesatii*, killing the ascocarps and then living on in the moribund tissues of the leaves of the host [4].

Owing to the rarity of perithecia, powdery mildew in frames and glasshouses probably survives from year to year in the form of conidia or mycelium on host-remains of the previous season. Moreover, the fungus has been found throughout autumn and winter on volunteer plants of various species of cucurbits, and in California susceptible host plants may be found in almost unbroken succession throughout the year [9].

As already mentioned, powdery mildew of cucurbits is encouraged in the greenhouse by excessive humidity [6], and out in the open the disease is liable to develop during wet periods after the plants have reached a certain age, the trouble increasing more and more as the foliage becomes luxuriant [2]. But even during comparatively dry periods the fungus may still be active provided heavy dews occur at night [5, 17], but long periods of dry weather definitely arrest the trouble.

Water-culture experiments, incorporating silicic acid, have shown the beneficial effect of this substance in increasing the resistance of cucumber plants to mildew, and the incubation period of the parasite is prolonged, owing probably to the deposition of silica in the epidermis [18]. When boron was supplied to sunflower plants exposed to infection they showed little or no mildew [16].

Control of humidity in the greenhouse or frame as a check to mildew is a difficult problem in view of the great needs of all cucurbits for water to encourage succulence in the fruits. These can be met, however, if by watering the soil the atmosphere in the house is not rendered too moist, and retention of water around the roots is greatly assisted by adding plenty of humus to the soil.

Good protection against mildew can be given by dusting with finely divided sulphur, and appreciable increments of yield are reported after this treatment [5, 6]. Good results also follow upon spraying with liver of sulphur, 1 oz. per 3 gallons of water or 1 per cent. Bordeaux or Burgundy mixture, either of these mixtures being applied twice — first, when the disease breaks out, and again 3 or 4 weeks later [5, 6]. Satisfactory control has also been obtained with ' shirlan paste plus agral N ', the spray being given every 3 or 4 days during the first month of attack, and at weekly intervals after ; during this programme it is advisable to remove the oldest leaves so as to help thinning out, and the plants should be sprayed until the leaves drip [12].

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Anthracnose of Cucumber, *Colletotrichum lagenarium* (Pass.) Ell. & Halst.

Anthracnose is a destructive disease of cucumbers, watermelons, cantaloups, and gourds [1, 2, 14]. It was first reported in England in 1871 to attack cucumber [3], and occurs locally on this host as well as on vegetable marrow and melon in England. The disease is common in Europe, America, and other areas [7, 8, 13].

The disease affects cucumbers in the open and under glass [4]. All parts of the host are liable to attack. In the crop the general effect is a parched or scorched appearance and spotting of the leaves, the chief damage being due to the destruction of the lamina, though petioles, stems, and fruits also suffer. The disease shows itself somewhat late in the growing season, breaking out in rather restricted areas, extending slowly at first but spreading quickly in wet weather. As a damping-off disease of cucumber seedlings, anthracnose is not common, but when affected, the young plants are attacked at soil-level and a shrinkage of the tissues brings about their collapse [5, 14]. On established plants, early spots on the leaves usually appear above a vein as pale-green water-soaked areas, soon becoming dry and of a reddish-brown colour, with a yellow zone around. The spots increase in size and join together, the browned areas cracking or falling out, and the leaf dies. On the upper surface of the leaf lesions, small pink acervuli may be found. On leaf stalks and stems the lesions are linear or oval, slightly sunken, water-soaked and yellowish, but quickly dry out and become powdery. Sometimes stem lesions are so deep, or girdle the stem so completely, that entire shoots perish. Spots on the fruit are pale-green, more or less circular, sunken areas, which sporulate abundantly to form buff or pink masses, later turning black. On ripe fruits the black lesions may show white craters on which the old acervuli appear as black dots. As the fruit lesions dry out, the tissues below may become exposed by cracking of the surface ; if such fruit lesions attain any

great depth it is not improbable that seed infection may sometimes occur in this way [9].

Anthracnose of cucurbits is caused by *Colletotrichum lagenarium* (Melanconiales) [6, 12]. In Russia, the form of the fungus which attacks cucumber, melon, and water-melon does not affect the vegetable marrow, though the form attacknig the marrow is pathogenic to other cucurbits [19]. Acervuli arise from sub-epidermal stromata. The spores are unicellular, oblong or ovate-oblong, slightly pointed at one end, 13 to 19 by 4 to 6 μ, pink in the mass ; setae are numerous, 2- to 3-septate, brown, thick-walled, 90 to 120 μ long [9]. Some have described pycnidia in addition to acervuli [17], but the former are believed to be only a stage in the development of the acervular stroma [10, 11]. When cultures of two strains of the organism growing together were exposed to ultra-violet rays, numerous perithecia of a ' Glomerella ' type formed along the line of junction [20], but these bodies have not been observed in nature. So-called ' sclerotia ' which have been observed in fruit lesions appear to be only a stage in the further development of the stromatic tissue under the lesions ; they have also been found in culture [9].

Spore germination is strongly aerobic ; the minimum temperature is 4° C. and the optimum between 22° and 27° C. Conidia have been observed, in water at 14° to 18° C., to produce chlamydospores which developed a mycelium ; the latter is at first colourless and thin-walled, septated, but later becomes thick-walled, dark brown, the cells again appearing like chlamydospores [9]. It is not clear how the fungus survives from season to season. It is suggested that one method may be by chlamydospores [15] or through sclerotia developed on host debris [18]. The fungus is known to survive saprophytically on rotten wood in the glasshouse and in straw manure from beds in infected houses, and infection may thus spread to fresh crops. There is no substantial evidence that the disease is carried by seed, though it may sometimes occur if infected fruit-pulp still remains adherent to fresh seed, otherwise it is difficult to account for the introduction of the disease into new areas [9]. It appears to be the custom, therefore, for growers to use older seed in preference to new. In general, early infections are seen to arise on the under side of the leaves, due probably to spores being splashed by rain from infected debris on the ground, or carried through the soil in drainage water.

With constant high humidities of 87 to 95 per cent. the disease is produced over a wide range of temperature, but most severely at 24° C. By keeping the temperature unusually low or high the trouble is materially checked but the effect of these extreme conditions on the host is injurious [14]. In Hungary, on the other hand, the disease was favoured by comparatively low temperatures at 12° to 14° C., the conidia remaining viable after exposure to 0° C. during the winter [11].

Since the fungus grows saprophytically on various materials such as wood and straw, efficient cleansing of glasshouses with disinfectant is essential [16]. For this purpose an emulsion of cresylic acid with pure, potash soft soap, 1 gallon of acid to 8 lb. of soap, heated until dissolved and used at a strength of 1 in 50 of water, on the woodwork, is recommended [5]. At the first signs of disease the plants should be sprayed with potassium sulphide, using flour paste for better adhesion, the proportions being 5 lb. flour, 4 lb. sulphide, per 100 gallons of water, a second application being given a week later, preferably in the evening,

and the surplus spray should be removed next morning with water [5]. Excessive humidity and sharp fluctuations of temperature in the glasshouse should be avoided, the maintenance of a constant day-and-night temperature being most important [5]. When seed contamination is suspected, disinfection may be ensured by steeping for 5 minutes in a solution of 1 in 1,000 mercuric chloride [9, 21].

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Blotch Disease of Cucumber and Melon, *Cercospora melonis* Cooke

This disease was first discovered in England in 1896 on leaves of melon. It was seen again, in 1904, on leaves of cucumber in Hayes, Middlesex, and in this instance loss of fruit was reported to be very heavy [5]. Since the discovery of the resistant variety, Butcher's Disease Register [4], 'blotch' has practically disappeared from cucumber culture, but sporadic outbreaks are still reported to occur on other varieties in various parts of England and Wales.

The symptoms on the leaves of cucumber consist of numerous irregular spots of various sizes on the upper surface, pale green at first, turning yellow and then brown, and the discoloration spreads so quickly, the leaves becoming dry and crumbling, that entire leaves perish within twenty four hours of infection [1]. The fruit remains small, or fails to develop.

The fungus causing leaf blotch, *Cercospora melonis* [2], is a member of the Hyphomycetes (Fungi Imperfecti). The mycelium from the interior of the leaves breaks out, spreading on the surface to form a dark, olivaceous mycelium which gives rise to erect, slender conidiophores. The hyaline to brown, inversely club-shaped and tapering conidia, 80 by 8 μ in a widest diameter, are borne singly or in a row of two or three on the conidiophore, and are from 1 to 12 or 15-septate [2, 5]. In agar cultures the abundant mycelium is at first colourless, then changes to a greenish brown and sporulates so profusely as to develop a velvety pad covering the substratum. Conidia germinate within a few hours at 20° to 22° C., the germ-tubes being developed from the two end cells of the spore [5].

When infected leaves fall to the ground the contained mycelium may grow out on to the surface of the soil and may even form conidia — somewhat smaller than those on the leaves — which, if dispersed by wind or splashing water bring about fresh infections. If the soil remains moist the mycelium continues to extend and produce more conidia as long as moist conditions and high temperatures last. Thereafter this soil mycelium passes over into a resting condition and is said to survive from one season to the next. The disease is only epidemic under glass when moisture is excessive and temperatures are high. Under these conditions when the foliage is rendered soft, especially if the plants are in shade, infections are easily established [1]. In the open the plants are rarely infected, and lightly affected ones taken out of the greenhouse sometimes recover.

This disease is difficult to eradicate from glasshouses and frames, and drastic cleansing with disinfectant and thorough removal of all soil which has become contaminated with mycelium and infected plant debris, is essential before another planting of cucumber in fresh soil is made. Fortunately, the variety Butcher's Disease Register is highly resistant, and though it may sometimes show a few disease spots on the cotyledons, these usually develop no further [4]. Owing to the survival of the fungus on leaves left on the soil over winter, all leaf debris should be collected and burned. Little benefit is reported from spraying the plants to check the disease, but with adequate ventilation good results were obtained using potassium sulphide, 2 oz. in 3 gallons of water and adding 2 oz. of soft soap, the spray being directed especially towards the under side of the leaves [1].

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Bacterial or Marginal Spot of Lettuce, *Pseudomonas marginalis* (N. A. Brown) Stapp

This disease first appeared in England during the summer of 1921 in a garden where 75 per cent. of the lettuce plants were attacked [8] and is now fairly common throughout Britain. Several bacterial diseases of lettuce are known in different parts of the United States and the one called ' Kansas disease ', on greenhouse lettuce, is probably the same as ' marginal spot ' [3].

The disease is caused by the bacterial organism *Pseudomonas marginalis* [4a, 5, 9], the specific name being descriptive of the characteristic symptom of a brown discoloration along the margins of the leaves. Marginal withering of lettuce leaves may also be caused by *Botrytis cinerea* (see ' grey mould ', p. 657), and may sometimes be due to deficiency troubles particularly of potash in short supply [7a, 10].

Early symptoms appear when the lettuce plants are about half grown. On leaves in the same whorl, of much the same age, the marginal discoloration appears at various points, and may vary from mere specks to areas 2 to 3 cm. long, which by coalescence may extend into strips up to 7 cm. long. The oldest leaves on

the outside may show a wilting only at the tips, but these outer leaves are frequently entirely unaffected. From the browned margin the discoloration works backwards until the veins of the leaf appear as a fine brown network [8]. Later the entire leaf droops, the midrib turns a rusty brown colour, and finally becomes soft and pulpy [7]. The infection does not extend down the entire leaf, and is not of the nature of a rot or soft decay, but the discoloration mars the appearance of the leaves, making the plants unacceptable for market [3].

The organism of bacterial spot of lettuce has been variously named by different authors. It appears to be synonymous with *B. marginale* [4], *B. pyocyaneus* [7], and closely related to *B. aptatum* [6]. It is described as " motile, with 1 or 2 polar flagella; rod-shaped; occurring singly, or in pairs, or in short chains ; forms capsules but not spores ; measures from 1·5 to 3·0 by 0·75 to 1·65 μ ; gram-negative ; not acid-fast ; facultative anaerobic ; green fluorescent ; liquefies gelatine ; nitrates not reduced ; produces ammonia ; colonies on beef-extract-agar (*p*H 6·9) in 8 days, white to creamy, round, or with slightly irregular edge, smooth " [4]. Other bacteria, *P. viridilivida*, and *B. phytophthorum* (p. 483), have also been isolated from lettuce affected with marginal disease of the foliage [7 a].

The organism is soil borne, and is highly resistant to extremes of temperature ; it survived in a refrigerator for 9 to 10 months and retained its virulence for more than a year [3]. On a peptone-broth medium + 15, the temperatures for growth were 38° maximum, 25-26° optimum, with a minimum below 0° C. [3].

Infection takes place through the stomata of the leaves, or at points where drops of water collect at the leaf margin. The disease is frequently associated with heavily manured autumn crops watered overhead [1].

In well-ventilated greenhouses ' marginal spot ' is hardly known, but is greatly fostered by high humidities. Its spread in the greenhouse is effected chiefly through careless watering, for since the organism is a soil inhabitant, overhead watering causes soil to be spattered on to the leaves and infection of the wetted leaf is therefore ensured. Watering should be directed to the roots or done by sub-irrigation.

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Downy Mildew of Lettuce, *Bremia lactucae* Regel

Downy mildew of lettuce is a disease mostly of young seedlings under glass, but plants of all ages may be attacked, though less severely in the open.

The disease has been known in Europe since 1843 [9], in America, about



FIG. 308.—Downy mildew of lettuce (*Bremia lactucae*) (photo by Ogilvie, by permission of Long Ashton Res. Station)

1875 [4], and is now of common occurrence wherever lettuce is cultivated. It is of more importance on winter and early spring crops than on those grown during the warmer months of the year. Appreciable losses from downy mildew are experienced in storage and during transit overseas, and the disease is hardly checked even at the low temperature of the refrigerator. Still greater losses may occur if the mildew is succeeded, as so often happens, by the *Botrytis*, 'grey mould' disease.

Seedlings in the frame are attacked very soon after germination, but seed transmission is considered negligible, each crop becoming infected from a previous one [14]. Its spread during the winter is mostly in patches, but sometimes it may sweep through an entire stand of young plants. Seedlings attacked soon after emergence may be killed off, and older ones, from loss of the early leaves through disease, remain stunted and make but poor growth. The check to growth is not so evident on plants which are more or less well established, but this does not decrease their liability to infection.

When older plants are attacked, early symptoms of downy mildew may be seen on the lowermost leaves, especially on those shaded over and in contact with the soil. These early signs consist of light-green or yellow spots, $\frac{1}{2}$ to $\frac{3}{4}$ inch in diameter, scattered on the upper surface of the leaves, and which later become covered on the corresponding areas on the under surface with patches of the snowy white mycelium and sporangia of the mildew (Fig. 308). Individual spots may spread, join together, and later turn brown in colour.

Downy mildew of lettuce is caused by the fungus *Bremia lactucae* (Peronosporales). The asexual fructifications are developed in great profusion on affected foliage, but the oospore stage occurs rarely [8]. The sporangiophores emerge through the stomata in groups of two or three, and have an average length of 190 μ, before they branch out in dichotomous or trichotomous fashion near the top, to form numerous short branches which finally terminate in flattened expansions, like the upturned palm of the hand, the digits representing so many fine sterigmata each bearing an oval-shaped spore (Fig. 309 A.) The spores are hyaline, papillate, and measure on an average 18·5 by 17·5 μ; they germinate mostly direct by germ-tube, but also indirectly by formation of zoospores (Fig. 309 E, F). It is reported that spores which were developed at the lower temperatures from December

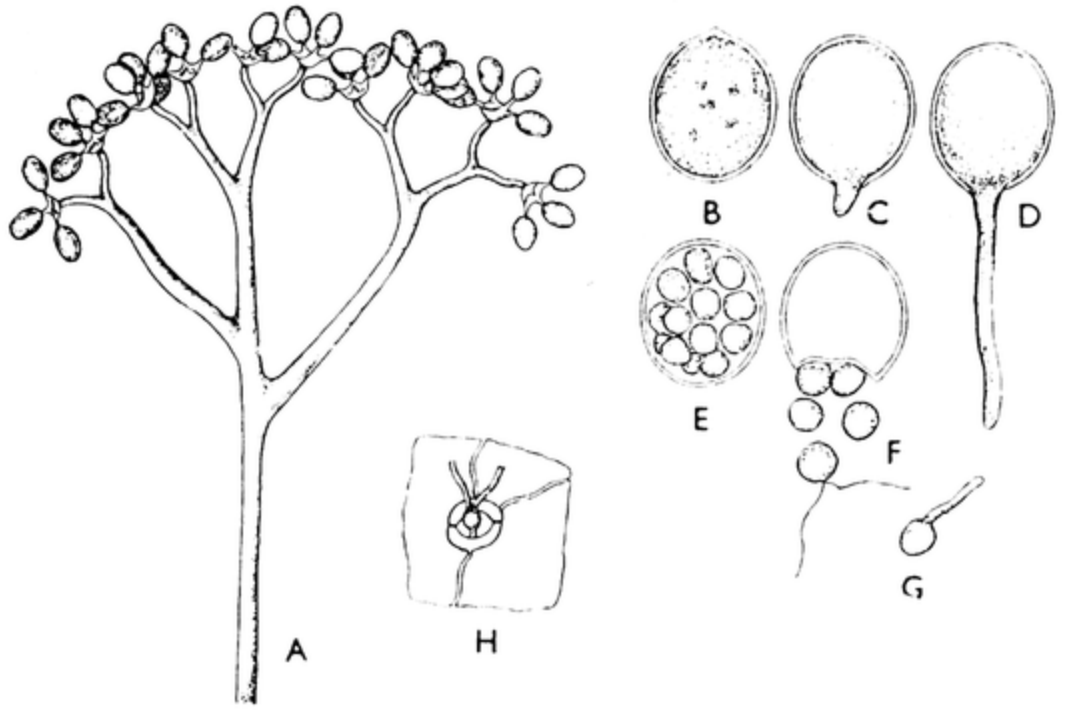


FIG. 309.—*Bremia lactucae*. *A*, sporangiophore and sporangia. *B*, *C*, *D*, sporangium showing direct germination. *E*, *F*, indirect germination of the sporangium. *G*, germinating zoospore. *H*, sporangiophore emerging through a stoma (after Milbrath, *J. Agric. Res.*)

to March formed zoospores more readily than those developed during the warmer parts of the year. Subdued light or darkness, and a temperature of 10° C. are most favourable to the development of zoospores, and it is probable that infections which take place at comparatively low temperatures are effected by zoospores, and those at somewhat higher temperatures by direct formation of germ-tubes from the spores themselves [9]. The latter are fairly resistant to dryness and are apparently able to survive in the soil of the greenhouse from one season to the next ; whether they actually survive in the soil itself or on remains of infected material is not clear, but they are reported not to survive frost [8] ; others state that their viability is lost in 6 or 7 days [14]. The light-brown, spherical oospores, 26–35 μ in diameter, have not, so far, been found in the host [8].

Bremia lactucae attacks also various wild species of the lettuce kind, a few ornamentals [9a], and also a number of weeds, and the frequent proximity of these to greenhouse or frame has suggested that they may serve as carrier hosts. The fungus exists in a number of strains or physiologic races, some of which are believed to be specialised on their respective hosts ; at least two are known in England, one of them showing variable degrees of pathogenicity on particular varieties of lettuce, but all varieties are susceptible to the other strain [5, 6, 10a, 10b, 12]. The form (or forms) of the fungus affecting lettuce in England is probably a different race from that attacking the plant in the United States [5]. But an American strain found on *Lactuca scariola* var. *integrata* is apparently similar to the second English strain [10b].

Infection takes place through the stomata and is favoured by temperatures, from 15° to 17° C. The mycelium is intercellular and is furnished with club-shaped haustoria. Sporangiophores and spores appear within 8 to 9 days following infection.

Downy mildew of lettuce is favoured by comparatively low temperatures and high relative atmospheric humidities [11]. Few, if any, fresh infections occur in the open during the summer and during spells of dry weather. The rapidity with which fresh attacks come on immediately after rain indicates that infections may already have been established at an earlier stage but held in abeyance until the arrival of more humid conditions [10, 12].

Lettuce varieties differ considerably in their resistance to downy mildew [1, 7]. Strain *1* of *B. lactucae* isolated mainly from outdoor lettuce attacks: Arctic King, Attractie, Blatchford's Giant, White Cos, Borough Wonder, Cheshunt Early Giant, Feltham King, Forcing Mildew Resistant, Webb's Wonderful, and Celtuce, while Finney's '27' and Grand Rapids are resistant. Strain *2*, found mainly on indoor lettuce, attacks all cultivated varieties tested [10b].

Though the lettuce plant is essentially moisture-loving excessive moisture should be avoided in the greenhouse during early growth at low temperatures if seedlings are to grow healthily. Spraying the seedlings with Bordeaux mixture 4 : 4 : 50 is recommended two or three times before transplanting [1, 3, 8]. Overhead watering, especially on dull days, should be avoided, and there is less risk from disease if the plants are sub-irrigated, the surplus water being allowed to drain away [3]. Treatment of the seed-bed with formalin [2] or 'folosan' dust gives some measure of control over the mildew [13].

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Ring Spot of Lettuce, *Marssonina panattoniana* (Berl.) Magn.

'Ring spot' disease of lettuce is of importance in Britain only on winter lettuce (grown from seed sown in late summer), on plants set out in the open to head in the late spring. There is little danger of the trouble occurring on spring lettuce, and it is rare in the frames, though the first record in England was on lettuce under glass [5]. Summer lettuce is also fairly safe from attack under dry, warm conditions, but may suffer if allowed to grow on to the autumn [14]. In other countries the disease is reported to do great damage to greenhouse lettuce [4], and in New York State where it occurs on early spring and late outdoor plantings it is prevalent too, in the greenhouse [7].

This disease was first discovered in Italy in 1894; the first record in Britain

occurred in 1912, but not on outdoor crops until 1922, at Swanley, Kent, where growers reported that whole areas of several acres were " so badly attacked that we shall have to plough them in " [12]. In America the disease was first reported in 1895, in Ohio [13], and in Victoria, Australia, in 1919, probably from importation of contaminated seed [3].



FIG. 310.—Ring spot of lettuce (*Marssonina panattoniana*) (photo by Foister & Noble)

Ring spot attacks lettuce under cool, damp conditions, at all stages of growth, from young seedlings to mature plants. The former are rapidly browned and destroyed and soon become converted into a soft, slimy mass. In the beds, the affected plants are yellowed or browned, the discoloration progressing from the outer to the inner leaves until entire heads are often destroyed. On the expanded leaves early infections begin as small, water-soaked areas which vary from mere points to spots 3 to 4 mm. across, circular, or angular in shape if they are close to the larger veins. As they enlarge, the spots turn yellow and then brown, the tissue gradually dying off from the centre towards the margin (Fig. 310). A characteristic feature of the disease, giving it the name of ' ring spot ', is the ultimate dropping out of the central part of a spot, leaving a shot-hole, so that a perforated lamina helps, at a later stage, to distinguish this disease from two other maladies of the lettuce, which otherwise it closely resembles, viz. ' marginal spot ' (p. 650), and ' grey mould ' (p. 657), but the bacterial leaf spots are more yellow brown than this, and those of the *Botrytis* grey-mould dry out and turn black [14]. The brown spots on the lamina may extend to about 2 mm. in diameter before they develop a white or pinkish-white layer of spores, or the centre may drop out, leaving the hole with a white or pale-pink fringe of spores, but most commonly the edges of the holes are brown [14]. The symptoms on the stem, and on the larger veins, the midrib in particular, as seen from the under side of the leaf, are somewhat different, consisting of a series of sunken lesions, rather elongated in the direction of stem or vein, ranging from 4 to 5 by 2 to 2·5 mm. in diameter, and are straw yellow, or reddish yellow as they get older, a feature which prompted some of the earlier observers to call this a rust disease of lettuce. Sometimes the lesions on the leaf petioles may be so deep as to cause the lamina to bend over [14]. Rusty-brown spots are also reported to occur on the inflorescences and fruit clusters [11].

Ring spot is caused by *Marssonina panattoniana*, a member of the Fungi Imperfecti, of the group Melanconiales [1, 2, 6]. The spores (Fig. 311) breaking through the epidermis

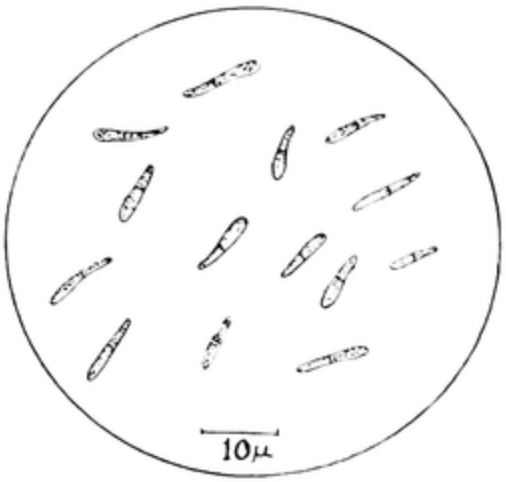


FIG. 311.—*Marssonina panattoniana*. The spores

of the pustule (acervulus) are borne on short conidiophores, are colourless singly, but white or pale pink in the mass, club-shaped, slightly curved, uniseptate, 12 to 17 by 3 to 4 μ. They germinate at temperatures up to 25° C., producing in culture a slender, septated mycelium, with scant sporulation [4, 10, 15].

There is no clear indication of the manner in which this disease is carried over from one season to the next. The chief source of infectivity is believed to be from diseased plant debris being allowed to remain on land in which the crop is planted over and over again, though such infected material is said to be innocuous after eight months or less [7, 14] but may possibly survive longer under certain circumstances. There is also the probability of its longer survival in the soil, and it is certainly carried to clean land with manure containing lettuce trash [12]. The organism also attacks certain weeds, near relatives of the lettuce, notably *Lactuca scariola* (believed to be the progenitor of the cultivated plant), *L. spicata*, and *L. canadensis*; and several other species of the genus yield to infection in the seedling stage [7]. There is little doubt that the disease may be seed borne [14] but others have failed to establish this [11].

Whatever be the means of primary infection, whether from contaminated seed or from a renewal of sporing on infected leaf debris in the soil, the first signs of disease consist of small spots on the under side of some of the outer leaves, such as would be caused by the splashing of spores from the soil. It is recorded that, from the sowing of contaminated seed, a latent period, as long as two months, is passed through before the appearance of symptoms, and it is suggested that spores carried up in germination, in contact with the cotyledons, remain ungerminated, but viable, for a considerable time [14]. Infection having been established in the outer leaves, the fungus may proceed direct from leaf to leaf in contact, so that an entire plant may thus become infected. Even when direct penetration may not go as far inwards as to reach the heart leaves, secondary infections brought about by spores being splashed from the lesions on the older, outer leaves may form fresh lesions on the upper parts of the inner leaves, or on any of the leaves of neighbouring healthy plants. In wet weather, spores produced in immense numbers in these leaf lesions are washed down to the crown of the plant, and so the leaf stalks become infected, with the result that there is considerable withering of older leaves and fresh infections of younger ones [12]. The disease is most prevalent in damp or rainy weather, and is favoured by rather low temperatures [15].

There is little doubt that the disease is aggravated by growing lettuce in the same ground from year to year. Remains of diseased plants should not, therefore, be allowed to remain on the soil, or be dug in, or conveyed to the manure heap, but collected and destroyed by burning. Rotations of three years are recommended [3]. If seed treatment is decided upon, this should be done with a clear

solution of bleaching powder (1 part in 10 of water, by weight, allowed to stand for ½ hour, and then strained), steeping being done for 4 to 6 hours, keeping the temperature below 25° C., and sowing as soon as possible [14]. Spraying the soil, after sowing, with a 1 per cent. solution of lithium nitrate is reported to reduce the amount of disease [15]. Spraying of seedlings with a 3 : 6 : 50 Bordeaux mixture is also recommended [11], but plants dipped in the mixture previous to planting out were checked and no apparent control was obtained [9].

It appears that no variety of lettuce is immune from ring spot disease, but there are various grades of resistance to it. The varieties Trocadero, Lobjoits' Green, Hardy Winter White, are all susceptible [12], while May King, Syston Glory, and Standwell show least infection [8].

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Grey Mould of Lettuce, *Botrytis cinerea* Fr.

'Grey mould' attacks lettuce practically all the year round and is most destructive on the plants in the open. Early planting out from cold frames, even as early as January and February, naturally appeals to the commercial grower, and such fostering of growth to induce early maturity is greatly encouraging to the disease. Under crowded conditions and enclosed humidity existing in the cold frame during winter when ventilation is attended with risk from frost, when daylight is poor and fogs are common, the plants are tall and spindly and highly subject to grey mould disease [5]. Under these conditions mortality of seedlings is very high and up to 75 per cent. of the crop may be lost within a few weeks of being set out in the open [2, 3, 6].

This disease attacks young plants at ground-level and is a typical 'collar rot', causing a wilting and collapse of entire plants. Under normal conditions, in a dry or moderately moist environment, the young plants cast off their cotyledons and sometimes their lower leaves as well, as soon as their permanent leaves have started to develop. The scars of the rudimentary leaves meanwhile become healed up in the normal way, thus rendering the seedlings tolerably safe from attack by micro-organisms of the soil. Under conditions of excessive humidity, however, in crowded growth in the frame, the young plants either fail to throw off the cotyledons and lower leaves, in which case these organs quickly decay, or if



FIG. 312.—Grey mould of lettuce (*Botrytis cinerea*) (photo by Ogilvie, by permission of Long Ashton Res. Station)

they are discarded, their scars on the stems fail to heal over owing to excessive moisture. The seedlings are, in consequence, open to immediate attack through these channels by various organisms, the most common of these being *Botrytis cinerea*, a fungus which is responsible for the grey, mouldy condition of a wide range of plants, including flowers and fruits as well as vegetable matter, under humid conditions. This fungus is especially favoured when first presented with moribund tissue such as, here, the decayed cotyledons and lowermost leaves of the seedlings, or failing these the exposed tissues of wounds or open scars. Stimulated by this pabulum, its virulence is increased and the fungus is enabled to penetrate further through the diseased tissue, or open scar, into the stem. When seedlings are crowded together in the frame, these incipient stem or collar infections are very difficult to detect, and there is little doubt that a very high percentage of seedlings unsuspected of harbouring the disease are planted out in the open.

About a week after transplanting, older leaves of affected plants collapse and become dull and yellow (Fig. 312). Such leaves show at the base a characteristic reddish-brown discoloration which may extend not only to the leaf margin but round and up the stem as well, the tissues of which become more and more involved. Disintegration of the stem at the collar may set in so rapidly that the green shoot above falls over before there is any wilting of the younger leaves [1]. Over the surface of the browned diseased portions, the whitish-grey conidial fructifications of *B. cinerea* usually appear in great abundance (Fig. 77 D).

On plants set out in the spring, about April, and examined some two months later when well established, there is considerably less disease than on the earlier plants and it is confined mainly to the outer leaves at their basal parts in contact with the soil. In these parts, dark, almost black lesions may extend about half-way around the stem and a number of reddish-brown sclerotia may be found embedded in these lesions. These hard bodies, often as many as six in a lesion, are club-shaped with the narrower end projecting downwards, and may be accompanied by a sparse growth of conidia covering the surface of the lesion. On the decay of the host the sclerotia are liberated into the soil where they are capable of survival over long periods and serve as a means for the renewal of the disease.

Lettuce tissue affected with grey mould almost invariably yields, in artificial culture, not only one or more strains of *B. cinerea*, but also one or more other fungi, mainly those of the damping-off kind such as *Pythium* and *Rhizoctonia* (pp. 578, 660), together with *Bremia lactucae* (p. 651) the organism causing downy mildew of lettuce. It is pointed out [9] that these organisms often contribute to the general effect of grey mould, if indeed,

they may not actually be the precursors to the more serious *Botrytis*. Grey mould appears, therefore, to be a congeries of infection in which, however, the *Botrytis* organism early becomes predominant though not always the first to attack. Two fairly distinctive strains, or groups of strains, of *B. cinerea* are recorded to be capable of causing grey mould of lettuce [1]. They are called ' A ' and ' B '. The ' A ' group has a much greater capacity for forming sclerotia than the ' B ' group, but ' B ' produces conidia in much greater abundance than ' A '. The following are the dimensions of the conidia : ' A '—ovate-oblong, 6·0 to 15·0 by 5·0 to 9·2 μ (average, 11·1 by 7·25 μ) ; ' B '—round, 5·0 to 13·2 by 5·0 to 10·0 μ (average, 9·7 by 8·0 μ). They are borne on branched conidiophores forming dense, more or less apical tufts, grey or turning brown, each conidium being carried at the end of a minute sterigma. The sclerotia, of variable size, are club-shaped, dark-brown or black, about 6 mm. long, with a rough warty surface [1].

The fungus hibernates on the surface of the soil in the form of sclerotia, but may also quite possibly survive under the protection afforded in the frame, in the form of mycelium or conidia kept alive on lettuce debris from the previous season. When the sclerotia germinate they produce a coating of mycelium which soon gives rise to conidiophores and conidia.

Infection of seedlings is believed to take place very early, soon after the seed-coats are carried up out of the soil during germination. Conidia lurking on the surface of the soil may possibly be picked up by the emerging seed coats, and are therefore close at hand for infection. The chief way of attack is through the open scars on the young stem, left after the fall of the cotyledons or first leaves, at the time when the plants are crowded together in a humid environment. As long as the seedlings are capable of retaining their cotyledons and first leaves intact long enough to carry them well above the soil, and become green, or if after the natural fall of these early leaves their scars are allowed to heal, the seedlings are tolerably safe from attack provided the young tender leaves do not make contact with diseased seedlings. Older leaves may become infected if, by drooping over, the tips or margins come to touch contaminated soil. In some cases, under crowded growth, there appears to be direct infection of the stem base from the soil, but this is due probably to actual contact of the tender stem with infected trash. Other leaf injuries, such as those caused by slugs, frost or sun scorch, may also invite infection.

Notwithstanding the recognised fact that *B. cinerea* is a parasite of weak propensities, it kills the host tissues in advance of the actual extent of invasion. From a point of inoculation, say on the stem, discoloration extends for a considerable distance above the point of penetration, and this may be present in the leaves and wilting take place without their occupation by the fungus at all. This shows that grey mould is a true vascular wilt which results from the toxic action of fungal secretions being carried up along the vascular system of the plant in advance of the limits of fungal invasion [7]. The same lethal effect is produced when moist, infected debris comes into contact with healthy plants, as in the case of direct stem infection cited above.

The factors of the environment most favourable to the incidence of grey mould are a high degree of humidity and a low temperature. Under the comparatively drier and warmer conditions existing over winter in the frame the

trouble is not serious, but when the plants are transferred to the open, the drop in temperature, the wetter and colder conditions in the soil, danger from frost and sudden thaw, all contribute to the progress of the disease, and plants but lightly attacked in the frame soon develop more serious symptoms of browning and decay in the open [2].

There are various kinds of lettuce which are comparatively resistant to this disease. The lax open type, such as the variety Trocadero, is more prone to the disease than the compact cabbage type. The varieties Lee's Immense, Hick's Hardy White, Bath's Black-seeded, Arctic King, Imperial, Winter White, and varieties of the Gotte group, are all satisfactory winter varieties, while Continuity and Trocadero are very susceptible. Early spring varieties recommended are May Queen, May King, and Feltham King [1, 4, 4a].

For control of the disease in the frame some growers use a powder fungicide ('folosan') for light dusting, but the practice tends to check growth and the plants thereafter are more sensitive to frost. The powder may be worked into the soil to a depth of 2 in. at the rate of 1 oz. per square yard, or it may be sprinkled on the surface after sowing [5]. If the mould appears when the seedlings are very young they should be dusted very lightly, but older ones can stand dusting at intervals of 3 to 4 weeks, preferably when they are slightly moist, a final dusting being given before the plants are lifted. No dustings should be done during periods of severe frost. Soil treatment may be carried out about 10 days before planting, with a 2 per cent. solution of formalin, the surface of the soil being well saturated at the rate of 1 gallon per square yard [7].

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Damping-Off or Bottom Rot of Lettuce, *Corticium* (*Rhizoctonia*) *solani* (Prill. & Delacr.) Bourd. & Galz.

'Damping-off' and 'grey mould' diseases of lettuce have much in common and, as above indicated (p. 658), the fungus *Corticium* (*Rhizoctonia*) *solani*, which is the cause of damping-off, is also associated with *Botrytis cinerea* in the symptoms presented by grey mould disease. *Pythium* sp. may also be implicated [2a].

Damping-off is actually only the first phase of the disease affecting young seedlings in boxes or frames. Mature plants may also be attacked and the name 'bottom rot' applied to the disease in America is descriptive of the later stage when fully grown plants are destroyed at the base. In the United States it is held to be the most important disease of lettuce and has been extensively studied there, and losses of about 30 per cent. of the crop are quite common in New York State every year [1, 2, 4, 5, 6, 7].



FIG. 313.—Damping-off (*Rhizoctonia solani*) of lettuce on seedlings, on right (photo by Ogilvie, by permission of Long Ashton Res. Station)

In the seed beds damping-off breaks out sporadically and entire patches of young plants may be ruined (Fig. 313). Later, they may fall prey to *Botrytis* and become covered with the characteristic grey fructifications of that organism. Under conditions of high humidity the seedlings are attacked at or a little below soil-level, the stem becoming brown and constricted and young seedlings so affected fall over and die. The symptoms of typical bottom rot are seen when the plants are mature. First signs appear on leaves in contact with the ground, in the form of small, rusty to chocolate-brown spots on the under side of the midrib. Infection extends along the midrib to the blade, and entire leaves may be destroyed, but the stem is more resistant. The outer leaves are the first to wilt and brown and the entire head eventually turns brown and slimy, but later dries out becoming almost black and mummified. In this condition the heads fall off owing to the complete disintegration of the stem at the base.

Rhizoctonia solani, already described in this book in connection with ' black scurf ' of potato (p. 524) and ' damping-off ' of sugar beet (p. 583), is a common inhabitant of the soil, and there are several races of the parasite. The race attacking lettuce has bigger sclerotia than the one infecting the potato. Sclerotia may be found embedded at the base of the petioles, and are more or less covered with a web of mycelium which is almost devoid of colour. On lettuce again, as on potato and sugar beet, the existence of *Corticium*, the perfect stage of the fungus, is rare. The sclerotia, on decay of lettuce heads, are liberated into the soil, and survive the winter if not too deeply buried. There is evidence, too, that the mycelium is capable of over-wintering in the soil [7].

New infections may arise from the germination of sclerotia, and perhaps also from the rejuvenescence of resting mycelium, in the soil. Sclerotia placed on the soil under mature plants in the greenhouse produced mycelium in twenty-four hours, and within a similar period lesions appeared on the petioles. The fungus may penetrate uninjured leaves through the cuticle, not merely through stomata, to develop an intercellular mycelium in both epidermal and mesophyll tissues. It further spreads over the surface of the leaf in many directions, forming appressoria-like clusters of hyphae from which new branches are developed for further penetrations. The mycelium develops mostly in the epidermis, but may also be found in the phloem and even in the xylem vessels.

This disease is favoured by warm, moist weather. Infections in the greenhouse, at temperatures of 24° to 32° C., were more serious than at lower tempera-

tures of 16° to 24° C. [3]; the sclerotia do not germinate, and the disease is checked, at low temperatures. In the case of attacks on mature plants, air temperature is more important than soil temperature, presumably because the attacks are made above ground. The reverse is the case during the early, damping-off phase of the disease, for the attacks are then directed at the parts of the seedling just below or at soil-level, and are favoured by comparatively low temperatures. Infections demand high degrees of atmospheric humidity and soil moisture. The trouble is aggravated in a heavily manured soil of high water-holding capacity, but the reaction of the soil appears to have little influence on it [7].

As so many crops are liable to attack from *R. solani*, the usual rotations are attended with risk. Cultivation of lettuce in ridges helps to ward off the disease, thus ensuring better ventilation and soil drainage than when grown on flat ground. Good results are reported following treatment with the dust product ' folosan ', on the lines indicated for control of grey mould. A preparation known as ' DuBay 738 ' (ethyl mercury phosphate) is also recommended for control of the ' bottom rot ' condition [6]. It is applied before the disease is liable to appear, which is usually two weeks before the crop is mature, and is better done when the plants and soil are dry. The dust must be blown well under the plants and against the stem, covering the surface of the soil under each plant with a fine dusting of the powder. The chemical is poisonous, and protection of the face during operations is advised ; no harmful effects follow from eating the treated lettuce, but it is recommended that no dusting should be made 12 days before harvesting the crop [6].

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Lettuce Mosaic

This virus disease of lettuce, frequently called ' rust ' by the growers, was first described in Florida [4], and appears to have been recorded in England in 1931 [6]. It occurs on both ' cos ' and ' cabbage ' varieties of lettuce, doing considerable damage, chiefly to winter plants ; it is not commonly found on greenhouse lettuce [2, 4]. Weeds such as common groundsel and prickly sow-thistle have been found to harbour the virus, and both sweet peas and garden peas are susceptible to the virus of lettuce mosaic [9, 10].

On fully grown varieties of cabbage lettuce in the field the disease has a dwarfing effect, the plants are imperfectly hearted, and show a mottling or yellowing with a distortion and scorching of the leaves (Fig. 314). A clearing of the veins is a common feature in both young and old plants, this being usually the first sign of the trouble in varieties of cos lettuce. The discoloration on the leaves may

consist of an irregular pale blotching, or the leaf may turn yellow all over and become hard as if frosted. In some cases the leaf may present a blistered or ballooned appearance, and by developing an increased roughness around the margins the leaves look abnormally serrated. Brown necrotic spots which later develop on the leaves may be found either between or along the veins, and the leaf margins may also present a browned, scorched appearance. Vein necrosis does not commonly occur in cos lettuce, though these plants too develop a marginal scorching of the leaf blades. Affected plants allowed to bolt are generally to be seen with somewhat shorter stems than is usual with bolted plants, and the leaves on these stems again show a pronounced mottling with sometimes necrosis, and in certain varieties of lettuce necrotic lesions may develop on the stem, bracts, and flowering buds, the latter frequently failing to open, and the yield of seed is often reduced. The above are the general symptoms of lettuce mosaic as they present themselves in the field, and differences due to various factors, such as varietal reaction, environmental conditions, the presence of other diseases on the host, unfavourable soil conditions, especially on outdoor winter lettuce, emphasise the importance of making inoculation tests for a correct diagnosis of the trouble [1]. On winter varieties in the spring, mosaic is usually favoured by dry weather, a condition which seems to encourage the increase of aphides during the previous October and November when the plants were at the seedling stage [1].



FIG. 314.—Lettuce mosaic. *A*, healthy plant. *B*, diseased. *C*, *D*, leaves affected with the mosaic (photos by McKay)

Lettuce mosaic virus (*Lactuca virus 1* Jagger) may be transmitted by seed and has been found in sowings of commercial seed at least three years old. It is spread by insects, *Myzus persicae* being the most important, but in certain parts of England *Macrosiphum gei* is the commonest vector, and *M. sonchi* is also said to be implicated [1, 3, 5, 6, 8]. Plants inoculated in the glasshouse, either by mechanical means or by insects, develop the first symptoms in a week or a fortnight [1]. During the germination of affected seed, the cotyledons show no signs of disease but the seedlings are stunted and develop a mottling of the second or third and of the subsequent foliage leaves. The disease does not usually develop on seedlings which appear to be healthy at the fourth to the fifth leafing stage. Initial infections thus appear to come from infected seed, while aphides are responsible for the spread of the trouble [1, 2].

As already mentioned, marked varietal resistance to mosaic exists in the host plants. Observations at Long Ashton showed the varieties All the Year Round, Feltham King, May Queen, MacHattie's Giant, and the Arctic types to be severely affected; Exceller and Spring Beauty were little less affected; Trocadero and Lee's Immense did not react uniformly, while Stanstead Park and its related varieties Early Spring and Tremont Winter were highly resistant [8, 8a].

For the control of lettuce mosaic only virus-free seed should be sown. Young seedlings should be examined carefully for symptoms before transplanting, and in the field affected plants should be rogued out as soon as detected. Later plantings of winter lettuces, when aphides are scarce, are less likely to be severely affected than earlier plantings [6]. Infection in the field, spread by aphides, is difficult to control, especially if the susceptible weeds above mentioned are not eradicated.

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Foot Rot and Damping-Off of Tomato, *Phytophthora cryptogea* Pethybr. & Laff.; and *Phytophthora parasitica* Dastur

This disease of young tomato plants under glass was first discovered in 1913 in Ireland and has since spread to various places in Britain and elsewhere [4, 5, 6]. The same disease affects diverse plants such as potato, turnip, mangel, swede, and a number of ornamental plants, e.g. aster, cineraria, petunia, wallflower, gilia, and also beech seedlings.

The 'damping-off' phase of the disease occurs when the plants are young, in the boxes, and heavy losses are often incurred on young seedlings owing to early onset of the disease. Its action is very rapid, and collapse may follow within 18 hours of infection. The 'foot rot' stage attacks the plants later when, despite a good start in the boxes, they develop the disease after potting up, or planting out in the houses. Foot rot becomes apparent when the plants are about 5 or 6 inches high, and thereafter they appear to become more resistant. Early signs of the rot consist of a brown or black discoloration of the stem at, or just above, soil-level, shrinkage of the tissues in this region being followed by collapse of the shoot above the lesion, and though the leaves may remain green for a while, they later become rolled at the margins, turn yellow and die (Fig. 315). When affected plants are pulled up the discoloration at the base of the stem is seen to extend into the roots, but is not progressive above into the green shoot. The primary root

system suffers severely and is often rotted, and even adventitious roots which develop to replace the lost roots may also be attacked and the plant dies.

Both phases of this disease are caused by two species of *Phytophthora*, namely *P. cryptogea* and *P. parasitica* [6].

In a moist environment affected plants show the sporangiophores of *P. cryptogea* to be long and sympodially branched and bearing inversely pear-shaped but often much-elongated spores. The latter are blunt, non-papillate, and measure from 24 to 50 by 17 to 30 μ (average, 40 by 27 μ) [4]. The oogonia are terminal or lateral; the antheridia are amphigynous; the oospores are furnished with a wall about 3 μ in thickness, hyaline at first but later straw coloured; they are about 25 μ in diameter; the oospores have not been observed to germinate. Direct germination of the spores is not common and they function mostly as sporangia forming a number of zoospores within an extruded vesicle, and after escape of the zoospores new sporangia may arise by proliferation.

P. parasitica, besides playing a part in this disease, also affects the stem (see 'stem rot' below) and causes a 'buck-eye' rot of tomato, affecting the unripe fruit and causing a brown discoloration at the top end [7]. It attacks seedlings and older leaves of the castor plant in India, and also a number of other plants. The sporangiophores are slender and unbranched, and measure from 35 to 500 μ (usually from 100 to 300 μ) long; the spores are single, terminal, colourless, ovoid or round, and measure from 25 to 50 by 20 to 40 μ; they have a papillate apex and germinate as sporangia to form zoospores, but old spores, or when not sown in water may often germinate direct. The germ-tubes may form a mycelium or give rise to secondary spores which again may either germinate direct or develop zoospores. Chlamydospores are also sometimes produced; they are round, thick-walled, yellow, and measure from 20 to 60 μ in diameter; they retain their germinative capacity for many months after the sporangia and hyphae are dead; they give out one or more germ-tubes which may form a mycelium, spores, or chlamydospores again. The oospores, found in culture, are round, colourless, and measure from 13 to 24 μ (average, 18·6 μ) in diameter, and are formed within oogonia, 15 to 27 μ wide, furnished with a thick yellow wall. Like the chlamydospores, it is probable that the oospores, if formed in nature, serve the purpose of carrying the parasite over from one crop to the next.



FIG. 315.—Foot rot and damping-off of tomato (*Phytophthora cryptogea* and *Phytophthora parasitica*). Top, the plant breaking at stem base. Below, left, affected stem; right, healthy stem (photos by Foister & Noble)

Infection probably originates from the soil, but in what form the fungus survives in this medium is not known; when tomato seeds were planted in soil

in which a diseased crop had grown, young seedlings showed infection as soon as they were about 2 inches high [1]. The disease starts in the roots and travels up for a short distance into the stem but does not enter the latter much higher than the level of the soil; successful infection may also ensue if inoculation of the stem is made at soil-level. The tissues affected are those of the pith and cortex. The non-septate and branching mycelium is intercellular at first, but in the moribund tissues it also occupies the cells. In young seedlings hyphae may sometimes be seen in the wood vessels, but in older plants the xylem does not become invaded until the rotting is well advanced [6].

The optimum temperature of 25° C. for the growth of *P. cryptogea* is lower than that for *P. parasitica*, which is 36° C. Below 12° C. both grow slowly, and to check the disease the temperature of the glasshouse should be as low as possible without impairing growth of the crop. A relatively high percentage of soil and air moisture favours the spread of the disease [1].

As the trouble does not attack the shoot, cuttings may safely be struck from diseased plants and the remainder destroyed [6]. Since the disease is soil-borne, soil for raising seedlings should be treated with Cheshunt compound [3, 8] (see p. 242), and the young plants may be watered during growth with the same mixture, directed towards the roots. If signs of the disease reappear, affected plants should be removed immediately and the hole watered with one pint of the compound before inserting a new plant [3]. Another method of treatment consists of top-dressing with a mixture of 10 parts of lime and 1 part of copper sulphate, applied to the soil at the rate of $\frac{3}{4}$ oz. of the mixture per square foot [2]. Soil sterilisation may also be effected by adding a mixture of 15 parts of formalin and 85 parts of sawdust, charcoal, or air-dried soil, at the rate of 4 to 5 parts per 1,000 parts of soil [5a].

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Stem Rot of Tomato, *Didymella lycopersici* Kleb.

Stem rot, or canker of tomatoes, under glass and in the open has long been familiar to growers in Europe. In 1906 severe losses were witnessed in the Lea Valley district, and though the trouble abated for a while it appeared again in several English counties [13]. A few other members of the tomato family, weed and cultivated, are liable to the same disease, but as the trouble is conveyed in soil, none of these hosts has so far been reported to carry infection to the tomato.

It is not usual for this disease to attack very young seedlings (they did not become infected when sprayed with a spore suspension [18]), and the first symptoms appear mostly about 4 weeks after planting out. There is some evidence, how-

ever, that the disease may be seed-borne [4b]. Recently, too, in the West of England, seedlings in glasshouses bore evidence of cotyledonary infection, picked up probably from the seed. Even when such seedlings perish, they remain on the soil to infect older plants [11a]. On these, the first symptoms occur on the main stem at soil-level (Fig. 316), and from primary infections set up in this region, occasionally below, the disease spreads secondarily to all parts of the shoot, including flowers and fruit, but the main stem is the part to suffer most. Just above soil-level small brown spots appear and spread rapidly to form a dark-brown lesion which may girdle the base of the stem. Here the cortex rots away and, if scraped off, the woody tissues of the stem are seen to be stained brown for a few inches above the lesion; wilting and death of the shoot usually follow. Young plants may throw off infection if they produce new roots above the lesion, and may grow forward for a long time before finally wilting [16]. Later in the season secondary infections follow, and parts of the stem higher up, including leaves, leaf stalks, flower stalks, and sepals, become affected, apparently by air-borne spores [12, 17a]. Infection of the fruit may occur at any point, mostly at the stalk end, and the fruit falls off. Fruit infection followed by rot is more common on outdoor tomatoes and rarely occurs under glass. Experiments on outdoor tomatoes at Evesham have shown the existence of two strains of the stem rot organism. One of these which attacks only the fruits will not infect the stems of outdoor or glasshouse plants, but the strain attacking outdoor stems will also infect the stems of glasshouse plants [17a]. Secondary infections are caused by spores developed within pycnidia on old lesions, and which may be carried over the crop during watering, or by wind. On the leaves, under very damp conditions, small, grey, circular spots arise which may spread so as to destroy the entire leaf. The surface of the fruit may also become covered with a black crust laden with pycnidia [13].

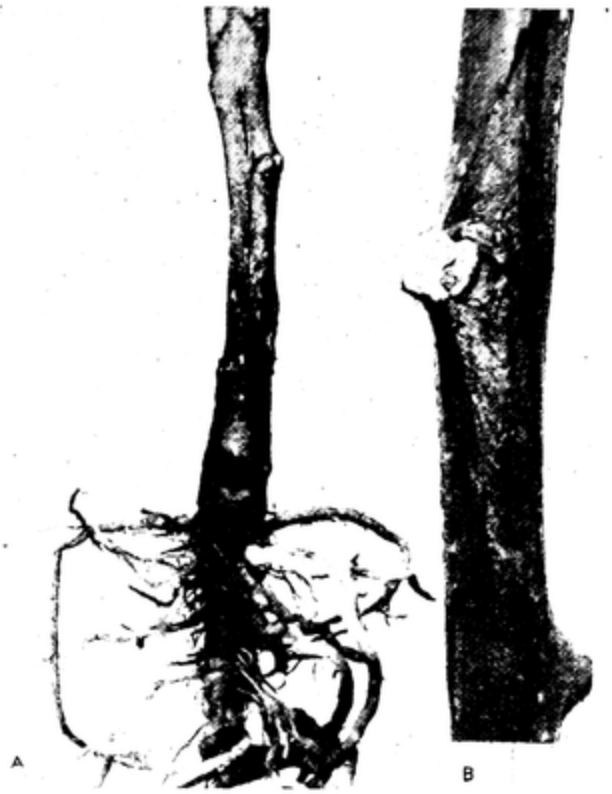


FIG. 316.—Stem rot of tomato (*Didymella lycopersici*). *A*, showing lesion at soil-level; splitting of the stem sometimes occurs in old lesions. *B*, pycnidia on stem above ground (photos by Bewley)

Stem rot is caused by an Ascomycete *Didymella lycopersici* (Pyrenomycetes). (In 1943, however, a few cases of stem rot in Hertfordshire and Hampshire were caused by the foot-rot fungus *Phytophthora parasitica* described above (p. 665); long, greyish-green lesions were found on the stems, the cortex and pith were affected, and the stems eventually became girdled [17]). The pycnidia (*Diplodina lycopersici*, see p. 53) are commoner than the perithecia, and the latter have only been seen in Britain quite recently [4a].

Pycnidia occur numerously on stem lesions and on the fruit. On the former they are small and black ; on the fruit, pale brown at first, then black, exuding pycnospores in flesh-coloured tendrils ; they have also been found on the seeds [14], but infected seeds rarely give rise to diseased plants, though occasionally a reduction in percentage germination has been observed [12]. The pycnidia are scattered, or aggregated on raised spots, sub-epidermal, ostiolate, from 100 to 270 μ in diameter ; pycnospores are 1- or 2-celled, sub-cylindrical, from 4·5 to 17 by 2·5 to 5 μ [2]. Perithecia have been found intermixed with pycnidia on tomato stems kept dry, or over-wintered in the open. They are subglobose, dark brown ; asci are cylindrical, 70 by 95 by 9 to 10 μ, 8-spored ; ascospores are spindle-shaped, 1-septate, hyaline, 16 to 18 by 5·5 to 6·5 μ [5]. In liquid cultures, with tomato juice, the fungus develops a dense mycelium, and from pycnospores sown on tomato agar mycelium and pycnidia are formed, but no perithecia [1, 12]. The cardinal temperatures for mycelial growth are, minimum, 4° to 5°, optimum, 20°, and maximum, 31·8° C.[8, 12].

The fungus survives in decayed stems and leaves [11] in the open for over twelve months, and infection may be contracted from soil in which diseased plants have grown. The fungus has been isolated from soil to a depth of 5 cm. and is capable of saprophytic existence [12]. An instance is known where the ploughing-in of infected debris was followed by almost complete loss of the crop on the same ground the following season ; and the fungus is known to live from year to year in cracks of supporting sticks [7], crevices of boxes, wooden supports, on string, wire, etc. Infections are carried over from such contacts, but mainly from contaminated soil, and secondary infections are effected by pycnospores which may also be air-borne to glasshouses. Recent observations in England [4a] have shown that while pycnidia may be found on the upper part of a stem lesion, the perithecia were found below them and down to soil-level in the disintegrating cortical tissues. Moreover, the perithecia may occur at any time of the year ; their occurrence on the stem near to soil-level suggests that moisture supply and, possibly soil nutrients may be factors governing their formation.

Infection of the stem is possible through unwounded cuticle, or stomata. After dissolving the middle lamella the fungus kills the cells of the entered stem, early destroying the cortex, and in severe lesions penetrating as far as the woody tissues [4, 8]. Inoculation of the fruit, green or red, is apparently successful only through wounds in the skin, such as would be caused by careless tying-up of the plants with bast or string, or when shoots or flowers are removed [7, 9, 17]. Infection of the stem at soil-level is also more rapid after wounding than without, and infection can also be carried from one plant to another on the pruning knife [13].

Stem rot of tomato is greatly encouraged by a high degree of atmospheric humidity and comparatively low temperatures of 5° to 15° C., for above 22° C. resistance of the host is found to increase considerably. Infection is not so evident on light as on heavy soils, but soil texture seems not to affect the degree of fruit infection [12]. Seedlings in very acid soils are particularly liable to infection, and spore germination takes place over a range of *p*H 4·3 to 7·4, and mycelial growth over 3·9 to 8·10, the optimum being at *p*H 5·23 [8].

Since stem rot of tomato is carried in the soil some growers adopt the plan of sterilising the soil by heat or chemicals. Soil disinfection may be carried out

with carbon bisulphide, at a rate of 5 c.c. per square metre, to a depth of 10 cm., or uspulun dust spread at a rate of 50 gm. per square metre [10]. Experiments at Cheshunt [13, 18-21] have shown, however, that soil sterilisation appears to have little control over this disease which recurred after sterilisation either with steam or chemicals. Air-borne spores of *D. lycopersici*, carried from tomato haulms left outside during the winter appear continually to reinfect the soil and may enter the glasshouses in this way, or in soil on the feet of workers, on knives, or by splashing when watering. The spores percolate to a depth of $\frac{1}{2}$ in. and even deeper if the soil has been steam sterilised. But good control was obtained when the soil was treated with ethyl mercuric phosphate (1 gall. per sq. yd. of a 1 in 16,000 solution) two days before planting. As the point of attack on the stem is at soil-level, sterilised soil or peat should be packed around the stem bases of the seedlings some 14 days after planting [6, 12]. Greenhouses and all supports for the plants should be treated with 1 per cent. formaldehyde [15] and all infected debris burned. No varieties of tomatoes are, so far, known to be immune from this disease [8, 12].

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Verticillium Wilt of Tomato,

Verticillium albo-atrum Reinke & Berth. & *V. dahliae* Kleb.

' Verticillium wilt ' is common in Britain on tomatoes under glass. It occurs also in the Channel Islands [2], in widespread localities in the United States and Canada [4, 11], in Holland [9, 12], New Zealand [5], and in the cooler parts of Australia [3].

This disease usually makes its appearance early in the season, about the middle of April, increasing in severity up to the third week in May, and declining as the



FIG. 317.—Verticillium wilt of tomato (*Verticillium albo-atrum* and *Verticillium dahliae*) (photo by Bewley)

soil temperature rises, up to August, but may reappear as the plants die down [2, 3].

Encouraged by incorrect conditions of the soil, this disease attacks the roots and, by interfering with the water supply to the growing plants, induces a wilting of the shoots (Fig. 317). Affected plants may, however, recover overnight when transpiration is reduced, but lose turgidity and wilt again during the morning. Wilting of older plants starts with the lowermost leaves and proceeds upwards. The yellow spots which appear on the older leaves may become so numerous that entire leaves, yellowed and shrivelled, drop off. But some varieties of tomato, though showing considerable wilting, may not show so much yellowing of the foliage as others. A diseased plant exhibits considerable browning and rotting of the roots at the tips, and if the plant is split in half from root to stem the brown discoloration may be traced from the root to the base, and sometimes all the way to the top of the stem [3]. The actual browning is present in the walls of the wood vessels and a brown gummy substance is frequently seen to line them inside, and in places to block some of them more or less completely.

This disease is caused by *Verticillium albo-atrum* and *V. dahliae* (Hyphomycetes). Considerable discussion has, however, arisen over the identity of these two fungi [1, 5, 6, 7, 8, 9, 10, 11, 13, 14, 15], some maintaining them to be distinct species, while others believe *V. dahliae* to be only a strain of *V. albo-atrum*. While there is some measure of agreement that the two differ in the type of resting body produced, *V. albo-atrum* developing a resting mycelium composed of dark, thick-walled septated hyphae, and *V. dahliae*, better-organised bodies of the nature of microsclerotia, it has recently been reported that monosporic cultures of *V. albo-atrum* isolated in Minnesota [14] developed a diversity of saltants, and both the dark resting mycelium associated with *V. albo-atrum* and the microsclerotia typical of *V. dahliae* were obtained from a monospore culture of the former. It is also asserted that the resting bodies of *V. dahliae* are really pseudosclerotia, as they result from the budding of a single hypha, becoming eventually dark, knob-like, thick-walled structures, and are not true sclerotia since they do not possess an intertwining system of hyphae and a cortex [1]. *V. albo-atrum* is said to be more pathogenic than *V. dahliae*, from which it also differs in having a lower maximum temperature and a slower rate of growth on artificial media [8]. Eleven strains of the former and four of the latter have been isolated from tomatoes in England [16].

The fructifications appear chiefly on the surface of the stems at soil-level, and consist of small tufts of erect conidiophores which break through the epidermis. The conidiophores are branched in characteristic fashion, forming 1 to 3 whorls of branches with 1 to 7 branchlets in each whorl. The main conidiophore and the various branches bear terminally either single conidia or what appear to be spherical sporangia, but the latter are really

only dense aggregations of conidia held together in mucilage. These conidia are set free when the mucilage is dissolved in water. The conidia are continuous, rarely septate, hyaline, elliptical, and measure from 4 to 8·5 by 2 to 3·5 μ (average, 5·1 by 2·6 μ). In culture the septated mycelium, at first hyaline, later becomes dark brown and thick-walled, showing all gradations from slightly swollen, sparsely septate, to much swollen many septate hyphae, from 2 to 4·5 μ in diameter ; numerous chains of chlamydospores measuring from 5·5 to 7 by 6 to 8 μ, were also produced in old cultures of potato-dextrose agar [5].

The organisms are believed to over-winter in the soil or on host debris in the form of the dark, thick-walled resting mycelium, or microsclerotia as the case may be, and probably also as chlamydospores. The two fungi are also capable of saprophytic existence on organic matter or on remains of host plants in the soil [12] ; and while conidia formed at the base of the dead plants may not survive through the winter, they readily germinate and, thriving upon the decayed debris, produce a resistant mycelium which is capable of living through the winter [2]. The conidia, carried in air currents or splashed up from diseased stems during watering, serve to start new infections during the season.

Experiments have shown that *Verticillium* is capable of penetrating the roots of young seedlings without previous injury [2, 8], but it is not clear that older plants can be entered at root or stem except through wounds. Inoculation into the hypocotyl, or into an internode of the stem, produces the symptoms of wilt much sooner than when the inoculum is placed in the soil [2]. The fungus penetrates the root or wounded stem as far as the stelar tissues, entering the wood vessels, as already stated, staining the lignified walls brown, and growing up into the vascular bundles of the stem for quite long distances. But the actual wilting is believed to be due not so much to the occlusion of the water channels by mycelium as to the secretion of a toxic substance by it which is carried up to the leaves in the transpiration stream, thus causing their wilting and death.

Temperature plays an important part in the incidence and control of *Verticillium* wilt. It is essentially a disease of moderately cool temperatures ; an average temperature of 20° C. is favourable to its spread ; at 12·5° C. it is not so active, while a temperature of 25° C. practically checks it, and no infection occurs at 30° C. [8]. The wilt may, therefore, be controlled if the temperature is raised to 25° C., but this temperature must be maintained for several days otherwise there is a set-back. Long exposures to this temperature are more lasting than short ones applied intermittently. Recent experiments at Cheshunt suggest that while *V. dahliae* is checked to some extent by the higher temperature and humidity, the results are not so decisive as in the case of *V. albo-atrum* [17]. During such periods of higher temperature, affected plants may become re-established by virtue of the opportunity given to develop new adventitious roots at the base of the stem, and may thus grow into productive plants.

The wilt occurs most commonly in soils which contain a large amount of organic matter, and is also prone to appear on clay loams, the reason in both cases being perhaps the greater retention of water around the roots under these conditions. Moreover, the cooling effect thereby produced favours the fungus, an effect which would not be so evident in a light porous soil [2, 3].

To check the disease, any plants showing early signs of wilt should be carefully dug up along with some of the soil around their roots, and the hole watered with a solution of Cheshunt compound (p. 242), before putting in a healthy plant, after which the soil around is watered again with the same solution. Older plants should be encouraged to develop new roots by earthing-up around the base and covering the soil with peat litter. As above stated, the disease in the greenhouse can be controlled by maintaining a temperature of about 25° C. (77° F.), or above, over several days, during which it is also advisable to give shade by a light application of whitewash to the glass. The soil must not be watered too freely but the foliage should be given a light watering [2]. As these fungi are soil inhabitants, treatment of the soil during winter by saturation with a 2·5 per cent. solution of formalin is also recommended [12].

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Tomato Leaf Mould, *Cladosporium fulvum* Cooke

'Leaf mould' is the most serious disease that affects tomato plants under glass; it is rather unusual to find it on outdoor crops. It was first described in 1883 by Cooke [7], in England, from material which had been sent to him from South Carolina, and it is probable that the disease originally came from South America, the home of the tomato. It is essentially a disease of the foliage leaves though it is known in some countries to attack the flowers, and on rare occasions the fruit as well, but in England only a few cases of fruit infection are recorded. It occurs on no other host than the tomato, very few varieties of which are resistant to it.

In this country there is usually little evidence of the mould in the glasshouse before April or May, though it has been known to break out as early as January on the cotyledons of plants just potted [20], but in general the first outbreaks occur in June or July, on the fully expanded leaves, usually on those at the base of the plant. The upper leaves may remain quite clean right up to the end of the season, except in epidemic attacks when entire plants may be destroyed. To the casual observer the first symptoms consist of pale-yellow spots on the upper surface of

the leaves, but actually, on the under surface, below these discoloured spots the mildew organism has already been at work, and on these areas there are tufts of mould, at first downy and light grey in colour, later changing to pale buff, and finally, as the spots enlarge, to a tawny brown colour (Fig. 318). Where the spots are advancing on the leaf tissues the margin remains downy white, while the brown, smooth, and velvety centre indicates that the spots are in active sporulation, the parts being densely covered with brown conidiophores laden with smooth hyaline conidia constituting the sole fructifications of the fungal organism causing this disease.



FIG. 318.—Leaf mould of tomato (*Cladosporium fulvum*). The spots on the leaves ; upper surface (left) ; under surface (right) (photo by Bewley)

The causal organism is *Cladosporium fulvum*, a member of the Hyphomycetes (Fungi Imperfecti). The conidiophores emerging through the stomata on the under surface of the leaf (Fig. 319), rarely the upper, are septated, with knee-like joints, olivaceous in colour, producing terminally or subterminally a few hyaline or faintly brown conidia which vary considerably according to size, shape, and degree of septation. For the most part, conidia are 1-septate and ellipsoid, slightly constricted, measuring from 10 to 20 by 4 to 6 μ [7] ; unicellular, and up to 4-celled conidia may also occur, especially in culture, even on the same conidiophore [25]. Germination may take place by one or two germ-tubes [14]. The conidia are highly granular and uninucleate [15]. Sporulation is abundant within a range of temperature from 20° to 26° C., the optimum being about 22° C., the maximum, 31° to 33° C., and the minimum, 0° to 1° C.[14].

There is little doubt that the fungus can survive in the greenhouse from the time the old plants are cleared away to the planting of the next crop, in the form of conidia which lurk in the crevices of brick- and wood-work and on window sashes, as well as on soil left uncleared from the benches. Even the smallest bits of the dried, infected foliage left over winter in the greenhouse are sufficient to start fresh infections in the spring [26], but there is, so far, no conclusive evidence that the fungus can subsist as a saprophyte under natural conditions. In certain cases of fruit infection recorded in America, it is said that the mycelium in the diseased pulp, while in no case being capable of penetrating the embryo within the seed, may, however, collect in more or less dense masses within and upon the seed-coat, remaining on the seed and resisting desiccation for long periods. During the germination of such seed it was reported that the empty testa contained abundant conidia when the cotyledons had emerged and there was copious sporulation on both surfaces of the cotyledons. The planting of infected seed may in certain localities, therefore, set up primary infections, and thus supply sufficient

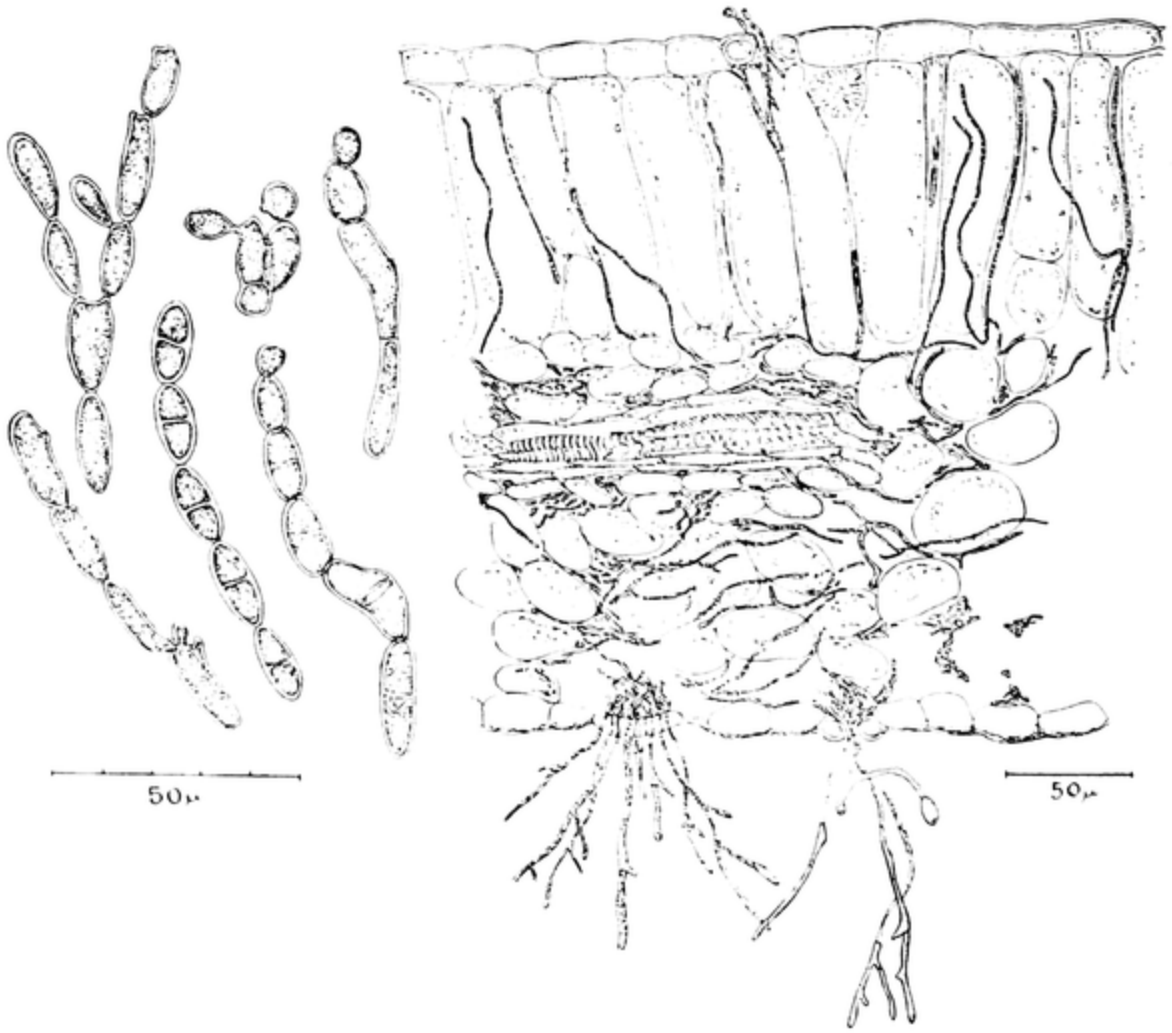


FIG. 319.—*Cladosporium fulvum*. Section of tomato leaf showing the thin hyphae chiefly in the intercellular spaces of the spongy mesophyll and the conducting parenchyma; long 'runner' hyphae pass between the cells of the palisade mesophyll. The conidiophores emerge chiefly at the lower surface, few at the upper surface; sphaero- and needle-crystals are seen in the palisade tissue. Left, various modes of development of the one- or two-celled conidia (from a slide by Macfarlane)

conidia for dispersal by air currents and watering of the plants, to carry infections to other plants in the bed [9]. Others, who do not believe the disease to be seed-borne, suspect that cotyledonary infections may be brought about during germination in contaminated soil [20]. There is abundant evidence that the spores are highly resistant to desiccation, chemical action, and the low temperatures of winter, and may survive these extreme conditions for several months [11].

The conidia germinate readily (under conditions specified below) if they settle on the under side of the expanded leaf. The germ-tubes, without development of appressoria, enter the leaf through the stomata and long, straight runner hyphae are soon established in the spaces of the spongy mesophyll (Fig. 319). After coming into contact with the host cells in this region, especially with the conducting parenchyma investing the veins of the leaf, the fungus goes further afield into the intercellular spaces of the palisade mesophyll, and apparently is only able to thrive as long as the host cells remain alive [4, 5]. Only after the death of

the cells does the fungus actually enter them, and the appearance of the yellow-coloured spots mentioned above as occurring on the upper surface of the leaves is associated with the death of the palisade cells situated above the sporulating areas.

Infection of the flowers usually takes place through the sepals, but may also occur through the pedicel, receptacle, or the style, and it is noteworthy that all these parts are furnished with stomata. Whether the complete absence of stomata from the wall of the fruit makes it impossible for the fungus to attack it direct is not known but it appears that all attempts to inoculate the fruit through the skin have failed. Fruit infection which results in a blackening of the tissues, especially towards the stalk end, is believed to take place indirectly by way of the stomata of the sepals or pedicel, the fungus finding its way into the receptacle and finally into the fleshy placenta of the fruit, which in consequence turns black, and according to some, the blackening extends to the seeds as well [9]. In seed infection the mycelium within the blackened pulp of the placenta has been observed to pass into the funiculus and thence into the outer integument of the seed, but in other cases even the blackest of seeds from affected fruit have failed to show the presence of mycelium either inside or outside the testa [20]. In any case, the effect on the fruit is a mere internal blackening and there is never sporulation of the fungus on the fruit itself, but abundant spores may be found on the attendant sepals and flower stalk.

In preparation for sporulation, the fungus aggregates in the sub-stomatal spaces of the leaf or other affected organ to form small stromata from which bunches of conidiophores pass out through the stomata to the exterior, and with the dissemination of the conidia by wind, secondary infections take place on a wide scale.

Infection is greatly influenced by the interaction of numerous factors, amongst which high temperatures and excessive atmospheric humidities play the most important part in increasing the severity of the disease [26, 27, 28, 30]. The optimum temperature for infection is about 22° C. (72° F.), and if humidity is as high as 98 per cent. atmospheric saturation the disease in the greenhouse spreads apace; at the same temperature but with the relative humidity down to 79 per cent. there is less disease, while at 65 per cent. there is hardly any, few spores being capable of germinating on the surface of the leaves at this relatively low degree of moisture, and at a humidity of 58 per cent. diseased spots may actually dry out; at 95 per cent. atmospheric humidity the incubation period was found to be only 12 days, whereas at 50 per cent. it was 15 days [31]. But even if the weather during the summer months is hot and dry, warm humid conditions existing in the greenhouse at night are responsible for much disease, and this despite all attention given to the ventilation of the house during the day. In general, it would appear that the persistence of a humid condition *during the summer nights when temperatures are high* is the main factor encouraging spread of disease, and in the proper control of night humidity lies the best means of combating the trouble. The fact that the disease is favoured by high temperatures does not mean that infection may not occur during cool weather; it is highly probable that infection is constantly taking place, but the prevalence of low temperatures retards or checks

the progress of the fungus within the plant. As soon as warmer conditions arrive, however, the fungus develops, sporulation is stimulated, and crops which to all appearances looked healthy may in a few days be ruined. Such quick changes of temperatures, from low to high, no doubt explain the rapidity with which the disease appears on seemingly healthy plants.

As already mentioned, the plants are more prone to an attack during the night, their exposure to light having a retarding effect on the fungus, and spore germination may actually be checked in strong light [11, 16]. Under the conditions existing in a humid atmosphere, therefore, the environment presented by the expanded leaf, at its lower surface, appears to be ideal for infection of the plant, for here the stomata, far more numerous than they are on the upper surface, are moreover prevented from transpiring too freely by the presence of capitate glands on the lower epidermis, and, screened from direct light, the spores have all the conditions for successful attack.

Extensive studies conducted by various authors [2, 11, 15], have shown the existence of several physiologic races of *C. fulvum*, exhibiting diversity in such cultural characters as growth rate, topography, colour, amount of sporulation, etc., and these differences arise whether the inoculum used consists of unicellular or multicellular conidia. It is important to note, however, that, in addition to variability of pathogenic powers possessed by genetically different races of the parasite, variations may also be observed when a pure line of the host is infected by a specified race of the parasite under variable conditions [15].

The tomato plant does not react to infection in the same way at all times of the year. It seems to have a winter reaction and a summer reaction to this disease, influenced possibly by the length of day and relative intensity of light during these seasons. A reduction in the amount of light falling on the plant, as in winter, is held to be largely responsible for the lack of expression of the genetic factor which controls resistance to the disease [15]. A remarkable feature, due possibly to the interaction of numerous factors which control the nutrition of the host, is that *C. fulvum* attacks its host with greater vigour when the plant is well nourished ; on starved, chlorotic plants the distribution of the fungus is restricted and appears to be confined mostly to the younger leaves, and sporulation on these anaemic plants is much less than on vigorous plants.

In a series of experiments on host nutrition, infection was favoured when excessive quantities of all the usual minerals were given, as well as by increased nitrogen, and by a deficiency of potash. An alteration in the type of infection, shown by a variation in the colour of the mycelium, indicated that changes in the nutriment of the host altered not only the quantity, but also the quality of the food supplied to the fungus [21]. Experiments in Moscow showed that a toxin produced by *C. fulvum* in culture affected the development of the seed embryo, and also poisoned the host cells and destroyed the chlorophyll [8].

Since the disease is favoured by high atmospheric humidity the best means of control is by ample ventilation of the greenhouse, especially at night. Some growers attain this object by maintaining a high night temperature when the ventilators are open, but this procedure is open to objection, since during warm, rainy periods it may not always be possible to maintain a sufficiently low degree

of relative humidity to prevent infection [1]. Watering the plants should be carried out in the early morning preferably on bright days, so that the ventilators can be kept open and thus ensure comparatively dry conditions at night. Houses specially erected for commercial growing should be planned with due regard to location and facilities for ample ventilation. Forcing the plants, especially when planted late in summer, should be avoided. Should the disease appear, it may be advisable to remove the leaves at the base, and the plants treated with fungicide. Treatment with finely divided sulphur dusted on to the leaves, directed particularly at the under surface, or applied by means of a vaporiser, is usually beneficial if applied in time, usually in early April, and repeated at intervals of 10 days until the end of June, but care must be taken to avoid scorching the leaves. Good control has been obtained by spraying with a fluid made with salicylanilide paste $\frac{1}{8}$ oz., and $\frac{1}{4}$ oz. 'Agral I' or $\frac{1}{8}$ oz. 'Agral N', in a gallon of water, applied at the first signs of the disease, the operation being repeated 7 days later [3, 29]. A utility programme, for combined attack on the fungus and red spider trouble, is the employment of petroleum oil emulsion, 1 gallon in 100 gallons of water, to which is added $\frac{1}{2}$ oz. of colloidal copper for each gallon of the diluted emulsion [18, 19]. After the plants have been cleared out of the house, thorough cleansing should be carried out, for which purpose various disinfectants are recommended, such as cresylic acid with soft soap [26], or fumigation may be carried out with formaldehyde or sulphur dioxide [6, 13, 17].

Some varieties of tomato show considerable resistance to leaf-mould disease, e.g. Main Crop, Norduke, Satisfaction, and Up-to-Date; the variety Giant Red is very susceptible. Most of these resistant forms, however, yield fruit of inferior quality [26]. In trials conducted at Cheshunt, the Canadian variety Vetomold (a hybrid between Potentate and the Red Currant Tomato, *L. pimpinellifolium*) proved resistant to *C. fulvum*; it appears to do well where the soil is rich and open, and where warm, moist conditions can be provided [3a]; but in some localities its resistance has apparently broken down, due, perhaps to the appearance of a new strain of the parasite [33], for two such strains are known to exist in Canada, Vetomold being immune from other strains [2a, 32]. There are good hopes that by hybridisation, especially of the highly resistant *Solanum racemigerum* and *Solanum* (*Lycopersicum*) *pimpinellifolium* with various types of the commercial tomato, new types of large fruit tomatoes completely resistant to leaf-mould disease will be developed [10, 12, 23]. The expressed sap of the resistant *Solanum racemigerum* is said to contain an anti-germination principle in which the spores of *C. fulvum* do not grow [22, 24].

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Blossom End Rot of Tomato (*non-parasitic*)

This disease of tomato fruits is a physiological disorder, believed to be caused by a derangement of the water balance between the leaves and the fruit during its development [2]. Though numerous bacteria and fungi have been isolated from the rotted fruits, these organisms are not the primary cause of the trouble [4, 5, 6, 7]. The disorder is particularly prevalent on the early crop in Britain [2], and occurs every year in all areas where the tomato is grown. In West Australia [8] it does considerable damage during hot dry periods; in New Zealand and Tasmania [11] it is a serious fruit rot both in the glasshouse and in the field. The considerable reduction in yield, due to the trouble, is said to be correlated not so much with the number of affected fruits produced by an individual plant, as to the different fruit-setting capacities of individual varieties [15].

On the earliest formed fruits, first symptoms of the trouble are seen at or near the blossom end of the young fruit. The part affected presents a water-soaked appearance and may be confined to quite a small area, or may extend so as to cover about half of the fruit. The affected area soon turns dark brown or even black, but does not usually extend much once the fruit has turned red, and the area finally remains as a flattened dark-coloured lesion at one end of the fruit (Fig. 320). The tissues below the affected part eventually collapse and may become invaded by numerous saprophytes, but the final condition is that of a hard leathery rot [9].

As the trouble is closely related to the water supply of the plant, it is obviously influenced by numerous factors. A plant endowed with a healthy root system, growing under equable conditions, in a soil sufficiently damp, is rarely subject to this disorder, but if the water supply is inadequate to meet the general require-

ments, there is a tendency for water to be withdrawn from the developing fruit, with the result that tissue changes take place at the blossom end which result in the symptoms above described [2]. Plants receiving a moderate supply of water are less liable to the trouble than those lightly or excessively watered. In the same way, a sudden check to the normal intake of water by the roots, especially of those of fast-growing plants, may also induce the trouble [4], as may also a dry soil or a defective root system that is unable to cope with the requirements of the plant [15].

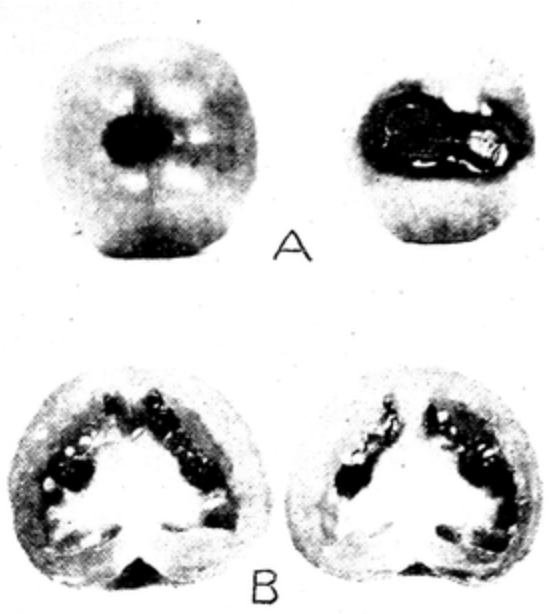


FIG. 320.—Blossom end rot of tomato. The lesions at the top of the fruits, and fruit in section (photo by Foister & Noble)

Experiments with tomato seedlings growing in culture solutions have shown the plants to be dependent on abundant water supplies for rapid growth and fruit development. In such solutions of low concentration of the minerals growth was facilitated, but in those of high concentration growth was limited. No symptoms of blossom end rot were manifest in cultures of the lowest concentrations, but about 80 per cent. of the fruit suffered in the series of highest concentrations. Moreover, the trouble was evidently associated with wide fluctuations in the rate of transpiration, so that any factor seriously restricting the rate of absorption of water or effecting a rise of temperature will favour it [13]. In New Zealand, plants grown on a light sandy soil, especially on dry, gravelly ridges, suffered little when regularly irrigated, but when left for a period without water very heavy losses were experienced, and the more vigorously growing, sappy plants were the most susceptible to this disorder. The trouble appeared, too, when vigorous plants with well developed fruits were removed from a shaded to an unshaded glasshouse, while it did not occur on similar plants left in the shaded house [9].

The disease is aggravated by excessive use of nitrogenous manures, for by the development of much top growth and greater demand on the root system, the supply of water to the leaves is rendered still more difficult [2, 6, 10]; increased application of phosphatic manures, on the other hand, tends to reduce the trouble [10]. It is asserted that unbalanced mineral nutrition is liable to cause the disorder by preventing the utilisation of the necessary amount of calcium for normal fruit production; when the calcium in the fruit fell below 0·2 per cent., the rot followed [12a].

The most obvious method of controlling blossom end rot is to maintain an equable environment, and especially to keep the soil uniformly moist [3]. This is particularly important when the fruit has begun to develop [9, 11].

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Tomato Mosaic

Mosaic, sometimes called mild or tobacco mosaic, is the best known of all virus diseases which attack the tomato plant. It has a wide range of hosts and is extensively distributed. The virus is so highly infectious to its susceptible hosts that it has probably been carried to the tomato crops in various countries in smokers' tobacco in which the infective principle remains viable over long periods. Though the commonest of virus diseases of tomatoes under glass, it is not so severe a malady as the yellow, so-called aucuba mosaic of tomatoes described below, but nevertheless accounts for considerable reduction in yield, its adverse effects on fruit-setting being more severe when infections occur in the early spring than later in the season [68, 80].

Symptoms of tomato mosaic differ somewhat according to the season (Fig. 321). Spring and early summer infections are familiar in the form of a mottling of the leaves, consisting of slightly raised dark-green areas, accompanied by some distortion of the youngest foliage. Leaf deformity is more marked in the winter, a 'fern leaf' effect being produced, while the development of anthocyanin in the stems of young plants often imparts a distinct purple colour to these parts, and there may also be some degree of stunting. When infections occur in older plants, after they are about a foot or so high, the mottling effect shows only in the new foliage, the older leaves having remained healthy from the start [1, 10]. There is no necrosis of stems or leaves, and while the fruit may appear quite normal, blemishes sometimes appear before the fruit is ripe if infection happens to have been early, otherwise there is little evidence of disease on the ripe fruit.



FIG. 321.—Tomato mosaic
(photo by McKay)

Tomato mosaic is caused by one of the best known of all plant viruses, *Nicotiana virus 1*, one of the numerous viruses which attack the tobacco plant. The following are listed as synonyms : *Tobacco mosaic virus* (Allard 1914) ; *Tobacco calico virus* (Clinton 1903) ; *Tobacco mosaic virus* (Clinton 1909) ; *Ordinary mosaic virus* (Johnson 1926) ; *Tobacco green mosaic* (McKinney 1926) ; *Tobacco mosaic virus A* (Fernow 1926) ; *Tobacco true mosaic virus* (Valleau & E. M. Johnson 1927) ; *Tobacco severe mosaic type 1 virus* (Johnson, E. M., 1930) ; *Tobacco distorting mosaic virus* (Duggar & B. Johnson, 1933) ; *Ordinary field type tobacco mosaic virus* (Kunkel, 1934) [77] ; *Ordinary or mild tomato mosaic virus* (Ainsworth 1933) [86].

The virus of tomato mosaic is sap-transmissible and so infectious that mere contact of a leaf surface with a contaminated source is sufficient for transmission. So far as is known, unlike most of the virus diseases of plants discussed here, it is not spread through insect agency, though some do not preclude this possibility, but the virus is conveyed with the greatest of ease by the pruning knife or on the hands or clothing of workers. Even cigarettes and cured tobacco have been shown to carry the virus and the infective principle can be disseminated by smoking or chewing of contaminated tobacco [20, 38]. The virus is known to be potent in dried plant remains for years, this being an undoubted source of spread in the soil. It is of the greatest importance, therefore, when working in the glasshouse to wash the hands frequently when handling new plants, and no contact should be made with infected tomatoes, for the merest snapping of an epidermal hair on a leaf will start infection. Where possible, all material suspected of infection should be placed in the care of one person. The apparent ease with which tobacco plants became infected by merely atomising the diluted virus (even as low a concentration as 1 in 100,000 was effective) on to the foliage, has suggested that infection might occur by the passage of the virus through the open stomata, but the mechanism of further penetration into the mesophyll from the sub-stomatal cavities has not been elucidated, and there is, as yet, no conclusive evidence of any connection between the size of the stomatal apertures and the incidence of infection [42]. However, it has been definitely shown that, following upon leaf inoculation, the virus travels right down to the roots, the path presumably being the phloem, the sieve plates in the walls of the cells no doubt assisting in the transmission (Fig. 174) [63]. After the virus has reached the roots it is probable that its spread upwards into the shoots is helped by the transpiration stream, for this process has been shown to be much more rapid in diseased than in healthy plants. Moreover, a sharp drop in the rate of transpiration was found to coincide with the appearance of symptoms of tomato mosaic, followed by a gradual increase [49]. It has been observed that living tissues of tomato plants infected with this virus hold certain crystalline inclusions which remain unchanged for an indefinite period, and it is believed that a considerable proportion of the potent principle present in infected plants is actually contained in this crystalline material [46].

As already mentioned, the virus of tomato mosaic remains potent in infected debris from previous crops for years [20]. Seedlings planted in contaminated soil contract the disease if they are at all injured or damaged by rough handling and tying [10]. Apart from direct contact of injured plants, there does not appear to be much danger of spread from a few such injured specimens acting as foci of

infection for the rest of the crop. Such infected plants should, however, be removed as soon as detected in the glasshouse. The possibility of transmission of the virus by seed has lately received considerable attention. While it still remains, however, to be fully proven, the rising trend of opinion is that seed transmission does take place [16, 17, 23, 24, 38, 58, 81]. But in the first instance it is not clear how the seeds become infected, whether from mere superficial contact with the diseased pulp, or internally, with possible penetration into the embryo ; the virus is present in the seeds of green and ripened fruits of the infected plants [17]. There is evidence that seeds collected from mosaic-diseased plants may give rise to diseased or normal plants, and marked differences have been observed between varieties raised from infected seed, both in the dates of the first appearance of the symptoms and in the apparent rates of spread within these varieties. A point of great importance has recently been raised, in that the transmission of mosaic-inducing viruses in the tomato crop might be considered to be a *delayed* phenomenon, a feature which is probably related to differences in the resistance of plants raised from seed of different origin, and to the multiplication and systemic spread of the virus. Moreover, under certain circumstances associated with malnutrition or incomplete maturation of the seed, viruses may enter the seed embryo, so that there would appear to be strong probability that a delayed type of transmission may sometimes occur [70]. Furthermore, observations in Oregon in 1935 showed that the number of infected plants growing from seed saved from an infected crop, decreased with the age of the planted seed, seeds 3 weeks old producing 11 mosaic plants out of a total of 168, while those aged 9 months produced only 5 diseased out of 677 plants, so that the tendency to transmit infection by seed appears to lessen with the age of the seed [58]. But it is obvious that, especially with breeding experiments for the search of varieties resistant to the disease, the possibility of transmission by seed should not be overlooked [40].

Various factors appear to influence the resistance of tomato plants to infection. Soil conditions controlling the effective functioning and development of the rooting system, texture of the soil, water supply, the adequacy and balance of mineral nutrients, are all important factors in this connection. Experiments on the variety Potentate, inoculated in the glasshouse with the virus of tomato mosaic, showed the symptoms to be delayed where growth was retarded by the application of lime and potash, and severe foliar mottle was confined to the most vigorous plants grown without addition of lime [69, 70].

For the control of tomato mosaic in commercial nurseries, the use of virus-free seed from virus-free plants is essential. All tomato debris in and on the soil should be collected and burnt. Should early symptoms show in the seedling beds, affected plants together with those on each side of them should be rogued out ; it is of little use to rogue out older plants as the virus has probably already been transmitted [10]. Soil sterilisation with steam or formaldehyde has been found to reduce the risk of infection, but infection is apt to recur later in the life of the plants. A great deal can be done to avoid the spread of the trouble if careful attention is paid to cultivation so as to induce even growth, and the maintenance of the soil in a moist condition to prevent any check to growth [80]. Strict hygienic

observances, by avoidance of tobacco, and washing of the hands after pruning in each house, should go a long way towards the raising of healthy plants [10].

Tomato Spotted Wilt

This virus disease of the tomato has an unusually wide range of hosts, including not only numerous members of the *Solanaceae*, e.g. tobacco, capsicum, datura, hyoscyamus, petunia, physalis, schizanthus and others, but of plants from such diverse orders as the *Papaveraceae*, *Leguminosae*, *Tropaeolaceae*, and *Compositae* [13]. Amongst the *Compositae* are well-known ornamentals such as aster, dahlia, chrysanthemum, zinnia, and cineraria. Though the disease is of comparatively recent appearance in Britain, it is undoubtedly on the increase in English nurseries, one reason, at least, being the ease with which tomato plants become infected in nurseries and glasshouses where susceptible ornamentals have harboured the virus over the winter. It has also been observed on tomato plants grown out-of-doors. The disease is widely distributed in those parts of the United States enjoying mild winters [44, 45], in France [41], and in the coastal districts of New South Wales [56] and New Zealand, where it is known as 'stripe' or 'brown top' [36].

Tomato plants may become infected by the virus of spotted wilt in the seed bed or after setting out, in various stages of early or late maturity. The rapidity with which the symptoms appear depends on the growth rate, young plants being much more prone to infection than older ones. The symptoms are characteristic (Fig. 322), though reported to vary somewhat owing, presumably, to climatic and environmental differences [28]. Early symptoms on the young leaves consist of a thickening of the veins and the development of spots or concentric rings of a brown colour. A striking feature is a bronzing of the lamina. Then follows a downward curling of the leaves, accompanied by a decided check to growth. If



FIG. 322.—Spotted wilt of tomato (photo by Bewley). Inset, the symptoms on the fruit (photo by Foister & Noble)

infection has occurred early in the life of the plant, the bronzing effect may change into a severe necrosis and the plant dies. In some cases, despite the check to growth, affected plants may recover somewhat if growth conditions are good, by making an appreciable amount of secondary growth. But a mottling and deformity of the foliage is still evident, the mottling consisting of small, angular, pale green spots, either few and scattered especially towards the tips of the leaflets, or very numerous and in places joining together to form indefinite yellowish-green areas accompanied again by leaf distortion. Bronzing of the lamina may also occur on the secondary growth, especially in the field. A mottling of the fruit of affected plants may also be observed, but is not common (Fig. 322 inset); sometimes mottled fruits may be harvested from plants showing no other definite symptoms, and diseased plants may be found bearing fruits that are not mottled. Blemishes on the fruit are of variable shape and character. They may consist of concentric patterns, or pale areas of red or yellow, rarely white, or sometimes the fruit may be yellow all over except for a few specks of natural red still remaining [62, 75].

The virus of tomato spotted wilt bears the following synonyms: *Tomato spotted wilt* (Samuel *et al.* 1930); *T.S.W.* (Pethybridge, Ainsworth *et al.* 1934); *Tomato virus* (Azevedo 1936, *fide* Costa & Forster 1941); *Tomato virus 1* (J. Johnson classification); *Lycopersicum virus 3* (Smith); *Lethum australiense* var. *typicum* Holmes *H*; *Pineapple yellow spot virus* (Illingworth 1931, Linford 1932); *Pineapple side rot virus* (Sideris 1927); *Ananas virus 1* (K.M.S.); there are numerous strains or related viruses, e.g. *Kromnek virus* (Moore 1933).

The virus is sap-transmissible, and useful indicator plants are *Datura stramonium*, *Hyoscyamus niger*, and *Petunia hybrida*, the lesions on the last named consisting of local circular spots with a reddish-brown margin around a paler centre. Infection with the virus permeates the entire tomato plant. Gentle rubbing of the inoculum (obtained in the juice expressed from leaves, stems, roots, and green fruits) on the healthy leaf (with a piece of muslin or a glass spatula dipped in the juice, merely so as to break the epidermal hairs, without injury to the mesophyll tissues) has proved sufficient to infect a young plant. The virus disappears from the ripe fruits, and since it has been proved to be so sensitive to heat and ageing, its absence is suggested to be due to chemical changes associated with ripening [13]. The virus is not seed-borne. It is transmitted by various insects, notably species of thrips, e.g. *Franklinella insularis* and *Thrips tabaci*. Experiments in Australia [13] showed that adults of the vector, fed for 4 days on a spotted wilt diseased tomato plant, failed entirely to infect healthy tomato seedlings on which they fed for 9 days. When, however, the progeny of these thrips (hatched in a breeding globe) were deposited as larvae to feed on a diseased plant, and transferred after they had emerged as adults to healthy tomato seedlings on which they fed for 7 days, all the experimental plants became infected with spotted wilt. That the virus of spotted wilt must first be picked up by the larva and not by the adult form of the vector shows that a peculiar relationship, as yet undetermined, seems to exist between vector and virus.

The effects of the virus in the plant is to disorganise and reduce the efficiency of the photosynthetic apparatus. Chlorosis and necrosis appear to be pronounced

in plants receiving potassic and phosphoric fertilisers, when exposed to sunlight, these features following presumably as the result of oxidation processes culminating in the precipitation of phenolic compounds. In plants receiving nitrogen, however, the necrotic symptoms were not apparent, the virus under this treatment tending to become systemic [41, 47]. Further, experiments on Marglobe tomatoes in Australia showed the disease to be associated with a reduction in the dry weight, leaf development, and water content of affected plants [47]. Infection is heavier if temperatures have been somewhat higher for a few days prior to actual infection, and less if lower temperatures have prevailed for a similar period, but in general prevailing high temperatures appear to depress the rate of infection. The period of development of the disease is shortest of all in very young vigorous plants, the normal period of ' incubation ' in a rapidly growing plant being about 12 days, increasing towards maturity of the plant [14].

As tomato spotted wilt has such a wide range of hosts amongst ornamental plants, tomatoes should not be raised in a house along with, say, chrysanthemums or arum lilies, both of which are highly susceptible to the virus, and even after the removal of these hosts, rigorous disinfection of the house should be undertaken in order to destroy lurking insect vectors. The control of the disease out-of-doors, however, is a much more difficult matter and hardly feasible. There are numerous reports of the efficacy of tartar emetic being employed as a spray on young plants in the cotyledonary stage of growth, the operation being repeated every week until the seedlings are ready for planting out ; in the early spraying the chemical is used at the rate of 1 oz., plus 4 oz. brown sugar, in 4 gallons of water, the amount of tartar emetic being doubled for the outdoor plantings [56]; others, however, have not observed any appreciable beneficial effects to follow upon this treatment [11, 39].

Affected plants should be rogued as soon as the symptoms can be detected. Tomato plants showing varietal differences in susceptibility to spotted wilt show these differences to a lesser degree when small and of rapid growth, but to a somewhat greater extent when the plants are in fruit. There is some evidence that the red currant tomato variety *Lycopersicum pimpinellifolium* shows resistance to infection from the virus of tomato spotted wilt [62].

Tomato Streak

This virus disease of the tomato (Fig. 323), also commonly referred to as ' *single virus streak* ' or ' *glasshouse streak* ', is frequently serious in commercial crops in Britain, where tomatoes are forced for the early market [52]. It is also prevalent in Eire, widespread in Holland, and occurs in localised areas in the United States, Canada, and New Zealand [4, 9, 37, 60, 85].

The symptoms are highly variable and at times are hardly to be distinguished from those of the ordinary mosaic of tomato (p. 680). This variability appears to be dependent on a variety of factors affecting the conditions of growth, such as those which are conducive to soft vigorous growth, or fairly high temperatures and relatively low light intensity, excessive application of nitrogenous fertilisers,

or excessive moisture, deficiency of potash, etc. Apparently under the influence of these changing conditions four types of symptoms are manifest (Fig. 323):

(*a*) Those resembling the symptoms of leaf mosaic only, that is, a mottling characteristic of the ordinary mosaic described above (p. 680).

(*b*) Those with leaf mottling again, but accompanied by necrosis of the stem and petioles in the form of dark-coloured longitudinal streaks or stripes, the most serious phase.

(*c*) Those showing only the latter features in (*b*), that is, necrosis of stem and petioles; no mosaic symptoms.

(*d*) Those showing, at first, symptoms of mosaic only, or necrosis only, but subsequently developing the other type of symptoms as well [52].

The following synonyms are listed for this virus (*Lycopersicum virus 1* Bewley): *Tomato stripe*, *streak*, or *glasshouse virus* (Jarrett 1930); *Tomato streak virus No. 1* (Ainsworth *et al.* 1934); *Single virus streak* (Ainsworth, Berkeley, Caldwell 1934); *Tomato virus 4* (J. Johnson classification); *Severe mottle streak virus* (Selman 1941); *Mild-mottle streak virus* (Selman 1941); *Tomato streak virus, yellow and green strains* (K.M.S. 1935); *Tomato streak virus 1 Ontario strain* (Berkeley 1936). The virus appears to include a number of strains [1, 19, 79].



FIG. 323.—Tomato streak (photo by McKay)

Owing to the close similarity of the symptoms of 'streak' with those of the tomato (tobacco) mosaic, and in view of the fact that the physical properties of the two viruses are practically identical, the virus of 'streak' disease is believed to be a strain of the tobacco mosaic virus [52].

The virus of tomato streak is sap-transmissible and although the means of spread is not fully known, the vector *Macrosiphum gei* is said to carry the greatest amount of infection, with *Myzus persicae* and *M. pseudosolani* to a lesser degree, in America [51]. The virus is more active and remains virulent for a longer period in heavy clay and humus, and least in sandy soils. Much infection is believed to take place through the medium of the soil in nature, especially soon after planting, and young seedlings may fall early prey when their young leaves come into contact with the soil, but the artificial introduction of the virus into the roots appears to be attended with difficulty [85]. In a few trials transmission

by seed has been effected, in one case [85] to the extent of about 1 per cent., and in another [16] to 66·6 per cent. infection.

Another virus affection of the tomato, closely resembling '*streak*', known as '*mixed virus streak*', is, as the name implies, a composite disease caused by two distinct viruses. The two components are those of the ordinary tomato (tobacco) mosaic and potato mosaic (virus *X*). The disease may be found in tomato crops which are grown near to or which follow potatoes. As it is the result of a mixed infection with these two viruses, a passage of the mixture through tobacco filters out the tobacco strain virus from the mixture [1]. The symptoms of mixed virus streak are identical with those of streak, except on the fruit, those of the mixed virus on the latter causing small, slightly raised brown spots on the skin, while those of streak are distinguished by the spots being larger and sunken.

For the control of these diseases, all hygienic practices as already indicated for other tomato diseases in the glasshouse should be observed, and no seed should be saved from affected plants. To counteract soft growth, a balanced manurial treatment is recommended. Infection from mixed virus streak will be lessened if the tomato crop is planted away from potatoes, and all 'ground' tubers should be cleared out of the soil [10].

Tomato Yellow Mosaic

Aucuba mosaic of the tomato, preferably called yellow or severe mosaic (as the term 'aucuba' is already in use with one of the virus diseases of the potato), is common in Britain where it sometimes accounts for severe losses. It is also recorded in Eire, Denmark, Holland, Australia, and New Zealand [29, 35, 38, 61, 73, 74, 78].

Diagnostic symptoms of yellow mosaic, in general, bear close resemblance to those of ordinary mosaic described above (p. 680), and indeed the disease is caused by a strain of the same virus [55]. But the symptom-picture of yellow mosaic is much more brilliant and striking than that of ordinary mosaic; the white areas are more intensely white, the green areas more vividly contrasted, and there is usually a sharper delimitation between the two [74].

Yellow mosaic does not usually destroy its host, and fruit formation is not inhibited. There is a decided check to growth as compared with normal plants of the same age, and affected plants tend to develop in a spindly manner (Fig. 324). The fruit



FIG. 324.—Tomato yellow (aucuba) mosaic (photo by McKay)

may or may not be mottled, though silvered areas may sometimes be present on the skin [10]. The general symptoms vary somewhat according to the variety, and under fluctuating conditions of the environment, especially of temperature and light intensity. Observations on inoculated plants have shown that first symptoms appear on the very young, developing leaves of the shoot, about the fifth day after inoculation, the plants then showing only three leaves large enough for inoculation. These early symptoms consist of a downward curling of the entire leaf, with a slight turning-down of the margins, and the surface is rough and wrinkled, but as yet, with no signs of chlorosis. The latter begins to appear on the seventh or eighth day, in the form of small spots on the curled leaves, sometimes at the base, more normally at the tips and margins of the leaflets. Later, the spots increase in number over the whole surface of the leaf and tend to join together. By about the twelfth or thirteenth day when six or more leaves have appeared, the symptoms differ in the different leaves. The original three leaves may still show no chlorosis, or only a slight yellowing of the veins; the youngest leaf may also be quite green. But the leaves which developed after inoculation, the fourth, fifth, and sixth, are extensively affected. In extreme cases almost the whole surface is pale-yellow to white, with, here and there, small islets of intense dark green, resembling small blisters. In less extreme cases the green areas are larger, but as a rule the area of white or pale-yellow is greater than the green area. The three original leaves show the disease in its most extreme form; the younger leaves at this time show only scattered patches of white or yellow, frequently angular or triangular at vein intersections. When the first flowers are in bud, a typical leaf will show most of the surface green, partly of normal tint and partly of a deeper and richer shade, but scattered over the leaf are patches of white, and others of yellow, usually sharply delimited, but sometimes shading into neighbouring areas. At other times the patches are irregular in shape and size, often angular, and occurring in all parts of the leaf. There is no necrosis and bronzing does not occur [74].

Synonyms for this virus (*Nicotiana virus 1c* Bewley) are: *Tobacco virus 6* J. Johnson 1937 *fide* Ainsworth *et al.* 1934; *Tomato yellow mosaic virus* (Ainsworth *et al.* 1934 [86]); *Tomato aucuba mosaic virus* (Bewley 1923, J. H. Smith 1928) [77].

The virus is spread in the same way as that of mild mosaic; it is not known to what extent, if any, insects may transmit the virus [35]. Both the viruses of mild mosaic and yellow mosaic have been 'cultured' in excised root tips of tomato. While the infected roots showed no external symptoms of disease, the virus was strongly retained within the tissues and did not escape into the medium, nor was it possible to transmit infection into healthy roots by contact with diseased roots [82].

There is abundant evidence that mottling develops only in leaves which were young at the time of infection, and while older leaves may show a certain amount of yellowing, due possibly to senescence of plastids in the discoloured areas, experiments have shown that the distinctive mottling of yellow mosaic never appears in fully developed leaves at the time of inoculation. An examination of the cells of the mosaic areas in young leaves revealed them to be practically devoid of chloroplasts, and as the mottling is present only in those leaves which were in active growth at the time of infection, it is believed that the virus is responsible

not so much for the actual destruction of chloroplasts already mature, of which there was little or no evidence, as for the inhibition to the full development of plastids from their primordia [72]. That the presence of the virus in the young tissues prevents the plastid-primordia (preplastids) from attaining their full expression is no doubt one reason why young seedlings lose weight; experiments have shown that the dry weight of infected seedlings was estimated at 7 to 8 per cent., while, following inoculation when they were in their fifth normal leaf, it was about 10 to 12 per cent., that of control seedlings being 14 to 15 per cent.: moreover, the content of carbohydrate in these experiments was in the proportion of 1·1, 1·3, and 1·6 per cent. respectively, the nitrogen content being not materially affected [31, 32]. During the metabolic processes in leaves affected with yellow mosaic there appears to be a lag in the conversion of reducing into non-reducing sugars, and while the mean concentration of reducing sugars in healthy and diseased leaves showed little difference, the amount of non-reducing sugars was much greater in the former [83, 84].

The presence of the virus of yellow mosaic in the plant also affects the normal differentiation between palisade and spongy cells in the leaf mesophyll. In fact the differentiation tends to disappear, the palisade cells becoming shorter than the normal, and with the reduction of the intercellular spaces in the spongy mesophyll all the cells tend to be alike [72]. The virus is much more abundant in the chlorotic spots than in the green areas of the mottled young leaves, which seems to indicate that the multiplication of the virus is not uniform throughout the tissues of the host [31, 32]. Moreover, the cells of the yellowed spots often seem to contain more than the normal amount of cytoplasm, and while in the healthy leaf the plastids may occasionally show a tendency to swell, this has been found to increase in diseased plants, the plastids becoming so swollen with starch as actually to fill the cell. As in the case of tomato plants infected with the virus of mild mosaic, various types of so-called 'inclusion bodies' have also been found in connection with the virus of yellow mosaic, and are believed to be of a protein nature. As many as four different types of inclusion bodies have been detected in the leaf hairs of young tomato plants infected with the virus of yellow mosaic, but all were found to be fundamentally identical [46]. They are, however, not confined to the chlorotic areas of the leaf, being formed in both green and yellowed tissues; they do not appear to be developed until after the plastids have been organised. Briefly, the effect of the virus in the young host cell is to check its growth and upset the normal development of plastid primordia. If plastid formation is not inhibited at an early stage, normal development is proceeded with, and the now organised plastids will persist even if the virus reaches them later [72].

For the control of yellow mosaic, the same precautions should be observed as outlined above for mild mosaic.

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Downy Mildew of Spinach, *Peronospora effusa* (Grev. ex Desm.) Rabenh.

Downy mildew of spinach is everywhere common and was first discovered in England in 1824 [9]. It also occurs in the United States [1, 10, 16]. Sown in winter and spring, spinach in some localities is cultivated as a green crop all the year round, and, since infection is possible at any time, losses from mildew are often very high, and entire crops may be lost within a few days.

Early signs of mildew occur on the outer leaves as pale-yellow spots of irregular shape increasing in area in wet weather, while isolated spots appear on the heart leaves (Fig. 325). Under damp conditions the discoloured areas soon develop the downy-white, later, greyish-purple conidial fructifications of the fungus *Peronospora effusa* [9, 15], causing this disease. While these may be found chiefly on the under side of the leaf, the upper side may also bear them if the weather is continuously wet. The petioles may also be yellowed and shortened and the plants in general have a stunted, wilted appearance.

This obligate parasite attacks no other plant, and recent evidence has shown that the once suspected *Chenopodium album* is not a carrier host for *P. effusa* attacking spinach [4]. The non-septate mycelium branches profusely between the mesophyll cells of the leaves and the parenchyma of petioles and stems but does not occupy the vascular bundles [16]; there is no systemic invasion of the seed-bearing branches [14]. The conidiophores (Fig. 326), at first white, later greyish purple, 200 to 300 μ long, emerge from the leaves through the stomata singly or in fascicles, branching two or three times from the main stalk



FIG. 325.—Mildew of spinach (*Peronospora effusa*). A young plant severely infected with mildew (photo by Richards, *Cornell Univ. Ext. Bull.* 718)

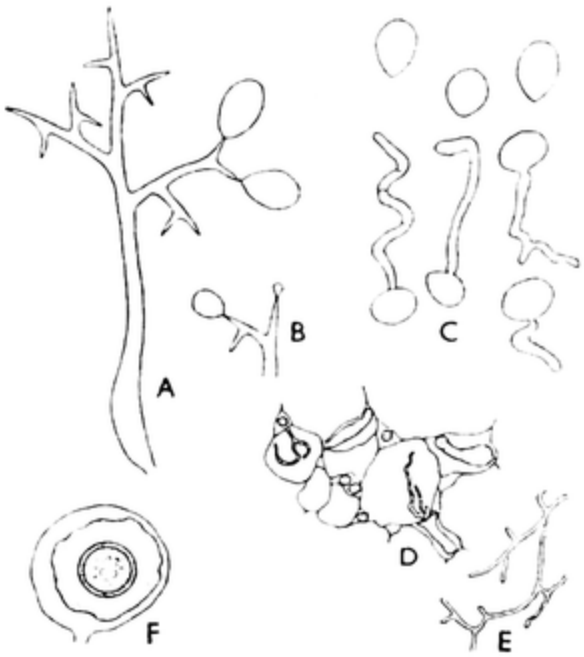


FIG. 326.—*Peronospora effusa*. *A*, conidiophore and conidia. *B*, formation of conidium. *C*, conidia and their germination. *D*, leaf tissue showing wide mycelium and narrow haustoria. *E*, the mycelium. *F*, oospore within the oogonium (after Richards, *Cornell Univ. Ext. Bull.* 718)

before they dichotomise to form a number of sharp-pointed sterigmata bearing ovoid conidia 17·0 to 26·0 by 22·0 to 38·0μ (average, 20·6 by 29·9μ). The oogonia, in living leaves, are spherical, 40·0 to 55·5μ in diameter; the antheridia are clavate. The oospores are spherical, thick-walled, deep yellow, 35·7 to 42·5μ (average, 36·8μ in diameter); they are infrequent or absent in some localities ([7, 8, 12]), and their rôle in nature has not been ascertained.

The mycelium of *P. effusa* has been found on and within the seed [2, 14]. The oospores have also been discovered on commercial samples of seed [5] and there is some evidence [3] that the disease is transmitted by seed, but nothing is known about the method of seed infection. The disease is not carried in the soil. The most likely source of carry-over is from mycelium hibernating in host tissues, and diseased spinach plants may be found practically all the year round [11, 13, 16]. In plants infected in the autumn, the fungus which had survived in the leaves over winter revives in the spring to produce abundant conidia, and secondary attacks follow. Leaf penetration by germinating conidia is direct, between epidermal cells [16]. Both sides of a leaf may be entered, and older leaves are apparently more susceptible than those near the top, presumably because they are surrounded by more humid conditions. The optimum temperature for the germination of conidia is from 8° to 10° C., the minimum 2°, and the maximum between 27° and 30° C. [5, 11, 16].

Downy mildew of spinach occurs over a wide range of temperature if atmospheric humidity is 85 per cent. or more. Acid soil conditions and high temperatures aggravate the disease [6, 16].

Little is known about the fate of the oospores of *P. effusa*. Their possible hibernation in the soil indicates the advisability of observing crop rotation as one means of controlling this disease, but no evidence is to hand as to the longevity of the oospores in the soil. If spinach is cultivated all round the year, winter and spring plantings should be grown apart. All infected plants should be lifted and burned and affected leaves should be removed as soon as they show signs of infection [8a]. No commercial varieties of spinach are known so far to be resistant to this mildew [16].

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Downy Mildew of Onion, *Peronospora destructor* (Berk.) Casp.

Downy mildew attacks every variety of onion and occurs also on shallots, garlic, and occasionally leeks. First recorded in England by Berkeley [3] in 1841, and in America in 1884 [21], the disease received its first scientific investigation in 1887 at the hands of the zoologist Shipley [17], who was sent to Bermuda by the Kew authorities to enquire into a serious epidemic of the onion attributed at the time to an insect pest but proved by him to be the work of the fungus *Peronospora destructor* [2, 18, 19, 22, 23].

The host plant may be attacked at all stages of growth; seedlings in the frames may succumb early, and when older plants are attacked in the open about July and August the withering of the foliage prevents the normal development of the bulbs, which remain small and deformed. The bulbs keep badly, turn soft in storage, and sprout prematurely [11, 15]. It is not easy to recognise lightly infected bulbs in storage or at transplanting, but the outer scales and sometimes the living scales next to them are frequently rough or puckered, and show watery, or brownish, or amber-coloured spots on the surface. The inner scales may not show any of these signs externally yet they are often found to harbour mycelium. Bulbs lightly infected may show no ill effects at all, and it is evident that they can tolerate infection for a long time without loss of turgor.

Briefly, the cycle of onion mildew in Britain may be taken to start on autumn-sown plants which become infected from wind-borne spores conveyed from a near-by diseased summer crop. Fresh infections of healthy plants begin on the leaves, from which they quickly spread into the other organs. All parts of the plant except the roots may be invaded [13]. The fungus may occupy the stem (Fig. 329), bulb scales, leaves, all the floral organs, and floral receptacle [4, 15]; its occurrence in the seed has been recorded only in very few instances, and there is no definite evidence that the seeds serve as carriers of infection. The mycelium remains dormant in the bulb until the spring when it revives again, growing up from the bulb into the developing leaves and finally sending out its conidiophores and spores through the stomata into the air for further dissemination of the disease to healthy plants.

The disease may, therefore, be described in two stages: (*a*) the primary, when an infected bulb is planted; and (*b*) the secondary, when a healthy plant, in leaf, becomes infected from spores produced by the primary stage.

Primary Stage

The appearance of the foliage leaves which grow from bulbs already infected with the hibernating mycelium is very characteristic. One or more of the leaves, or even the whole plant, appears yellow and glazed, as if varnished; all parts of the plant are, however, quite turgid, and there are no signs of any collapse of the tissues despite the presence of abundant mycelium within. The leaves may be packed with mycelium and yet show no external sporulation as long as the atmosphere remains dry. But in a moist environment infected leaves gradually collapse and wither from the tips down. Meanwhile the fungus in the leaves breaks out to sporulate (Fig. 327 A) and the conidiophores are pushed out through the stomata, laden with numerous conidia which are dispersed by wind to start secondary infections. Thus the planting of affected bulbs is, no doubt, the chief means whereby onion mildew is spread. The fungus keeps pace with the growth of the shoot, entering the leaves, the inflorescence axis, flower stalks, and in fact any part of the flower. Infections of the flowering stem, or of pedicels, may be so severe as to completely girdle them with lesions, causing the blossom to collapse before setting seed, and accounting for considerable reduction in yield of seed [24]. It is, however, rather unusual for the flowering shoots to become so infected as to

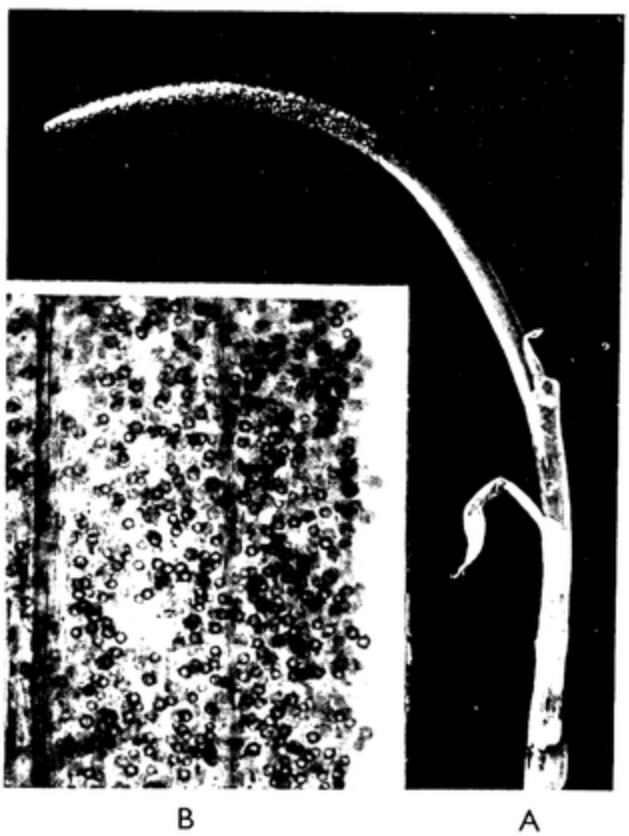


FIG. 327.—Downy mildew of onion (*Peronospora destructor*). *A*, the plant covered with conidial fructifications. *B*, oospores in the leaf tissue (× 45) (photo by McKay, *J. Roy. Hort. Soc.*)



FIG. 328.—*Peronospora destructor*. *A*, oospore within oogonium. *B*, germination of a thin-walled oospore (both × 450) (photos by McKay, *J. Roy. Hort. Soc.*)

interfere with the normal development of the seed, but in certain areas in England the mildew is troublesome on the seed crop [11 a]. There is evidence that the fungus may enter not only the wall of the ovary but the ovules as well and may, therefore, become established in the seed [5, 20], but many have failed to confirm the presence of the parasite in the seeds [6, 7, 9, 12]. Though the young embryo may not actually become invaded, it is not improbable that seedlings might sometimes get infected during the early stages of germination; it is a common observation that onion seedlings often retain the seed coat attached to the suctorial cotyledon for a long time, and may thus pick up infection from the testa [5].

Secondary Stage

In a series of experiments [11] conducted on seedling onions of the Ailsa Craig variety, from the time of depositing the conidia on the leaves, the first discoloured spots denoting successful infections appeared in about 14 days. Penetration by the conidial germ-tube is always stomatal. First signs of disease consist of light-coloured spots on the leaves, later turning yellow and then white, at which time they are somewhat sunken from collapse of the tissues. If wet conditions prevail the spots increase in size and number, and the green foliage develops a white, flecked or streaky appearance. The affected leaves finally lose turgidity, collapse and shrivel, becoming very thin and grey. The mildew then breaks out at the affected spots to form a purplish growth, the coloration being present both in the conidia and conidiophores [6]. In about 23 days from the time of initial infection, the fungus, travelling quickly between the cells of the leaf, chiefly downwards from the point of infection, reaches the tissues of the bulb. In the bulb the fungus collects mainly towards the upper surface of the short stem from which it invades the developing leaves, and may even enter the delicate leaf-primordia at the centre [13, 14]. There is no clear evidence that the fungus can bridge across from one scale leaf to another in the bulb, and all attempts to set up infection by external inoculations of the bulbs have failed [11]. The intercellular hyphae are entirely without cross walls and vary considerably in width according to the spaces in which they are confined. Haustoria are formed in abundance, and consist chiefly of simple, narrow tubes, but sometimes branched specimens also occur (Fig. 329 C, D). This is the condition of the fungus in the bulbs at the time of lifting, and as far as the alternation of primary and secondary infections usually go, the cycle of infection is complete, but there is still to discuss the sexual phase of the fungus and the possible rôle of the resting spores.

The conidiophores of *P. destructor* consist of stout unbranched tubes which, however, soon divide into four or more outspreading branches, the final numerous ramifications terminating in a pair of short sterigmata each bearing a lemon-shaped conidium. The conidia range from 43·5 to 69·0 by 22·5 to 30 μ (Fig. 329 F, J) [13]. Conidial production occurs on an immense scale, and it is stated that, after wiping off these spores from the surface of a leaf, as many as three times as they were renewed, new crops of conidia were regenerated within 48 hours. But their viability is very brief, and they hardly survive removal from the leaf unless collected early in the morning before the dew has dried [5, 16]; in dry air, light appears to kill them, but they can withstand exposure of several hours

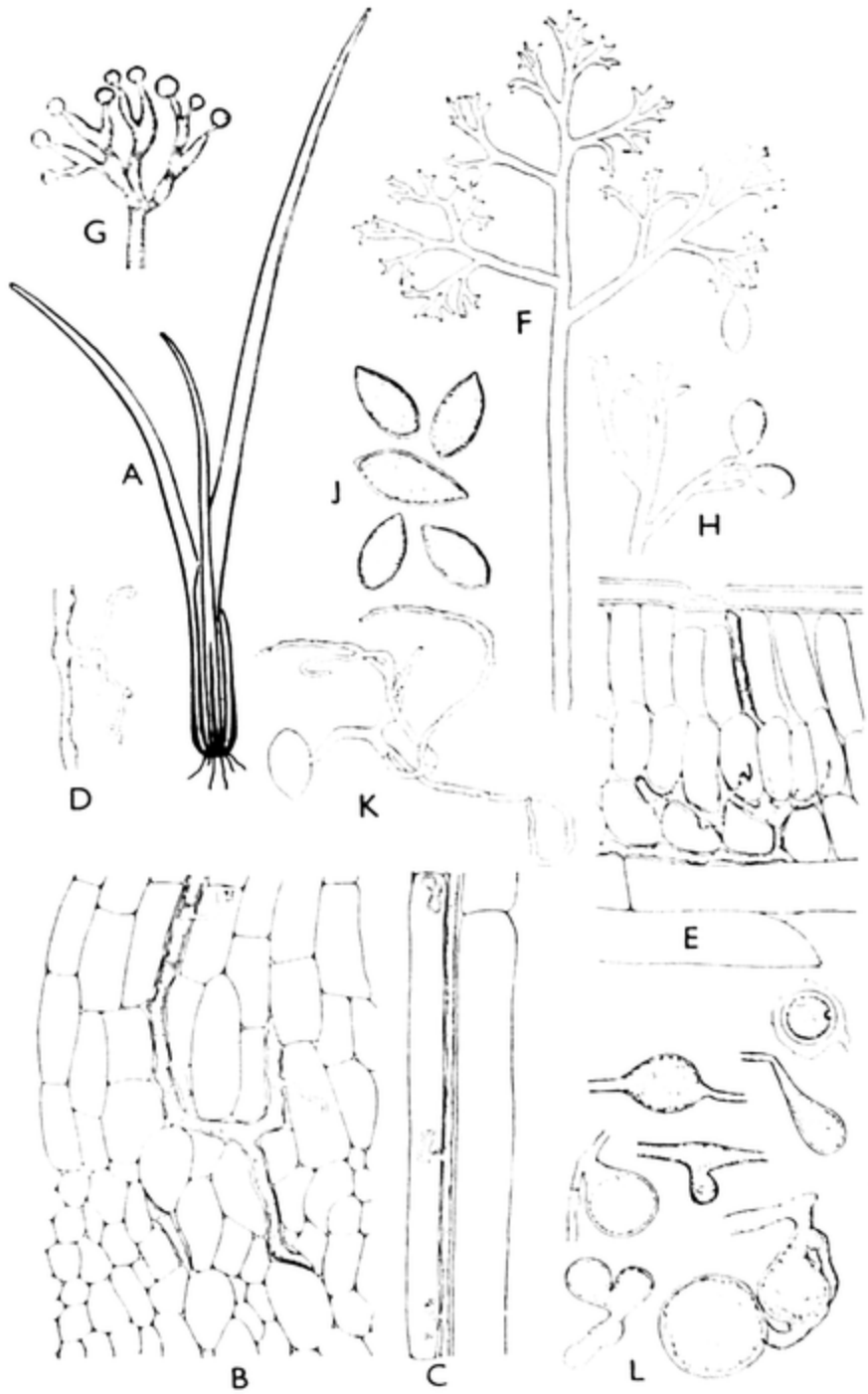


FIG. 329.—*Peronospora destructor*. *A*, diagram showing distribution of the fungus (heavy line) in a seedling onion, 23 days after infection. *B*, mycelium in meristematic tissue of the root-stock (× 170). *C*, mycelium growing up from root-stock into base of young leaf; note haustoria (× 170). *D*, a trifurcate haustorium, from leaf. *E*, invasion of leaf mesophyll prior to formation of conidiophores (× 104). *F*, conidiophore (× 102). *G*, *H*, development of conidia (× 170). *J*, conidia (× 170). *K*, germination of a conidium in soil-extract (× 170). *L*, development of oogonia and antheridia; top right, oogonium containing an oospore (× 170) (after Murphy & McKay, *Sci. Proc. Roy. Dub. Soc.*)

to bright sunshine provided the air is not too dry or too warm. With favourable conditions of humidity, conidia may continue to be formed on the host over a range of temperature from 13° to 18° C.; at 10° and 20° production is not plentiful, and below 6° or above 25° C. they are very sparsely developed; the optimum temperature for their germination is about 10° C., the minimum being below 5°, and the maximum about 28° C.[7, 9, 13].

In addition to conidia, *P. destructor* also produces thick-walled oospores, fertilisation of the oogonia by antheridia, presumably, taking place in the usual manner of the *Peronosporaceae*; the oospores, at first colourless, but later red, are round, and measure from 17 to 37 μ in diameter (Figs. 328, 329 L). They are usually found in the leaves and flowering shoots, and in a few instances have been discovered in the walls of the capsules and adhering to the seeds[5]. They are most typically found in parallel rows in the leaves, developing mostly alongside the long veins (Fig. 327 B). The oospores are difficult to germinate[10], apparently because of the thickness of the wall (3 to 4 μ), but it appears that the presence of oxygen is an important factor in their germination, and the addition of a weak solution of potassium permanganate (0·01 to 0·02 per cent.) at 20° C. greatly stimulated germ-tube production (Fig. 328 B). The germ-tubes are simple or branched, and sometimes form bodies resembling chlamydospores, 21 to 31 by 12 to 23 μ, but such bodies have not been found under natural conditions. Under no further trials, however, could the oospores be induced to make progressive growth, and it is doubtful whether they play any part in the spread of the disease[11]. Though there are no proofs that the oospores are capable of setting up infections, the possibility must not be ruled out, if the right conditions in nature are found, that they may establish scattered foci of infection in a crop. In certain parts of North America where it is not the custom to allow the plants to over-winter, it is believed that in the absence of a carrier crop, the oospores in the soil are probably a source of primary infections[5]. The presence of the fungus in the floral organs is confirmed by numerous authors[5, 7, 11, 12], and despite the fact that only in a few instances have the ovules or seeds been found to harbour mycelium, the opinion appears to prevail that seed infection may still occur. The oospores have been found even in the capsules in diseased flowers, but from the few seeds that were obtained and sown no infection resulted[7]; it is suggested, however, that oospores adherent to the testa may sometimes be a source of infection at germination[5].

While prolonged rainy or muggy periods favour the spread of onion mildew, conidia never being formed when the leaves are dry, the incidence of dew is a much more important factor in the production of conidia than actual rain[11, 13]. Though the fungus develops its conidial fructifications over a wide range of temperature, it is, on the whole, a low-temperature organism and the conidia appear generally when night temperatures are between 5° and 13° C.[5].

There is some evidence that physiologic races of the parasite of onion mildew exist. The fungus isolated from the variety Welsh Onion infected *Allium fistulosum* var. *caespitosum* and common onions, but no other variety[7]. Breeding experiments conducted in California[8] indicated a localised resistance in various parts of the host. Thus a certain selection of the Italian Red variety showed the seed stalks to be immune and the foliage highly resistant; another strain of the same variety while retaining similar immunity had only slightly resistant foliage, and seed stalk immunity was also established in the progeny of hybrid forms. Again, some varieties of onion may be resistant in the foliage but not in the bulb; thus

Cranston's Excelsior is resistant only in foliage whereas Up-to-Date is resistant both in the leaves and in the bulbs [15].

Since the perennating mycelium in the bulbs appears to be the chief source for the spread of this disease, it is obviously important to examine the plants carefully when transplanting and during the growing season, and any showing signs of the disease, such as softness of bulb or a yellowing of the young foliage, should be rejected or removed. Such plants must be destroyed by burning and not left on the ground or taken to the compost heap. As the parasite is tolerant only of rather low temperatures, dry heating affected bulbs—to 40°–45° C. for 8 hours or more, according to the size, number, and variety of the bulbs—is effective in killing the mycelium [13, 15]; whilst this temperature may not be harmful to the bulbs, the method is, however, risky in practice and it is doubtful whether the deeper-seated fungus is rendered innocuous by this treatment.

All spraying operations against onion mildew are very difficult to perform successfully because of the smooth, shiny surface of the leaves, but with an adhesive such as resin fish-oil soap, Bordeaux mixture applied early and repeatedly has been found beneficial; and dusting the plants when wet, either with finely powdered sulphur alone or mixed with half its weight of powdered lime, sometimes gives good results [1]. As a substance toxic to the conidia, malachite green was found to be more effective than copper sulphate, and a dilution up to 1 in 150,000 of water completely stopped the growth of the conidia, but few details are, so far, available as to its practical uses as a fungicide [16].

If possible, new plantings of onions should be made in soil not previously given to these plants, in order to avoid any possibility of the hibernating oospores restarting the disease. Since autumn-sown onions in proximity to the summer crop are a proved source of danger, autumn sowing should either be discontinued, or the plants raised under glass in December or January. If the crop must be sown in the open, this should be done as late as possible in the autumn, and the sowing of seeds for the spring crop should be made as far removed as possible from the autumn crop.

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Onion Smut, *Urocystis cepulae* Frost

Smut disease occurs on all varieties of onions, leeks, shallots, chives, and garlic. It is common in the British Isles [1, 2, 7], and in many areas of the northern States of America, where it is believed to have originated in 1860, probably on some wild species of onion [5, 13, 17]. The disease appeared in 1879 in France and Germany, and is now fairly widely distributed throughout Europe ; it was not recorded outside Europe and America until 1938 when it was reported first in New Zealand, and later, within restricted areas, in Queensland, and is believed to have been introduced into these areas with imported onions [10, 12].

The disease attacks only seedlings and young plants, for after passing a certain stage of development the host becomes immune. Soon after the seedlings appear above ground, infected specimens usually show one or more long silvery streaks below the epidermis of the cotyledon and sometimes of the succeeding leaves as well, the latter bending over, in marked contrast to the leaves of healthy plants, which remain erect ; affected leaves are also somewhat thicker than normal leaves (Fig. 330). When the silvery epidermis over the blisters is broken, the lesions become sooty black from the release of dense masses of dark-coloured spores (' brand ' spores) from the disorganised leaf tissues below. Within 3 to 5 weeks after germination, if infections have been heavy, mortality of seedlings may be very high. According to the degree of infection some seedlings may survive longer, but if new leaves become attacked as they develop from the growing point, the stripes, and in turn the blisters, break out on them anew. Sometimes, by vigorous growth, an infected plant may outpace the disease, and by casting off the infected cotyledon produce a healthy bulb. Other lightly infected bulbs may reach harvest whilst still retaining infection in one or more of the outer scales and, unsuspected of harbouring the fungus, may be marketed as healthy produce. In most cases, however, the disease keeps pace with the growth of the plants, and, with successive leaves developing the silvery lesions, they end by developing smut blisters not only on the outer scales but on the inner scales as well, producing shrunken bulbs considerably below normal size. There is, however, no progressive rotting of smutted bulbs while



FIG. 330.—Onion smut (*Urocystis cepulae*). *A*, smut lesions on base of two seedling leeks. *B*, on the leaves (photos by Foister & Noble)

in storage, and affected specimens can usually be singled out by the characteristic silvery stripes at the base of the scales.

When the lesions burst, the spores, for the most part, fall into the soil, in which they may persist for years. Spores may also be carried by wind or insects [7, 13] and may be dispersed by any means whereby tainted soil can be conveyed during cultivation, on implements, boots, etc., or in surface drainage water, but a sure method of spreading the trouble, locally or abroad, is by the distribution of infected 'sets' for planting, or of transplants (past the susceptible stage) raised in contaminated soil [17]. The disease is not carried by the seed.

Onion smut is caused by *Urocystis cepulae*, a member of the smut fungi *Ustilaginales*. Each spore of *Urocystis* is not merely a single cell, but is furnished with a close-fitting covering of as many as 20 sterile, hyaline cells, the whole forming a spore ball having a dark cell at the core—the true, fertile spore; sometimes, but rarely, there may be two spores within a spore ball. An entire spore ball varies from 15 to 20 μ, the central spore itself being from 11 to 15 μ in diameter. The spores are germinable in water, but make better growth in onion juice, especially with the addition of a sugar carbohydrate, but with starch, or in an acid medium growth is retarded [3]. When a spore germinates, a single hypha (rarely two) is pushed out between the investing cells of the spore ball to form a short, hemispherical tube, the promycelium or basidium which soon gives rise to a whorl of septated, filamentous branches consisting of uninucleate cells. These filaments may either bear a number of sporidia arising in lateral positions, or they may further branch and anastomose to form a mycelial plate or weft without bearing any sporidia at all [3, 6, 14]. The sporidia measure, on an average, 7·4 to 3·5 μ in diameter, and may bud freely after liberation or while still attached to the hyphae, but whether they may function as oidiospores in nature is not known, nor has any evidence been obtained of the conjugation of sporidia [14]. The filamentous branches arising from the short promycelium may, instead of forming sporidia, disintegrate in the soil, so as to liberate their component cells, which again are said to bud out and function as oidiospores [17]. Upon the germination of the brand spores, under whatever conditions in the soil that may induce them to give rise, at one time, to mycelium, with or without mycelial plates, or at another time, to sporidia, or oidiospores it is clear that the organism has an appreciable period of existence in the soil, purely as a saprophyte, for how long is not known (a period of 15 years, in the absence of a susceptible host is recorded), but it appears in this capacity to be capable of considerable resistance to dessication in the soil [6].

In the presence of a susceptible host the fungus, adopting a parasitic habit, attacks the young seedlings as soon as they emerge from the soil. Infection takes place through the cotyledon at one or more points below, or on a level with the soil; the 'collar', the part at the junction of root and stem, is the region at which penetration of the cotyledon normally occurs. Parasitic hyphae have also been found within roots growing around the collar, an observation which would appear to indicate that the root hairs are the first cells to be entered [14]. The cotyledon continues to be susceptible until it reaches maturity, which occurs usually 2 to 3 weeks after germination. If the seedlings can survive that period without penetration, the mature tissues of the cotyledon form a sure protection against further attacks, and the plants grow up to produce perfectly sound bulbs. Any

means, therefore, that can be adopted to induce young seedlings to make vigorous early growth so as to hasten cotyledonary maturity, confers upon them complete immunity from onion smut. But, maturity of the tissues of the cotyledon does not mean that the true leaves ensheathed and protected by it are no longer susceptible to attack; when a number of seedlings which had passed the susceptible stage were stripped of the cotyledon and the immature tissue beneath exposed to the same infection in the soil, 60 per cent. of the plants became diseased [15]. But no danger can accrue from the planting of healthy, established transplants or 'sets' in smutted soil, for they are well past the susceptible stage, but infected plants placed in clean land are obviously a source of danger when, with their decay, the brand spores are set free again into the soil. In general, it may be stated that the period of susceptibility to attack begins about the second or third day after germination of the seed, and the plants are deemed to be immune from infection when they have grown to a height of about 3 inches above the ground [2].

At infection, the tip of the penetrating hypha bores its way directly into an epidermal cell without any preliminary expansion to form an appressorium [3, 8] and, passing in quickly between the cells of the mesophyll, proceeds to branch and keep pace with the growth of the lengthening cotyledon without apparently causing any disturbance to the host cells. If initial infection occurs comparatively late in the growth of the cotyledon, that is, when it is approaching maturity, infection hyphae may be arrested in the epidermis and become disintegrated; it is probable that the occurrence of such disorganised hyphae in some of the host cells led to the pronouncement of haustoria being present [3], but later observations have failed to establish the presence of these organs in the host [8, 14]. The hyphae within the host leaf are practically confined to the tissues lying between the vascular strands, and it does not appear that the fungus ever invades the phloem, nor are the spore beds ever laid down in the outermost layers of the mesophyll, or within the epidermis. Having established itself in the cotyledon, the fungus makes its way into the growing point of the shoot, and it is from this region that the leaf-primordia become infected. Whether the young shoot can escape infection from the cotyledon will depend, as above stated, upon the rapidity with which the young leaves develop, and if they are well advanced before the fungus reaches the growing point, they develop normally and contribute to the formation of a healthy bulb.

The complete life-cycle of onion smut, from the time of infection to the appearance of mature pustules of brand spores on the cotyledons, may be completed in from 3 to 4 weeks. In the young spore beds all the cells of the mycelium remain uninucleate for a long time, and only just prior to spore formation is a binucleate condition seen to arise at various parts in the young sorus. It is not clear how this new condition arises, except that frequent anastomoses of hyphae are evident throughout the sorus, and certain cells become binucleate, presumably as a result of migration of nuclei from contiguous cells. Finally, fertile spores are produced by some of these binucleate cells, but some of the cells abutting on them, also binucleate, become closely adpressed to the spores to form the sterile investment, or pseudospores, as they are called, which are believed to act as 'nurse cells' to the developing central spore, and they remain permanently as

a covering to the spore ball. The paired nuclei in the cells of the spore ball, in both the fertile and the sterile, ultimately fuse, so that the mature spores are diploid zygotes. It is interesting to note, according to the foregoing observations[6], that, unlike the behaviour of the infective mycelium of other investigated smut fungi, such as those of the cereals, in which the cells of the hyphae are binucleate while yet on the surface of the host, that is before penetration, the mycelium of onion smut retains its haploid condition *within the host* up to the time of the laying down of sporogenous primordia. According to these observations the behaviour of *Urocystis cepulae* would appear to be unique among the smuts, and may provide the first example of a homothallic species in this group of fungi [10a, 11].

The temperatures prevailing in the upper layers of the soil have an important bearing upon the incidence of onion smut. In general, those most suitable for infection approximate closely to temperatures which also favour the germination of the seedlings [16]. Though the margin between germination and infection may be small, under conditions in Elba, New York, this narrow interval proved sufficient to afford some measure of control over the incidence of the disease, and, by sowing the seeds a little earlier than usual, the seedlings escaped infection because the seeds germinated at a slightly lower temperature than smut usually develops, namely, at 8° and 10° C. respectively. Moreover, a prevailingly low-soil temperature of 8° to 13° C. during early germination helps to lessen the amount of infection, and if the temperature in the top inch or so of the soil remains within this low range for an appreciable time, a reduction of as much as 60 to 78 per cent. in the amount of infection may be obtained in comparison with that which occurs at the optimum range for smut development, namely from 15° to 20° C. [9]. Furthermore, in relation to both soil and air temperatures there is usually a high degree of infection at soil temperatures of 20° to 25° C., even when the air temperature is higher, as at 30° C., but a soil temperature approaching the latter figure is fatal to the parasite, showing that air temperatures alone are not sufficient to check the disease [15]. At the higher temperatures, too, the growing plants are induced to make rapid growth, and may thus outpace the fungus in the cotyledon, so that the latter is cast off before infection enters the growing point, and the leaves develop healthily [1].

Germination of the spores in the soil is increased by a free access of air, and though the degree of soil moisture is not such an important factor as relative temperature in the incidence of infection [15], an excess of water, by lowering the temperature of the soil, has the effect of retarding the growth of the seedlings, and thus allows the fungus longer opportunity to attack them [12].

As onion smut is carried from place to place in diseased plants, and distributed also in contaminated soil, all infected material should be collected and destroyed by burning, but it is by no means an easy matter to eradicate infection from the soil. If the affected area can be given over to a complete change of crop, infection may, in time, be starved out by withholding all susceptible hosts, but as the period of survival in the soil is so uncertain, smutted land should be allowed to go out of cultivation altogether ; if this is not possible, and the area is given over to other crops, these plants must be confined to the area and not moved about to clean land, a process which would immediately render that soil contaminated from the

smut-laden soil carried over with them [2]. A considerable measure of success has, however, been obtained, especially in America [4, 9, 17], by treating the soil with dilute formalin; a solution of 1 pint of formaldehyde (40 per cent.) in 16 gallons of water is poured in a fine stream into the furrow, immediately after the seed has been sown, and before it is covered with soil [1]. For treatment of large areas the American method of fixing a small tank containing the formalin on to the seeder is used, so that the outlet pipe from the tank delivers the fungicide just behind the falling seed, and with a suitable tap on the outflow pipe, the correct amount of liquid can be estimated. The seeds are covered as soon as possible after the treatment, and unless the soil beforehand is exceptionally dry, the seeds suffer no injury. If onions are grown from transplants raised in boxes, they should not be placed in smutted soil until past the susceptible stage, which, as already stated, is usually 3 weeks, when the seedlings are about 3 inches above the soil. All boxes and soil used for sowing under glass should be sterilised by heat or by the above formalin method [1].

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White Rot of Onion, *Sclerotium cepivorum* Berk.

White rot attacks onions, leeks, garlic, and shallots, and is common in regions of cool climate. It was first discovered in England in 1841 [3], and was further investigated in 1920 when its destructive effect on onion crops was reported from many parts of the British Isles [4]. In some seasons losses may be very high, as in the autumn of 1937 when entire fields of onions in the west of England were devastated with white rot [11, 12]. First report of its occurrence in America came from Oregon in 1918, on garlic and onions [13]. The disease is also prevalent in north Italy, France, Spain, Holland, Germany, the Canary Islands, Egypt, and Australia [2, 4, 10].

White rot is caused by *Sclerotium cepivorum*, a soil-inhabiting fungus, in the life-history of which, as far as is known, no spores of any kind are developed, the organism surviving in the soil in the form of minute black sclerotia (micro-sclerotia). The fungus possesses numerous strains [10, 14].

The disease attacks its hosts during active growth and continues as a bulb rot after harvest [13]. Early signs of its presence in the crop are usually to be seen towards the end of May or early June, when some of the older leaves turn yellow from the tips downwards ; and the die-back may be so rapid, especially if the plants are young, as to cause complete wilting of all the leaves. But white rot is essentially a disease of the rooting system, and it is from infected roots that the bases of the bulb scales are attacked. In the case of late bulb infections, however, the scales may be penetrated directly from mycelium in the soil, so that light infections may sometimes occur on bulbs which are almost mature. Such incipient infections are often very difficult to detect and are a source of great trouble in storage or during transit of the exported product, for the decay is progressive and quantities of bulbs are often rendered worthless [13].

On account of extensive rot at the roots diseased plants are easily pulled up from the soil, and the bulbs usually contain abundant white mycelium around the basal parts of the scales, where it becomes woven into a plectenchyma, forming a whitish-grey felt on which tiny black sclerotia duly appear (Fig. 331). These small and hard bodies which measure from 0·28 to 0·6 mm. are often found in such great numbers on the bulb scales as to form a black, gritty crust, and may either be superficial on the webbed mycelium or embedded in the outer tissues of the scales. On the disintegration of the diseased bulbs the microsclerotia are liberated into the soil in which they are said to be still viable after 8 or 10 years ; they are highly resistant to all factors of the soil environment and appear to be the principal means of carrying the disease over from one season to the next. In cases where white rot has been reported on land in which onions or leeks had not been grown over a long period of years, or on land known not to have carried the crop at all, there is evidence that the common weed, crow garlic (*A. vineale*), may act as a carrier host ; the fungus has also been isolated from the inflorescence of leeks kept for seed, infective material being found among the seed collected from the dried heads ; such infective material may thus contaminate the seed and account for new infections in clean land [6].

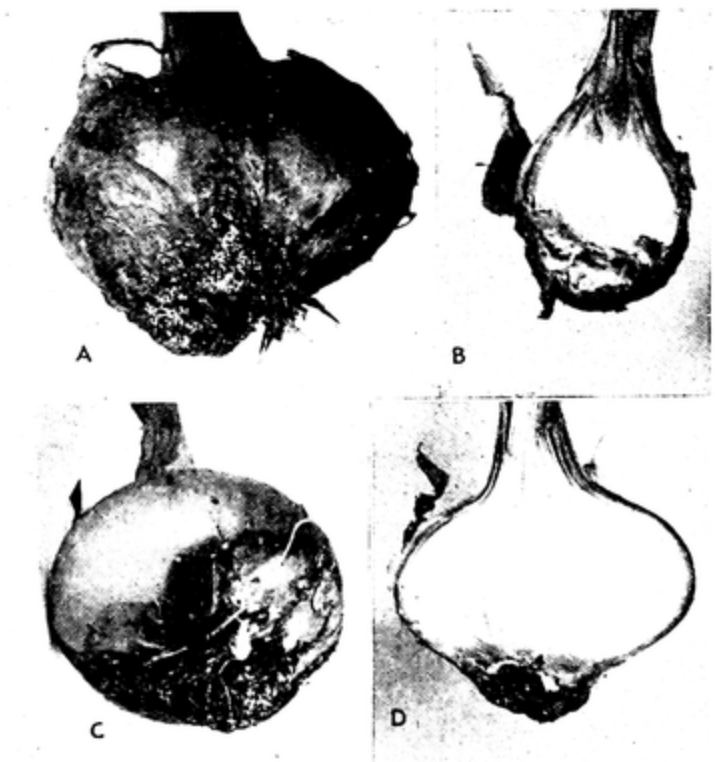


FIG. 331.—White rot of onion (*Sclerotium cepivorum*). *A*, large bulb with sclerotia near the base. *B*, smaller bulb, in section, showing rot at base. *C*, bulb showing basal rot. *D*, the same in section (photos by Foister & Noble)

The exact conditions which favour the germination of the sclerotia in the soil are not known,

nor is it clear whether they exist intact in a resting condition for indefinite periods or germinate forthwith to produce a mycelium which is capable of existing as a saprophyte until a susceptible crop is sown again [12]. Following upon early infection and destruction of the roots, the fungus soon appears as a white felted growth on the bulb scales; when the bulb is submitted to direct scale infection early signs of the disease are indicated by a semi-watery decay, which is soon followed by a vigorous growth of mycelium at the bases of the scales [10, 13]. Pure cultures of the organism have been obtained on a variety of media, both from the vegetative mycelium and sclerotia, and considerable differences were found between various isolates; colonies on prune agar, at 24° C., yielded in some cases the typical whitish mycelium which ultimately developed small knots of hyphae, the precursors of microsclerotia, and after 12 days the culture was covered with these black bodies [10].

White rot is most prevalent in cool weather, at temperatures of 15° to 18° C., and at medium soil moistures. In very dry seasons attacks are slight [7]. As the organism is essentially a root parasite, the soil-temperature factor is of great importance. In soil contaminated with sclerotia young onion plants were thoroughly diseased in 40 days at temperatures of 14° to 18° C., with a reduction to half the loss at 22° and 24° C., while between 26° and 30° C. infection was absent [13, 14]. Factors other than temperature and soil moisture are, however, not without their bearing on infection, for the critical point at which infection may be inhibited is not that at which there is an evident check to the growth of the fungus, but that at which the host is growing most vigorously [14]. The general conclusion is that white rot is a low-temperature disease, becoming aggressive at soil temperatures below 20° C., above which the plants may escape infection; it may occur at as low a moisture content of the soil as will support the growth of the host; but with a rise of the moisture content to 50 per cent. and above, a gradual reduction in infection will occur, despite the fact that the temperature may remain at the optimum for the pathogenicity of the organism [14].

For the control of white rot it is obviously of importance to prevent the liberation of the sclerotia from diseased bulbs into the soil, by early removal of such with as much adherent soil as possible to prevent their dispersal. On a large scale, in the field, there is no method of sterilising the soil to kill the scerotia, but on an experimental basis it has been found that if the disease is early detected on seedling onions, disinfecting the ground with a 2 per cent. solution of formalin may prevent sclerotial development [5], and the application of a mercuric dust, at the rate of 1 oz. per square yard of soil surface, before sowing, is also recommended [11].

There are several varieties of onions resistant to white rot but none immune from it. Those recommended include Up-to-Date, Rousham Park Hero, Improved Reading, and White Spanish; Bedfordshire Champion, Sutton's A1, and Wroxton Globe, are moderately resistant. The susceptible kinds include Ailsa Craig, Giant Zittau, Cranston's Excelsior, and Premier. The salad variety White Welsh is resistant, White Madeira only moderately so; White Lisbon is very susceptible [1, 5, 8, 9, 11]. Plants grown in England from seed of home strains are said to be more resistant than those raised from imported seed [1].

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Neck Rot and Leaf Rot of Onion, *Botrytis allii* Munn ; *B. byssoidea* Walk. ; *B. squamosa* Walk.

Neck rot usually attacks onions and shallots soon after harvest, and in storage. In some years it may be found on the crop before lifting with subsequent severe losses in the store ; autumn sown onions may also show it in the spring, and seedlings in the boxes or soon after planting may be attacked at the base [4a]. The disease was first described in 1876, in Germany, and was first reported in England in 1894 [4], and recorded here again in 1920 [1]. The first record of its occurrence in America was in 1890 [3]. The disease is also known in Denmark, France, Italy, Holland, and Japan.

The causal organism of neck rot has, for a long time, been known as *Botrytis allii* [5] ; but investigations in America [8 13] have revealed that two other species of Botrytis, *B. byssoidea* and *B. squamosa* (Fig. 333 A, B) are also implicated, and all three attack the host in the same way, through the neck tissues (Fig. 332). *B. allii* is usually associated with that form of the disease which is accompanied by abundant sporulation on the decaying bulbs, this well-established type being commonly called ' grey mould neck rot ', from the characteristic colour of the sporiferous branches. A second type of the rot, developing an abundance of mycelium, but showing poor sporing capacity, named ' mycelial neck rot ' is attributed to *B. byssoidea* (*Sclerotinia porri* is associated with this species [13]). A third type, caused by *B. squamosa*, is called the ' small sclerotial neck rot ' because of the production of smaller sclerotia, than by the other two species [12]. The two first-named species approximate very closely in many features, especially in their degree of pathogenicity and range of distribution, whereas the last named appears to be relatively unimportant, attacking only the white onion within restricted areas. It has, however, been recently observed, in 1941, in Worcestershire, to cause considerable leaf injury in onions [3a]. All three species have also been recognised in Holland [7], but in most areas *B. alii* is the principal cause of neck-rot disease ; *B. cinerea* is also reported in some parts of England to produce a shrivelling and death of the leaf tips of onion [4a].

Serious losses from deterioration in storage are attributed to neck rot, both in Britain and America, especially of the white varieties of onion, which appear to suffer more severely than the coloured yellow or red varieties [2].

Affected bulbs examined soon after lifting show small white spots on the still green but decaying leaves, which soon enlarge in a direction along the length of the leaf ; these spots may be surrounded by a yellowish area having a water-

soaked appearance. But, for the most part, the trouble starts at the neck of the bulb, extending downwards and spreading over the scales along one or more tracks which assume a sunken, brown, 'cooked' appearance, and clearly marked off from the adjacent, as yet, unaffected parts of the bulb. Dense tufts of conidiophores laden with masses of smoke-grey conidia soon arise over the affected areas [5]. The spread of the disease downwards from the neck into the scales is more progressive in a longitudinal direction into individual scales than across, from scale to scale, within the bulb [12].

With the advance of the disease, under favourable storage conditions, black sclerotia appear on the older, decayed tissues, chiefly in the region of the neck, either singly at various places, or in such profusion as often to form a continuous black crust around the entire neck of the bulb (Fig. 332). The sclerotia are embedded in the surface tissues of the scales, and may sometimes be found between the outer and inner scales, but at other times sclerotia may be entirely absent. When the disease is seen to have started at some point other than the neck, such as the base of a scale, or from any place at the side of the bulb, such infections are almost certainly traceable to wounds at these points, caused probably by careless handling at harvesting. Considerable infection through cracks in the scales and disc caused by second growth may occur in the field when rain follows a dry spell [4a]. Without such wounds for its entry, the fungus does not appear to be capable of penetrating the dry outer scales of the bulb, but can spread with the greatest of ease in the store by contact between bulbs with bruised surfaces. The progress of the disease is very much the same at whatever point it begins on the bulb, and as it causes a gradual drying-up of the tissues, the bulb becomes shrunken and mummified, so that the disease is actually of the nature of a dry rot, and unless



FIG. 332.—Neck rot of onion (*Botrytis allii*; *B. byssoidea*; *B. squamosa*). On large and small bulbs, the lower picture showing clusters of small black sclerotia on the decayed tissues of the neck (photos by Foister & Noble)



FIG. 333.—*Botrytis byssoidea* and *Botrytis squamosa*. *A*, *B. byssoidea* showing the characteristic features of the conidiophores, conidia, and their germination. *B*, *B. squamosa*, the same ; note, in the lower figures, the degenerating sporiferous branches shrinking back in accordion-like folds (× 300) (after Walker, *Phytopath.*)

saprophytic bacteria enter later, it does not assume the character of a soft, watery, and foetid decay.

The mycelium of *B. allii* is somewhat coarse, richly branched and septated, and exists in the tissues of the host both between and within the cells. During early occupation, the mycelium is colourless but darkens with age into a light-brown tint, but when it collects between the bulb scales it usually forms a dirty white, matted growth. In artificial culture, as on onion-decoction agar, the strongest growth of mycelium occurs under conditions of alkalinity or low acidity, but under comparatively high concentrations of either acid or alkali, the fungus passes over to the production of conidia [5]. The mycelium that comes to the surface to sporulate is of a smoky-grey colour and gives rise to dense masses of erect conidiophores, deep brown in colour. The conidiophores branch repeatedly near the apex, ultimately to produce very numerous, short, dilated tips beset with tiny sterigmata from which the conidia are abstricted in great number. After dispersal of the first load of conidia, a conidiophore may renew its growth by proliferation, again to branch and sporulate as before, until finally several conidiophores become flattened and twisted upon themselves. The conidia are pale or hyaline, oblong to elliptical, and measure from 7·1 to 16·2 by 3·8 to 6·3 μ (average, 10·3 by 5·1 μ) [5]. They germinate in water to produce one or two germ-tubes which branch out with frequent anastomoses. Smaller microconidia are also sometimes produced ; they are globose, about 3 μ in diameter, and borne on short, hyaline conidiophores [12, 14]. Sclerotia, during their early development, appear as white, velvety tufts of mycelium sunken somewhat into the surface of the bulb, but gradually change in colour from light brown to black, finally becoming very hard and horny ; on the side adherent to the bulb they are slightly concave, and rounded on the exposed side ; they vary in size from 1 to 5 mm. in length ; internally, they consist of densely interwoven pseudoparenchyma, and on the outside are protected by several layers of heavily pigmented cells which give the characteristic black colour. If diseased bulbs are allowed to decay and rot in the open, these highly resistant sclerotia pass into the soil, but for how long they are capable of survival in the soil and are responsible for the renewal of infections in succeeding crops is not fully known. The period of longevity appears to depend on the degree of moisture in the soil, and under very dry conditions the germination of the sclerotia may be long retarded [5]. While the sclerotia may, no doubt, be capable of withstanding the winter, it is not improbable, at least in some localities, that the mycelium itself, and perhaps the conidia too, may carry the disease over from one season to the next [12]. Conidia picked from dry cultures of onion agar were found to be viable after 2 years, and mycelium was rejuvenated from cultures 9 to 12 months old [5].

Neck rot attacks its host under conditions of high humidity. No infection of normal uninjured leaves, nor of neck tissues, occurs if these parts are dusted with the dry conidia, but if drops of water are supplied, or the plants are wet with dew or rain for two or three days, with prevalence of cloudy weather, infection is generally successful. There can be little doubt that the fungus is capable under humid conditions of penetrating unbroken cuticle of succulent, turgid scales, and that possibly some amount of infection may thus take place actually in the field, before the tops have dried out, for it is found that bulbs with immature tops show a higher percentage of infection than those which are thoroughly ripened. If, however, as already indicated, the host surface is bruised or in any way injured, the insertion of spores or sclerotia into such wounds never fails to result in rapid infection. In the great majority of infections occurring on matured bulbs, the spores gain access to the neck tissues through wounds made by twisting off the tops at harvest, or through careless handling during storage, or by bruising of the scales while screening. The spores grow easily in the material supplied to them by the moribund tissues at the wound, or in the dead remains of leaves in the tops left on the ground. Under natural conditions it appears to be very exceptional for infections to become established through unwounded outer scales, and though white varieties of onions are usually more susceptible to the rot than coloured kinds, both are equally susceptible when infected through wounds. Succulent, sappy bulbs are much more prone to this disease than hard, well-ripened bulbs.

Humid, cool weather during the growing season appears to render the plants susceptible to infection. In well-grown crops, ripening time is indicated by the leaves toppling over while still green ; thick-necked bulbs do not do this so readily, and bulbs with thin necks are hardier, less liable to infection, and are altogether better for storing. It should be the practice, at harvest time, to pull the plants during dry weather and allow them to ripen in the sunshine ; when this process is carried out under damp conditions, or when the bulbs are allowed to remain in the wet, much of the infection is given a start at harvesting [2].

All the three species of *Botrytis* cited above respond quite similarly to variations of temperature. At low temperatures of 3° or 4° C., growth was found to be very slow, the optimum lying between 20° and 25° C., and the growth of all three was meagre at 33° C. The most rapid decay of the bulbs occurred at 15° to 20° C., and above 26° C. the amount of disease and rate of decay decreased appreciably [12]. While it has been observed that moist, cool conditions are favourable to the growth of the fungus, these are also the conditions which delay the maturing of the crop, tending, as already mentioned, to increase of succulence in the neck tissues, and thus retarding the important process of the drying of these tissues, a procedure of greater importance as a means of preventing infection than any other devised for its control.

The selection of coloured varieties of onions in preference to white is advised, since they have been found to be generally more resistant to practically all diseases of this plant. The resistant principle appears to reside in the pigments existing in the outer scale and neck tissues of these bulbs, and is believed to be proto-catechuic acid, a substance which has been found experimentally to be extremely

toxic to the fungus of neck rot [6, 12]. While the cultivation of the crop, to ward off the disease, should aim at the production of sturdy growth, delayed growth should not be encouraged as it tends to increase the succulence and thickness of the tissues in the neck region. Only bulbs with firm, narrow necks, the dead tips of which easily fall off, should be used for storage purposes. Bulbs in store should be frequently examined for symptoms of decay ; they should be housed on latticed shelves for thorough circulation of air to keep them dry. If the weather at time of ' curing ' is unfavourable, it may be found advisable to resort to artificial drying, the bulbs being set out in shallow crates, and placed in a kiln through which warm air at 100° to 120° F. is forced for several hours until the neck tissues are thoroughly dry [2, 8].

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White Tip of Leek, *Phytophthora porri* Foister

This disease which is confined to leeks has probably been in existence in Britain long before its discovery in 1928 in Scotland [1]. Since that date it has spread and is now present in the Lothians down to the Borders. In England it has no doubt been confused with downy mildew of the onion for many years, and following soon upon the discovery in Midlothian, it was recorded in 1931 as being somewhat severe during 1929 near Cheltenham and around Bristol [3, 4, 5], but, so far, there are no reports of its occurrence outside the British Isles.

White tip causes a yellowing and die-back of the leaves, starting, as the name suggests, at the tips, these affected parts finally turning white (Fig. 334). The bleached effect may extend from the tip for about ½ to 6 inches, and the discoloured part may either collapse and decay or become crisp and curled. These early symptoms may, however, start at one or both margins of the leaf, at any point from near the tip to about half-way down ; a one-sided attack causes the leaf to contract and twist around the infected part. Infection causes some degree of water-logging of the tissues, either close to the bleached area or in advance of it, and may start half-way down or at the base of the leaf, in the latter case causing the whole leaf to wilt.

The disease attacks leeks young and old, the former remaining stunted, and the

latter, if severely infected, rotting away and breaking off at soil-level when pulled up. Even plants slightly infected wilt early when gathered, whereas healthy ones remain firm for several days. The trouble usually breaks out rather late in the season, but the time varies much with the locality and season. In Scotland, in 1928–9, it began in September; in 1929–30, in January and in 1930–31, in December [2]. In the Bristol area in 1930 it was fairly general by October, and in 1931 was observed on plants about to be harvested; and on winter leeks it commenced on infected ground early in November [4]. These discrepancies in times of outbreaks in England and Scotland are probably correlated with the prevailing temperatures and length of incubation, the latter being longer in Scotland [2].

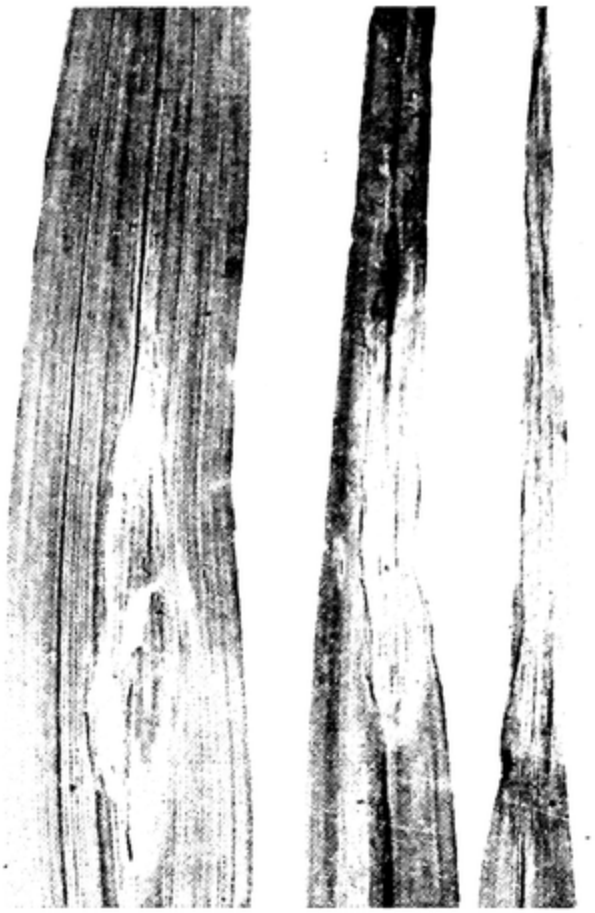


FIG. 334.—White tip of leek (*Phytophthora porri*) (photo by Foister, *Proc. Bot. Soc. Edin.*)

The fungus causing this disease is *Phytophthora porri* [2]. Its mycelium is present in greater quantity in the yellow, waterlogged parts than in the white portions of the leaves, and consists of hyphae which are exceedingly variable in width, from 2·4 to 12 μ (average 6 to 8 μ), and appearing in places almost vesicular; it is non-septate, moderately branched, and occupies the cells and intercellular spaces. The mycelium may travel from the leaves into the stem, even into the base of the plant but not in any great quantity. In culture, the mycelium shows peculiar coiled or spiral branches, especially when about to form oogonia and antheridia. The optimum temperature for vegetative growth on an oat medium was found to be 25° C., the minimum and maximum being 8° and 35° C. respectively. It is rather remarkable that while many species of *Phytophthora* develop their asexual fructifications in great abundance, the sexual organs being usually sparse or imperfectly developed, *Phytophthora porri* has not been observed to form sporangia on the host plant under natural conditions whereas the sexual structures are found in great profusion. In culture, however, sporangia, sexual organs, and oospores are produced abundantly. The sporangia are inversely pyriform, with or without an apical papilla, and vary in dimensions according to the nature of the medium; when cultured on the host they measure from 37 to 75 by 31 to 48 μ; on soil, from 31 to 55 by 28 to 42 μ; and in water from 31 to 82 by 23 to 52 μ; they are terminal or intercalary on the sporangiophores, and germinate direct by germ-tubes, or indirect to form zoospores which measure from 10 to 15 μ. Oogonia and antheridia are found in abundance in the yellowed parts of the leaves. The oogonia are rather thick-walled, hyaline, from 29 to 44 μ in diameter (mean between 38 and 39 μ); antheridia may be paragynous or amphigynous, and the species is homothallic; oospores are spherical, thick-walled, light yellow, and tend to mature more and more as the leaves get dry; they measure from 19 to 36 μ in diameter (mean,

between 32 and 33 μ) ; both oogonia and oospores are much smaller on the host than in cultures.

The mycelium is capable of growing saprophytically on the soil, but makes little headway in soil cultures unless other organisms are leached away or killed by soil sterilisation. In a series of experiments on host infection a culture of mycelium in sterilised soil failed to infect young seedling leeks, but developed sporangia in great profusion on the surface of the soil. In many instances aerial infection of plants was traced to infected soil, and it is not unlikely that sporangia produced on soil are carried by wind to set up infections of the green parts of leek plants. If sometimes sporangia do actually occur in nature on the host, they are probably produced only under conditions of very high atmospheric humidity ; during a series of inoculation experiments sporangia were actually found inside the leaf, having developed within the tissues, presumably owing to the higher degree of enclosed humidity than outside the leaf. They are developed readily if pieces of pure cultures are placed in water or liquid media, at 15° to 18° C. ; at 10° C. they do not develop.

The disease may be kept in check by dusting the plants with a mixture of 1 part of copper sulphate to 4 parts of hydrated lime, applied at the rate of 2 oz. per square yard, and repeated every 3 or 4 weeks, if necessary, and is advisable in places where the trouble has previously existed. But as the disease does not spread very readily, it can often be checked even after its appearance, and the dusting may not be necessary in the early spring as by then slightly attacked plants may outpace the infection. No varieties of leeks are known to be immune ; the varieties Lyon and Musselburgh are very susceptible in the field.

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Chapter XV

DISEASES OF FRUIT

Phytophthora Rot of Apple and Pear

Phytophthora cactorum (Leb. & Cohn) Schroet., & *P. syringae* (Kleb.) Kleb.

THIS type of fruit rot is fairly common on apples and pears in Britain, Europe, and America [10, 12, 14, 15, 19, 23]. In some cases it is due to the activity of two species of *Phytophthora*, *P. cactorum* [13] and *P. syringae* [11]; in other instances only one or other of these two fungi is implicated on either of the two hosts [12, 15]. In general, *P. cactorum* appears to be the species more commonly concerned; in America it causes a ' collar rot ' of apple trees in addition to a rotting of the fruit [2], and is also known in England and America to affect the fruit of the strawberry causing a ' leathery ' rot [1, 3, 18]. It has also been found to injure the roots of hops in Sussex and Kent [10 a]. *P. syringae* also attacks the buds and shoots of lilac (*Syringa*) [6, 7], and is implicated in ' ink disease ' of sweet chestnut (*Castanea*) [9].

Phytophthora rot was first observed in 1904 in Switzerland, on fallen apples, and the same disease was later reported to cause a die-back of the branches [16, 17]. It is observed mostly on windfall apples and pears on wet ground, rarely on fruit on the trees.

The general effect of the rot in both apple and pear is that of a brown discoloration of the core and flesh, both in the larger vascular bundles near the core and in the smaller ones throughout the fleshy pulp; the browning from the larger bundles may extend into the stalk of the fruit for a part or the whole of its length [19, 21]. In neither case has the affected fruit any marked odour or taste [19].

There are, however, some slight differences in the symptoms on the two hosts. In the apple the discoloured flesh is not so deeply browned as in the pear, and the external lesions are also a lighter brown than they are on the pear. Glistening clusters of asexual sporangia appear on these surface lesions and the spores are perhaps more plentiful on the pear than on the apple. In apples affected with *P. cactorum* the mycelium is often to be seen through splits in the skin, forming a whitish bloom on the surface [24]. When *P. syringae* occurred on certain apples, white tufts of mycelium were found to develop at the lenticels while the fruit still retained a more or less firm texture [12]. In another instance, the same fungus produced brown areas on the skin of the apple, rather like bruises, but without altering the shape of the fruit which also remained somewhat tough, a condition believed to be due to a different strain of this fungus [15].

P. cactorum produces various types of spores (Figs. 25, 335) between which it is not easy to distinguish in culture, by size, shape, or mode of germination [4, 5]. These include

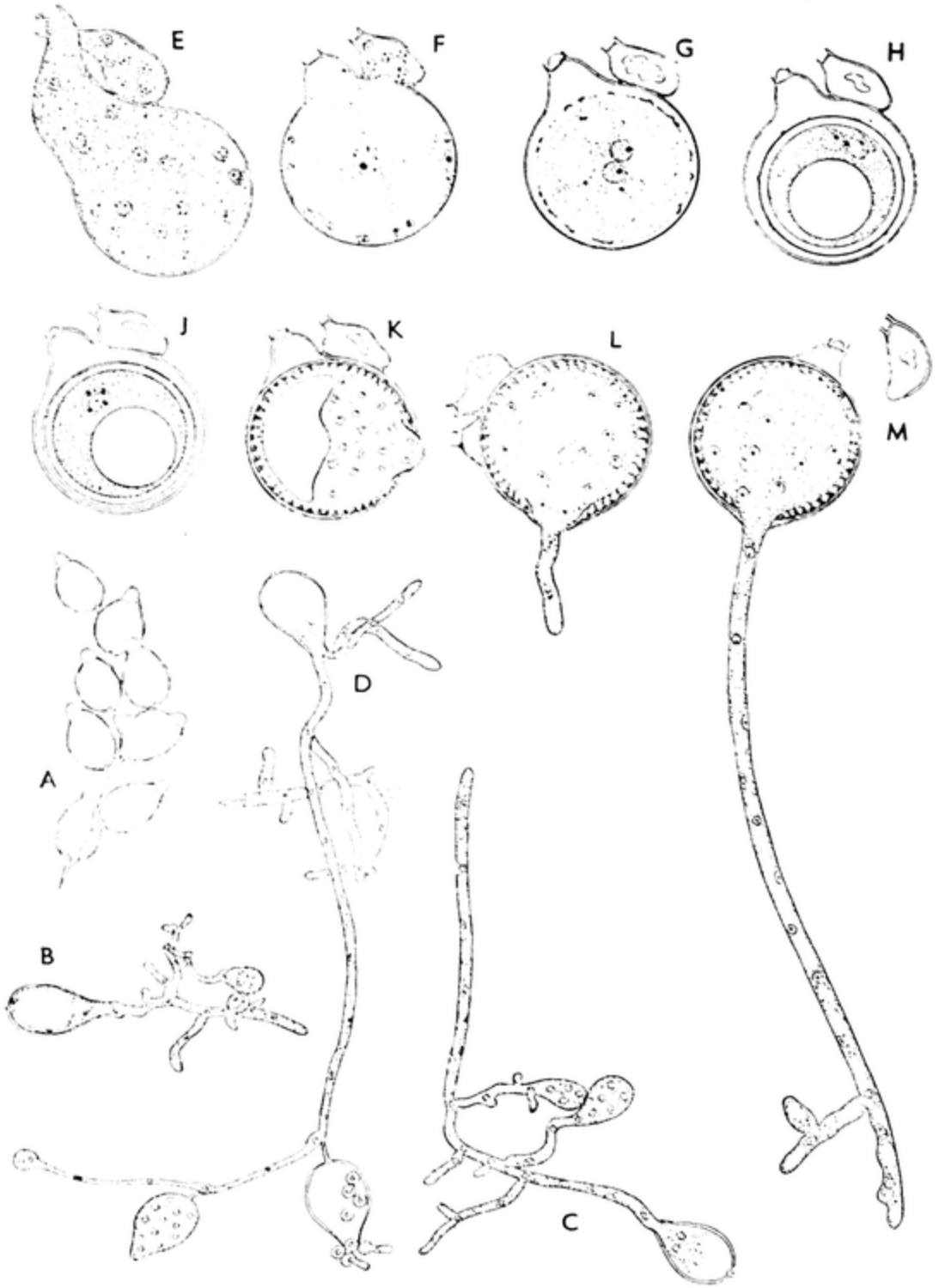


FIG. 335.—*Phytophthora cactorum* causing apple rot. *A*, unichasial sympodium of a sporangiophore grown in a moist atmosphere (in outline only × 270). *B*, *C*, *D*, resting sporangia which have germinated to give short mycelia and reproductive organs. *B*, *C*, bear a young oogonium and antheridium. *D*, bears sporangia (these resting sporangia were found in a seven-month-old culture which had been soaked for 2 days in dilute apple juice) (× 300). *E–M*, stages in the development of oogonium, antheridium, and oospore (composite drawings of median longitudinal sections) (× 700). *E*, oogonium and antheridium, a few hours old, grown to full size; the oogonium has about 24 nuclei and its antheridium about 9 nuclei. *F*, ooplasm separated from periplasm; nuclei of periplasm are in division prior to degeneration. *G*, antheridial nucleus has entered ooplasm; oospore wall forming. *H*, immature oospore; the two nuclei not yet fused. *J*, oospore after a period of dormancy; nucleus preparing for division, oil globule still

typical sporangia, resting sporangia, sphaeroconidia, chlamydospores (all multinucleate), in addition to the sexually produced oospores (uninucleate). The oogonia may be fertilised by paragynous (chiefly) or amphigynous antheridia. The non-septate mycelium produces the ordinary thin-walled, elliptical or pyriform sporangia singly and terminally on sympodially branching sporangiophores 20 to 40 μ long ; the latter emerge through the epidermis in small tufts ; the sporangia measure from 35 to 65 by 22 to 35 μ, and the papillae are from 4 to 6 μ long ; occasionally sporangia may be found to measure from 70 to 80 μ in length [23] but a typical one is about $36 \times 28\ \mu$ [5]. Conditions of high humidity and good aeration favour the production of sporangia [22]. Germination occurs direct by germ-tube or indirect by zoospore formation. Resting sporangia, formed in the same way as the ordinary sporangia, are adapted for long survival and are laden with fatty reserves ; they germinate direct to produce thin-walled sporangia which in turn produce germ-tubes and more sporangia so that continuous systems or chains of sporangia may result. Or, a resting sporangium may produce vegetative mycelium, or sporangia, or even oogonia and antheridia almost immediately on germination. Sphaero-conidia are extremely variable in size, and may arise in an intercalary, rarely terminal, position on short hyphae ; they are merely hyphal swellings, and measure from 33 to 40 μ in diameter. Chlamydospores are also spherical, terminal, rarely intercalary, and without a papilla ; they are thicker-walled than the resting sporangia and rest for a longer period ; they are laden with oily reserves ; they produce a germ-tube which may give rise to sporangia, or sporangia with zoospores. The oospores have a thick, smooth wall, and measure from 22 to 30 μ in diameter [23]. The oospores must be fully matured before they can germinate, a process which has been rarely observed, demanding a long period of dormancy or ' ripening ', but in a culture 6 to 12 months old they were found to grow when kept moist at 15°-25° C. [4, 5]. In its possession of a highly resistant mycelium, oospores, chlamydospores and resting sporangia, *P. cactorum* is eminently well supplied with means of perennation.

The sporangia of *P. syringae* are non-papillate and this fungus does not produce sphaero-conidia [12]. The sporangia, borne on sympodially branched sporangiophores, from 1 to 7 in number, measure from 40 to 75 by 30 to 42 μ [11] ; or have an average of 38 by 26 μ [12]. The oogonia are spherical, 26·4 μ in diameter, containing each a round or slightly oval, yellowish, smooth-walled oospore. The oospores have an average diameter of 24·4 μ [12], and may range from 18 to 36 by 17 to 25 μ [11].

This fruit rot is somewhat sporadic and localised in its occurrence and is severe only during wet weather at the time the fruit is approaching maturity [23]. It is not very clear how initial infections become established on the fruits of apple or pear, but under experimental conditions uninjured fruits of both were successfully infected when placed in dishes containing wet soil collected from an infected orchard [19]. The uninjured fruits were also infected through the lenticels by the application of an inoculum of zoospores [8]. There is evidence that in old orchards suffering every year from this type of fruit rot, the fungi concerned are capable of survival in the soil under the trees. It is not improbable that under such conditions contaminated soil may sometimes be splashed up to the lower branches

undigested, endospore becoming eroded in digestion. *K*, oospore on the point of germination ; nuclei about 32 in number, neither oil globule nor endospore completely absorbed ; germ-tube through exospore, but not yet through oogonium wall. *L*, *M*, later stages in germination ; the antheridium occasionally becomes loosened from the oogonial wall (after Blackwell, *Trans. Brit. Myc. Soc.*)

of the trees, especially those of cordon trees, and possibly through wounds, cause infection which may bring about collar rot, or a die-back of the branches, as above mentioned [2]. Artificial infections have also been successfully performed on fruit whilst still growing on the trees ; inoculum introduced into wounds in the skin of a pear resulted in infection, causing the fruit to drop, and in another instance when zoospores were placed in water at the stalk end of a pear on the tree the fruit dropped at the end of 10 days and showed a discoloration extending from the stalk-end for about 1 cm. down one side, but such infections were not always successful [23].

Since the fungus continues to develop on the fallen fruit this should be collected and burnt, and any rotted fruit on the trees should be removed.

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Mildew of Apple and Pear, *Podosphaera leucotricha* (Ell. & Everh.) Salm.

Apple mildew is common in all fruit-growing areas and in some seasons causes considerable damage in Britain ; pear and quince are also susceptible to the same disease [2, 5, 7, 8, 9, 12, 15, 22].

The mildew is usually seen on apple trees as soon as the buds burst into leaf. Sometimes buds are so heavily infected in the previous season that they are killed outright, and infected buds which succeed in making growth produce only weakened, mildewed shoots. Early symptoms on the leaves from such buds consist of small patches of white or grey mildew on the under surface (the first part of the young leaves to emerge) but as the leaves develop, both surfaces become covered with a white floury coating of mycelium and spores (Fig. 336). Affected leaves grow longer and narrower than ordinarily, and by a curling of the margins appear

to be somewhat blistered or crinkled. Later, starting at the tips, the leaves turn brown, the discoloration spreading gradually until the entire lamina presents a scorched, bronzed appearance, a condition which results in the death of the leaves. The effect of a severe foliage attack is to bring about partial defoliation, and except for a tuft of small leaves at the ends of the shoots some branches may be quite denuded of leaves. An affected tree may often be left with only a quarter of the foliage of a healthy tree, and if attacks are recurrent every year the amount of wood formed becomes less every season. Early attacks on young nursery growth may prevent the formation of wood entirely [5]. Fruit buds usually suffer more from the mildew than vegetative buds, often to such an extent that they fail to produce any blossom at all. Those attacked in blossom may show the flowers with distorted sepals and pedicels, with the petals unusually narrow, and pistils and stamens are frequently sterile so that no fruit is formed [6]. Fruits attacked when young remain small and deformed and tend to develop a roughened surface.



FIG. 336.—Apple mildew (*Podosphaera leucotricha*). The lesions on stems, leaves, and blossom (photo *U.S. Dept. Agric. Frmrs'. Bull.* 1120 by permission of H. P. Gould)

Apple mildew is caused by *Podosphaera leucotricha* (*Erysiphaceae*). The superficial mycelium, which sends haustoria into the epidermis (Fig. 337), gives rise throughout the season to a profusion of aerial conidiophores on leaves and young shoots. Each conidiophore develops a chain of conidia which are set free and dispersed by wind. The conidia are oval, and measure from 28 to 30 by 12 μ. The fungus also produces perithecia (cleistocarps), and though by no means scarce, they appear, at least in some countries, to play little, if any part at all, in carrying the disease over from one season to the next. They have been found in England on some varieties as early as May, and on strong-growing twigs may continue to be formed well into the summer, and on which they persist through the winter. They occur mostly on the current season's wood (rarely on older wood) and may sometimes be found on sucker growth at the base of the tree, on the midrib and larger veins of the leaves, or on leaf stalks, rarely on the fruit [21]. These fructifications [19] are globose-depressed, black, partially embedded in the mycelium, and measure from 75 to

96 μ in diameter. They are furnished with two kinds of appendages ; those which spread out from the apical, depressed part are long, stiff and bristly, while the others which emerge near the base are short and tortuous and evidently serve to secure the cleistocarps to the mycelium ; the function of the apical appendages is not clearly known but may possibly be that of retaining moisture within the depressed apex of the cleistocarp. The cleistocarp opens irregularly, and the single ascus, 55 to 70 by 44 to 50 μ, may be entirely and violently ejected, carrying with it all its 8 ascospores (which measure from 22 to 26 by 12 to 14 μ), or only its tip may extrude through a slit in the top of the cleistocarp, before it breaks open to set free the spores, but as a rule the entire ascus is shot forth and in contact with water it explodes to set free the spores. Little is known about the germinating capacity of the ascospores [21].

Though cleistocarps may in some localities enable the fungus to survive through the winter to bring about primary infections in the spring, they do not appear to fulfil this function in Britain. Here and in most places the fungus survives in the form of a resting mycelium in the buds. The early symptoms of mildew, above described, which appear in the spring at the bursting of the buds are traceable

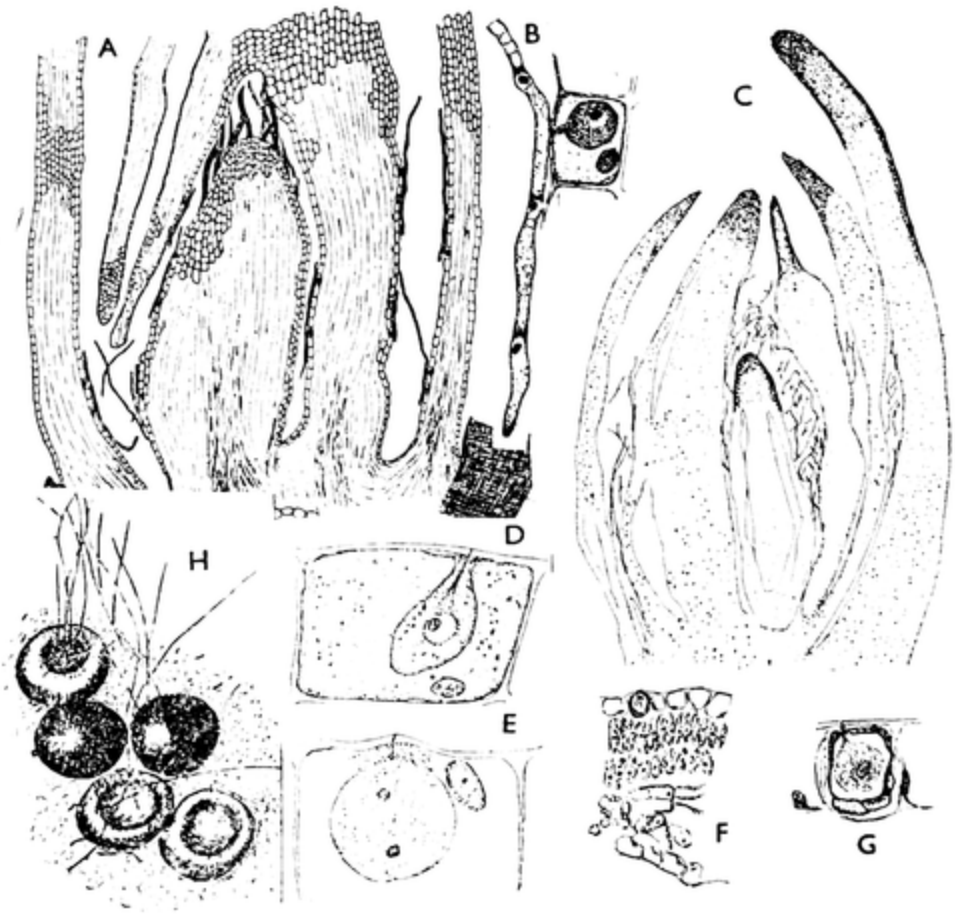


FIG. 337.—*Podosphaera leucotricha*. *A*, diagram of section through infected axillary bud (in January), showing normal hyphae and haustoria (× 40). *B*, a hypha with haustorium (× 330). *C*, median section through dormant bud of infected terminal shoot, after removal of bud scales (× 17). *D*, *E*, haustoria, latter binucleate (× 800). *F*, encapsuled haustorium in enlarged epidermal cell (× 140). *G*, the same, showing a disorganising nucleus, and barrier (× 580). *H*, the cleistocarps (after Woodward, *Trans. Brit. Myc. Soc.*)

to a hibernating mycelium which had become established within the buds during the previous season, and these early appearances of the fungus constitute the primary infections. Once the mycelium comes to the surface of these early infected shoots, conidiophores and conidia are soon produced in abundance; and whilst it is believed that no general infection occurs other than that arising from infected buds [21], observations are not wanting that the mildew can also spread by secondary infections [18].

A high degree of atmospheric humidity is more essential for successful penetration of the leaf by the germinating conidia, than actual moisture on the lamina. The conidia germinate at an optimum between 19° and 22°, or 25° C. [4, 10], and the superficial mycelium grows well at 20° C.; temperatures of 33° C., and over, are fatal to the conidia. A dry but turgid leaf, its cells of high water-content, offers the best conditions for mildew attack. The germ-tube penetrates the epidermis direct without forming an appressorium, and a simple, spherical haustorium early becomes established in the entered cell. In the leaf, most of the infections go no further than the epidermis before the developing hyphae spread out over the leaf to form the superficial mycelium, but in severe attacks the fungus may sometimes penetrate further into the mesophyll, and though it is difficult to establish their presence, there is some suggestion of the development of haustoria in the mesophyll cells, but normally these absorptive organs are confined to the epidermal cells. No doubt, such cases of mesophyll, in addition to epidermal penetrations follow upon decrease or loss of resistance in the leaf [21], and there is evidence that resistance to infection increases as the leaves get older, but even then such resistance can be partially broken down by abrading the cuticle [4].

From the time that infected buds produce their conidia-laden shoots in spring there is barely a month's interval before the fungus investing these young shoots proceeds to provide for next year's infections. This it does by attacking the tender buds in the axils of the mildewed leaves. At such an early stage of development the axillary buds are imperfectly protected against attack especially at their tips where scale leaves often fail to meet, thus offering an ideal means of entry for the parasite. Not only leaf buds, but fruit buds on the terminals of spurs also become similarly invaded. It all depends upon the severity of attack in these early bud infections whether the young buds are killed outright or whether they survive; surviving buds develop better protection by additional bud scales and become effectively sealed up. Thus protected by more effective bud scales, the mycelium nesting between the scales is enabled to survive on the tree throughout the winter, and remains dormant until the spring when the cycle of primary infections starts over again, the period of perennation being roughly about eleven months. Meanwhile the imprisoned mycelium has maintained itself within the buds by establishing haustorial connections with the bud tissues, and there is little doubt that the haustoria in the leaf tissues enclosed by the bud scales play a vital part in perennation [21].

There is no definite evidence that outbreaks of mildew are favoured by one type of climate more than another, and while it is stated that the trouble in some localities is worse in hot and dry seasons, in others it is said to be encouraged by warm, moist summers, or by cool, damp sunless weather [14, 15]. The disease

is generally worse in overcrowded orchards and on ill-ventilated espalier growth than on trees of the same age and variety, planted well apart [14]. Some state that the trouble follows on an exceptionally late and cold spring. Such was the experience in Czechoslovakia where, after an entire absence of the disease in 1927, the mildew broke out severely in 1928 following upon low temperatures in the spring, indicating that the disease had been dormant since 1926 when the trouble was reported to be very severe [6]; in 1927, in a locality in Czechoslovakia, *P. leucotricha* was found to be the cause of a graft-malformation resembling witches' broom [3].

Though there is no evidence that specialised forms of the mildew exist, the fungus reacts differently on some varieties of apple; for instance, it affects the fruit of some kinds more than others. The same mildew which, less frequently, attacks the pear shows no morphological differences from that affecting apple, but it is noteworthy that cleistocarps are of commoner occurrence on the fruit of the pear than on the twigs. These fructifications are also common on some particular varieties of apples, more than others, notably Lane's Prince Albert and Early Victoria, whereas on other apple varieties they are to be found, in general, on the twigs. Apples and pears differ considerably in varietal susceptibility to mildew and comparatively very few exhibit a high degree of resistance to the disease [15, 21].

Poor soil and wrong cultural practices are largely responsible for epidemics of apple mildew. But even slight differences of habitat, connected with exposure and altitude mainly, play an appreciable part in the course of infection [10]. In general, the trees are less resistant when grown on heavy clay than on moderately light soils. Good control over the disease can be obtained by improved cultivation, adequate manuring and well-balanced pruning to stimulate vigorous growth [1]. Since the initial outbreaks of mildew in the spring start from infected buds, all shoots mildewed during the season should be removed early because it may not be so easy to distinguish them, later on, from healthy shoots. When the buds open in the spring, all shoots showing signs of mildew should be cut off, and though this may entail considerable loss of blossom their removal will help greatly to reduce the chances of bud infection. Experiments connected with 'plant injection' in general pathology, showed that apple-shoot treatment with sodium thiosulphate controlled infection with *P. leucotricha* [11].

To protect the trees against external infection it is recommended to spray at intervals with lime sulphur as follows:

(*a*) 1/15 dilution, when the buds are green ('green-tip' stage);
(*b*) 1/35 dilution, at 'open-cluster' stage;
(*c*) 1/60 dilution, at blossoming ('full-pink' stage);
(*d*) 1/100 dilution, when about half the petals have fallen; and
(*e*) Followed at 10 to 14 days' intervals with the same strength of fungicide [1, 16, 17].

Obviously, winter spraying of the trees is useless since external application of the fungicide would have no effect on dormant mycelium in buds protected by thickened scale leaves [13]. Application of atomic sulphur in dry or liquid form is also recommended, the latter in the proportion of 10 lb. per 100 gallons of

water [5, 7]. As already indicated in sulphur treatment of apples against disease (p. 232), some varieties are intolerant of this mineral, and, for these, sulphur-sensitive kinds, spraying with washing-soda and soap (p. 820) is advised.

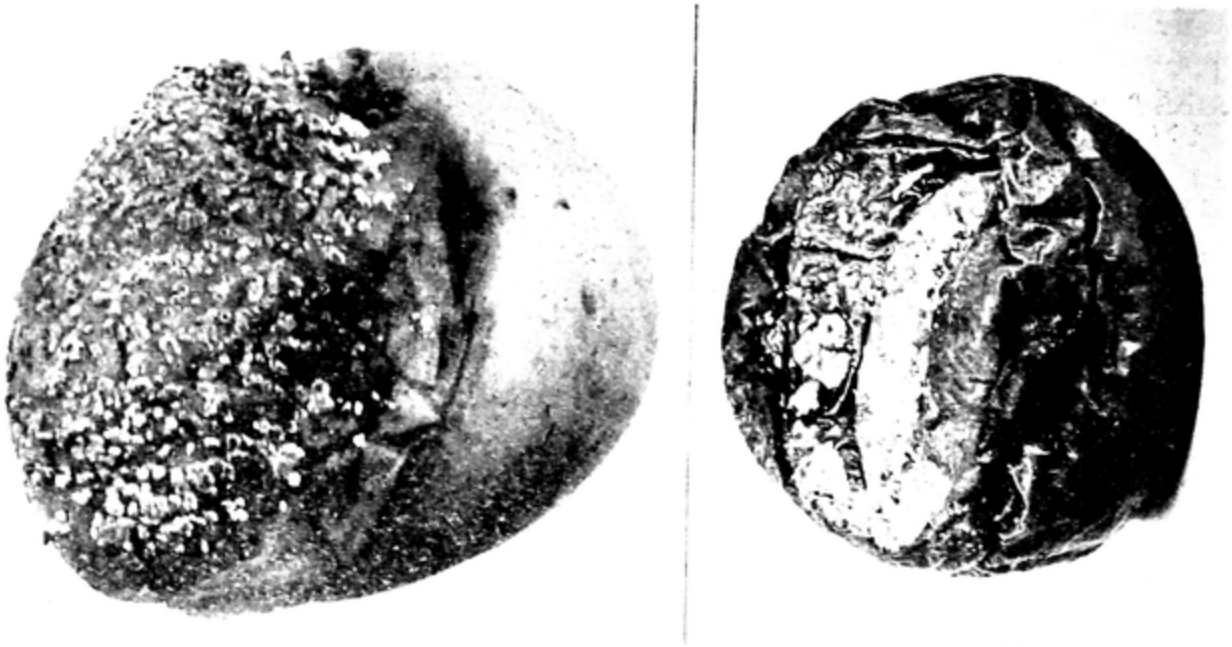
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Blue Mould of Apple, *Penicillium expansum* Thom

Blue mould is often responsible for serious wastage of all kinds of apples in storage. It produces a rapid, soft kind of rot which may start at any point on the surface of the fruit. The affected part soon becomes covered over with tufts of the bluish-green fructifications (Fig. 338) of *Penicillium expansum* causing the rot, and this fungus is probably the most common of all organisms which may be found at all times on harvested apples and other fruit [9]. In some of the larger fruit-growing areas, where it is the custom to wash the apples before packing, it has been ascertained that several million spores of this fungus pass through the washing tanks after a day's run, so that the number of spores ordinarily carried into the packing rooms and boxes of unwashed fruit sent to market, ready to attack the fruit through the smallest abrasion, must be enormous [8].

The fructifications of *P. expansum* on the apple are those which bear conidia; the perfect ascigerous stage of cleistocarps has not been found. The former consist of rather large, blue-green tufts or coremia of conidiophores, around the points of infection. The conidiophores are smooth, long, and intertwined into fascicles. The ultimate branches of the latter are arranged close together and finally terminate in narrow phialides 7 to 9 by 2·5 μ wide, which bear long, loose chains of broadly elliptical conidia (phialospores) measuring on an average 3·5 by 3·2 μ [13] (Fig. 77 c).

Infection of the fruit usually takes place through wounds in the skin, such as would be caused by insect bites, careless picking, or rough treatment in the process of washing or storing. Infection is also believed to occur through the



A B

FIG. 338.—Blue mould of apple (*Penicillium expansum*). *A*, showing clusters of the blue-green conidial fructifications (photo by Wormald, *Diseases of Fruits and Hops*, Lockwood). *B*, the early, white mycelial condition (photo by Foister & Noble)

lenticels in the epidermis of the fruit, though it has been established that the presence of small brown spots just below the lenticels is not always traceable to fungal infection but to a physiological disturbance of the apple tissue in the neighbourhood of the lenticel, and formed quite independently of infection [11, 12, 15]. Lenticels on the fruit, however, are normally not so well organised as those on the woody stems, which are furnished with phellogen, and those on the fruit may remain more or less permanently open for gaseous exchange, or become imperfectly closed merely by an extension of the cuticle or by cutinisation of the exposed cell walls, and, therefore, are more open to infection [7]. On the epidermis of very young apple fruits long hairs are also present, and if these are broken off near the base, infection may enter at the scars or hair pits. During the swelling of the fruit, the epidermal cells divide and keep pace with its growth, but at points where stomata, lenticels, and hair pits have been imperfectly sealed, breaks in the skin are liable to occur, thus giving the fungus open access to the pulp [1, 2]. It is also said that infection can enter the apple, after picking from the tree, by growing down the stalk into the fruit [5].

There are numerous factors which appear to encourage lenticular infection. It is said to be more prevalent in some localities than others, or on different varieties of fruit, and to occur more on fruit borne by young than by older trees [10]. While the number of lenticels per unit area is said to vary according to the variety, certain factors of the environment also appear to control their number and development. Thus the variety Winesap, when cultivated under conditions of high humidity, had a greater number of lenticels than under drier atmospheric conditions, but with the variety Delicious the reverse was the case [7]. On both of these varieties in Washington, the mould was more rapid in the fruit picked

from trees receiving manurial treatment than in the control trees, and in apples picked at their prime than at early maturity [4]. Other factors again, probably operating during the growth of the fruit, may bring about changes which induce the lenticels to close, and this phenomenon appears to be controlled by the rate of dehydration in the epidermal tissue ; apples picked about a fortnight before they are ripe respond quicker to lenticel healing than those left on the tree until the usual harvest period.

There is little doubt that *Penicillium* is often capable of infecting the fruit through the lenticels, not so easily perhaps as through open wounds. At the lenticels there is more rapid volatilisation of certain products of the fruit than at cuticle covered areas, and through the uncutinised cells immediately below the lenticels diffusion of nutrients takes place [6]. The presence of these nutritive materials at the lenticels assists the spores to germinate and penetrations naturally take place at these openings. When drops of water are placed over the lenticels the amount of infection may be increased up to 26 per cent., and up to 48 per cent. if drops of nutrient material are added [2].

Other conditions such as temperature and moisture relations in the store being constant, the rot tends to increase with the length of the storage period, the deterioration being due to increased susceptibility consequent upon increase in the amount of nutrients escaping by diffusion [10]. Decay is thus favoured by long storage.

This disease may be checked to a great extent if reasonable care is taken to avoid wounding the fruit at harvest. As already mentioned, picking the fruit before it is ripe decreases the chances of lenticular infection, and harvesting in dry weather obviously reduces the risk of disease. Low temperatures of cold storage actually do little more than keep the fungus in check ; they do not inhibit penetrations through wounds or lenticels, and once infected fruit is removed from low to higher temperatures, these suppressed infections make rapid advance [2]. Spores of *Penicillium* are capable of survival in packing boxes for a long time, and such containers offer infection every season unless periodically washed with fungicide. For this purpose baskets, boxes, etc., should be sprayed with a strong jet of steam or cleansed with a solution of sodium hypochlorite (having about 0·4 per cent. available chlorine) ; the fruit may also be steeped for a short period of 1 minute in the same solution [2, 3, 14].

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Apple Canker, *Nectria galligena* Bres.

Canker is, no doubt, one of the most serious diseases that affect apple trees. It is common in all fruit-growing districts of northern Europe, north-east and north-west parts of the United States, south Canada, Australia, New Zealand, and other areas [4, 12, 18, 19].

Trees of all ages may be attacked, including standard and bush kinds, cordon, and nursery plants. Trees which may show no visible signs of the disease at planting, but which later, perhaps a few months or the summer following, prove to be infected, may be lost during the first or second year through canker [14].

Early signs of the disease occur most frequently at leaf scars, or around the nodes, or at the base of small branches or spurs. The most serious cankers are those which develop at the crotch, where they may cause the death of several branches, if not of the entire tree [10]. An incipient canker developing at a leaf scar or around a wound begins as a small spot, at first only slightly darker than the healthy bark, but soon turns into a dark, purplish-brown area. The spots measure from 2 to 3 mm. in diameter and may increase up to 15 mm. By shrinkage of the covering tissues, young cankers develop a rugged appearance, the bark becomes sunken, and the area gradually extends so as to girdle the twig or spur, the depressed area by now showing a more or less concentric arrangement of ridges on its rugged surface. Infection, moreover, causes hypertrophy of the tissues around the developing canker, and when the overlying bark eventually gives way, deep cracks are formed which often reach down as far as the wood. It is due mostly to increasing exposure and drying of the woody tissues and those of the vital phloem and cortex, in the vicinity of the cracks, that progressive cankers become so ruinous to the trees (Fig. 339). Whilst the foliage itself is not directly attacked, considerable loss of leaves and fruit spurs follows upon a die-back of branches due to girdling cankers, especially if the latter occur low down on the trees.



FIG. 339.—Apple canker (*Nectria galligena*). A severely cankered branch (photo by Dillon Weston). Inset, a cluster of the red perithecia on a branch (× 24) (photo by Radford)

Apple canker is caused by *Nectria galligena* [1, 2], a member of the Pyrenomycetes. The same fungus causes similar cankers on pear trees, and the fruit of both trees sometimes suffers from a form of 'eye rot', causing it to become dry

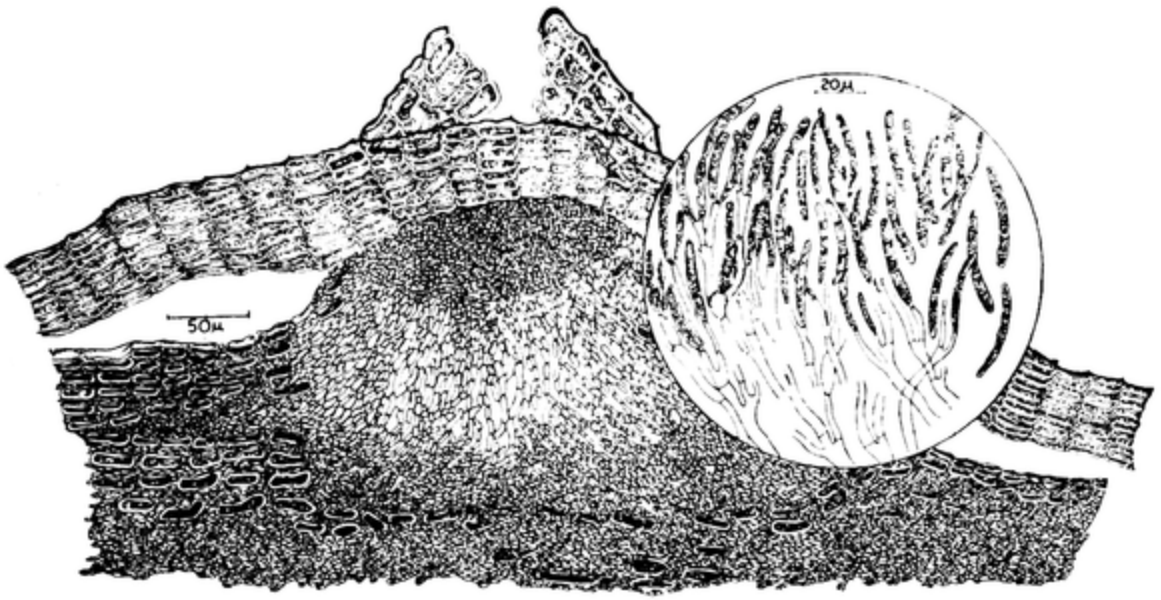


FIG. 340.—Apple canker (*Nectria galligena*). Cross-section apple stem showing early formation of a conidial lesion; the deep-seated mycelium disorganises the phelloderm and becomes aggregated into dense masses below the cork layers which are broken through (the section has not quite passed through the break). Inset, showing the formation of the massive sporodochium elements, from the dense cushion, and the formation of the septated conidia (from a slide made by Smith)

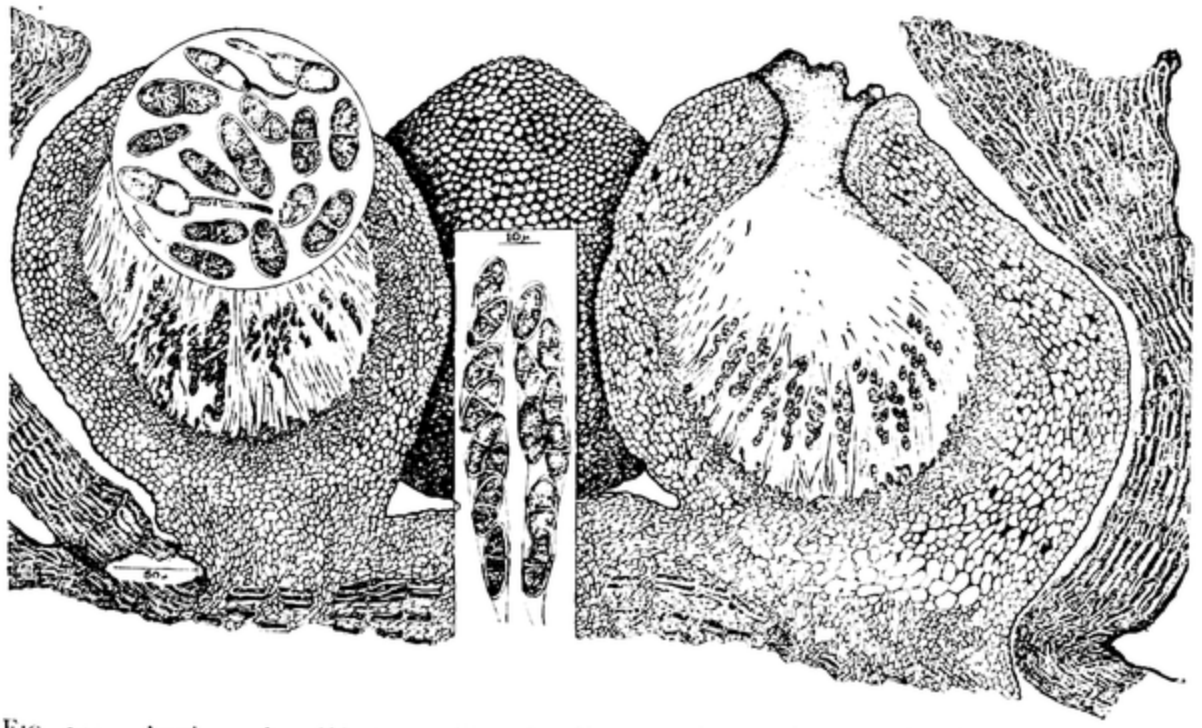


FIG. 341.—Apple canker (*Nectria galligena*). Cross-section apple stem showing three perithecia arising from mycelium beneath the bark. Insets, in circle, the ascospores, some one-celled, but mostly two-celled, with early germination; at centre, two asci with spores (from a slide made by Jamieson)

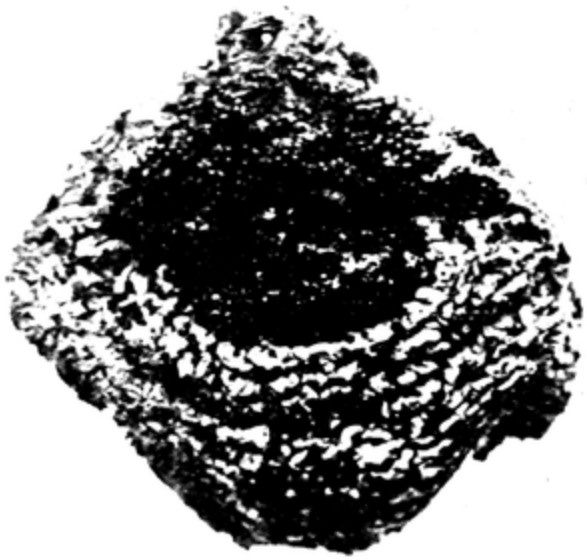


FIG. 342.—Perithecia of apple canker on a mummified apple (variety Worcester Pearmain) (photo by Dillon Weston, *Trans. Brit. Myc. Soc.*)

and mummified, believed to be due to this same organism [6, 7]. There are two types of fructifications, the conidial (*Fusarium willkommii*) and the perithecial (*Nectria galligena*). During late winter or early spring, many of the small cankers occurring at leaf scars on the previous season's wood, may be seen furnished with creamy pustules of conidia, frequently in concentric arrangement around the lesion. Warm spells in March and April favour their appearance and conidia may continue to develop as far on as October, if the weather is humid and warm with frequent rainfall [11]. Conidia examined from the cankers (Fig. 340) are cylindrical, hyaline, slightly curved, usually 5- to 7-septate ; they measure from 65 to 75 by 4 to 5 μ ; in culture, much smaller, hyaline, ellipsoid microconidia, 5 to 7 by 1 to 2 μ, are also formed, but so far as is known they are functionless [19]. On older cankers and on the ridged and cracked dead wood, bright red clusters of perithecia break through the bark from October onwards (Fig. 339, inset), and are usually fairly abundant in December if conditions are moist, but dry weather retards their appearance ; they are not numerous on small cankers on young wood. Perithecia may occur singly, or in dense groups, but with no definite stroma ; they are ovoid to pyriform, 300 to 450 by 250 to 350 μ (Fig. 341) ; asci, numerous, clavate, 8-spored, measure from 85 to 120 by 10 to 16 μ ; ascospores, hyaline or slightly yellow, 2-celled, often constricted at the middle, vary from 11·5 to 22 by 5·5 to 9·2 μ (mean, 16·3 by 7·5 μ) ; the ascospores emerge through the ostiole in white or buff-coloured tendrils [3]. Conidia and perithecia may also be found on fruit affected with ' eye rot ', and perithecia (Fig. 342) have been found on shrivelled apples that have over-wintered on the tree [8]. Discharge of ascospores, beginning about December, depends a great deal upon the degree of rainfall, is usually at its peak in February, declines to a minimum during September, and then gradually rises again in October and November [11]. It is evident, therefore, that the trees are surrounded with infective material, conidia or ascospores, practically all the year round, dormant period included.

Infection may be caused both by conidia and ascospores. Entry is effected through wounds in the bark, or through lesions previously made by the ' scab '-fungus (*Venturia inaequalis*), and especially through imperfectly protected leaf scars (Fig. 106), soon after fall of the leaves [14, 15, 16]. The spores, dispersed by splashing rain and wind, and perhaps by birds and insects, germinate on unprotected wounds or in tiny cracks in the leaf scars. In due time infection reaches the stem, and after much destruction of cortex and phloem, the fungus attacks and destroys portions of the cambium, often far beyond the area defined by the

limits of the future canker. In the parenchyma of the cortex, deprived of its starch contents consequent upon infection, the fungus is inter- and intracellular, and after crossing the phloem and cambium it passes into the woody cylinder by penetrating the living cells of the medullary rays. Though not actually invaded by the fungus, the lignified tissues become heavily browned far beyond the limits reached by the fungus in the parenchyma [19]. As more and more of the cambium becomes involved in infection, the wider and deeper the inroads into the wood become; inoculations with spores inserted into freshly wounded cambium are followed by much more severe cankering than when performed on natural lesions, such as cracks in leaf scars; and there is no period from October to March at which infection fails on newly made wounds [8].

Canker is a disease of damp climates. The trouble is more prevalent in the west than the east of England, largely on account of climatic conditions [11]. Though conidia and ascospores germinate over a wide range of temperature, from 2° to 30° C., they are sensitive to desiccation, but short intermittent periods of dry weather are not a check to the disease. The optimum for growth of the fungus lies between 18° and 24° C.

Canker is much more prevalent on low-lying sites, on clay sub-soils, in water-logged acid soils, and in soils rich in nitrogen but deficient in other minerals, than in moderately fertile, non-acid permeable soils [4, 13]. In culture, the optimum acidity for growth of the fungus lies between *p*H 4·2 and 5·2; the reaction of sap obtained from fresh bark varies from 4·2 to 5·0, values which closely approximate the requirements of the organism in culture [19].

There is no clear evidence that the type of root-stock has any considerable influence on the susceptibility of the scion to canker [14]. It is recorded at East Malling, however, that the varieties Cox's Orange Pippin and Stirling Castle turned out to be more susceptible when worked on root-stocks 'XIII' and 'XVI' than on any others [9, 10].

The varieties Lord Suffield, Cox's Orange Pippin, Warner's King, Worcester Pearmain are very susceptible to canker; Bramley's Seedling, Lane's Prince Albert, and Newton Wonder are more resistant; among cider varieties, Ellis's Bitter, Royal Wilding, and Silver Cup are very resistant, while Kingston Black and Cap of Liberty are very susceptible [18].

Trees heavily cankered should be destroyed. Others less affected may be saved if cankered branches are cut back beyond any trace of brown discoloration in the wood, the wounds being suitably protected. All prunings and shrivelled fruit should be collected and burned. Strict attention to cultural methods are especially desirable since infection is practically never absent from the orchards, and the method of 'grassing-down', to reduce vigour, helps to reduce susceptibility to canker, though in the case of younger trees and nursery stock strong growth is essential from the start [8].

Protection against canker by spraying is directed chiefly against the activity of leaf scar infections. The selected fungicide must have good adhesive and wetting properties; Bordeaux mixture, in the proportion of 8 lb. copper sulphate, 16 lb. hydrated lime, with 4 lb. casein and 1 gallon of petroleum oil, in 100 gallons of water, gives good results. It is usually applied first in April, and again in the

autumn as soon as possible after leaf fall. For the protection of pruning cuts and wounds the parts should be painted over with a paste made of 5 grams of monohydrate copper sulphate, 10 grams of hydrated lime and 9 ml. of boiled linseed oil. Pruning operations should be carried out during dry, frosty weather, when conditions are not favourable for spore-dispersal; late pruning, as in March, prolongs susceptibility as the wounds are slower to heal at that time, and with the advent of spring, rising temperatures become more and more favourable for spore germination [8].

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Bitter Rot of Apple

Glomerella cingulata (Stonem.) Spauld. & von Schrenk ; and *Gloeosporium album* Osterw.

Numerous fungi attack apples during growth and storage causing considerable losses and wastage [4, 5, 9, 11, 14, 26]. Those concerned in ' bitter rot ' are *Glomerella cingulata*, a member of the Pyrenomycetes, better known in its conidial stage as *Gloeosporium fructigenum* [18] and, to a lesser extent, a related species *Gloeosporium album* [15]. The former species has a wide range of hosts which include apple, pear, quince, grape-vine [2, 23], lemon [16], flowers and fruit of the mango [22], coffee [20, 21], privet [12, 13], and many others [2]. But it is by no means clear whether the fungus on all these hosts is *G. cingulata*, and other species attached by some to the genus *Gloeosporium*, and by others to the genus *Colletotrichum*, are also said to be implicated [19]. Bitter rot is believed to be of American origin [2], and in the southern parts of the United States it causes great destruction in apple orchards during warm, moist seasons [1, 2, 3, 6, 7, 8, 10, 17].

Apples are liable to be attacked during all stages of growth, and the rot may continue in storage. In Britain the trouble is apparently confined to the fruit (Fig. 343), but in America there is also a cankering of the branches. The leaves are not usually affected. Early spots or blisters on the fruit are small, brown, and rapidly increase in size up to 1 inch in diameter if relatively few in number, and

up to about $\frac{1}{8}$ inch if numerous and small. As the spots increase in area or become joined by coalescence, infection spreads into the interior of the fruit in the form of one or more cones of rotted tissue, and an entire fruit may become involved, or a cone of decay may develop no further and dry out, leaving a cavity covered over by the dried and blackened skin [10]. In this way affected fruits may become mummified while still on the tree, or in store, and are the chief means of infecting sound fruit.

The acervular fructifications appear in great number in more or less concentric groups over the sunken areas of the fruit and finally break through the skin as small black points exuding pink beads or tendrils of conidia embedded in mucilage. The conidia are colourless, cylindrical, unicellular, and measure from 15 to 30 by 5 to 6 μ [23]. The spots caused by *Gl. album* are smaller than those due to *Glomerella*, brown, and, under moist conditions fringed with white hairs ; they appear mostly around the lenticels ; the conidia are oblong, slightly curved, from 18 to 27 by 2·5 to 4 μ [11, 15]. Perithecia of *Glomerella cingulata* have been seen on old fruits and cankers [18], and in culture [3] but are very infrequently found in nature ; in the cankers they are grouped and sunken ; the asci measure from 55 to 70 by 9 μ ; the ascospores are similar to the conidia, and range from 12 to 22 by 3 to 5 μ.

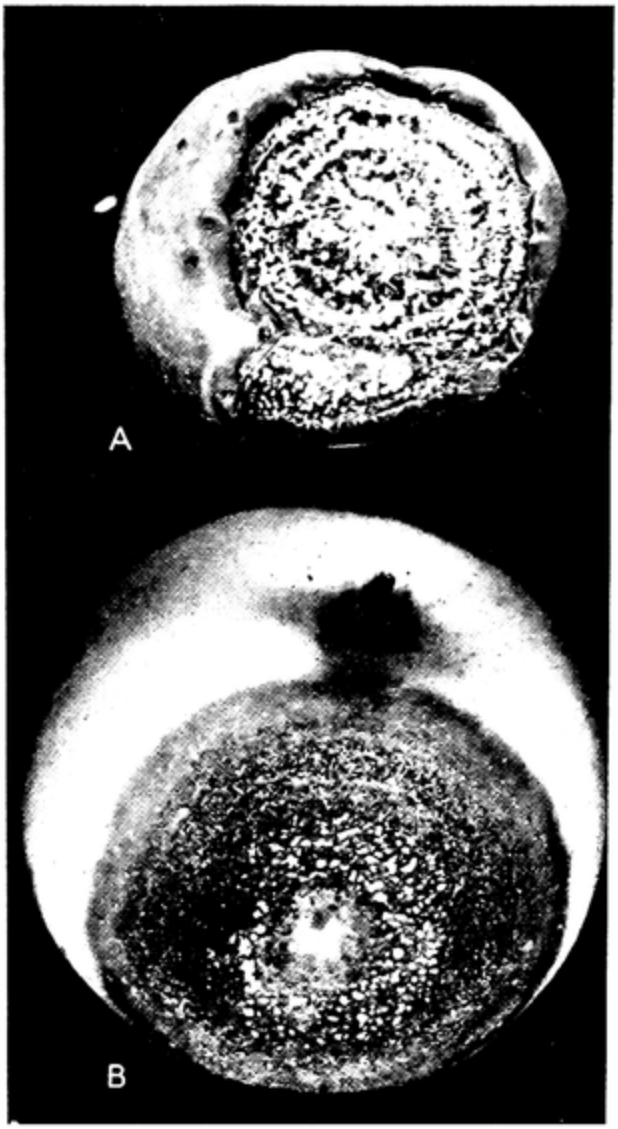


FIG. 343.—Bitter rot of apple (*Glomerella cingulata* and *Gloeosporium album*). *A*, the rot due to *G. album*. *B*, due to *G. cingulata* (photos by Wilkinson, by permission of Long Ashton Res. Station)

Cankers on the branches arise through wound infections, often around the base of dead twigs [2]. They are black, sunken in the bark, roughly oval in outline, old cankers showing cracks or fissures running parallel with the edges, giving the canker a zoned effect. The mycelium which survives in the cankers over the winter forms acervuli similar to those found on the fruit lesions, and the spores are exuded in tendrils in the same way. The fungus also hibernates in mummified fruit left on the tree, or on the ground, and is stimulated to growth when the warm season returns, giving rise to conidia which are mostly dispersed in rain-drops from mummified fruits on to sound fruits on the tree, or carried by birds or insects from the rotted fruits on the ground.

Conidia germinate on the fruit with the production of appressoria, and infection

is probably through small abrasions or by way of lenticels. Unopened pustules about to give rise to acervuli are filled with a dark-olive mycelium which forms a dense array of short conidiophores ; the conidia, hyaline singly, pink in the mass, are abstricted in great number, and are exuded at the ostiole in mucilaginous tendrils.

For the control of bitter rot, spraying the trees three times with 4 : 4 : 40 Bordeaux mixture at intervals of 10 days is recommended, the first application being given when the last of the petals are falling [1]. Mummified fruits should be collected and destroyed and all cankered wood cut out. To avoid bruising of twigs and branches, all pruning operations should be carefully performed, and hand-picking of the fruit will ensure freedom from injuries and so prevent the spread of the disease in storage.

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Apple Scab, *Venturia inaequalis* (Cooke) Wint.

Apple scab is probably the commonest of all diseases of the orchard. It occurs throughout Europe, and in America is prevalent more in the northern than the southern parts of the United States ; it occurs also in southern Canada, Australia, New Zealand, South Africa, Cyprus, Malta, and India [3, 6, 23, 39, 41, 42, 52].

The disease attacks the leaves, shoots, buds, and blossoms, as well as the fruit. In its familiar form of dark spots on the fruit it renders the apple very unsightly, and is responsible for considerable deterioration of fruit in storage, especially as the lesions allow the spores of common moulds, such as those of ' blue mould ' (*Penicillium expansum*, p. 721) and ' pink rot ' (*Trichothecium roseum*) to enter and cause decay [3]. Moreover, following upon severe infection of the foliage, disease and defoliation result in a weakening of the trees and premature dropping of the fruit [7].

Symptoms on the leaves consist of a number of scattered, roughly circular, brown or olive-green spots around which a dendritic margin (like fine branches) may be seen under the cuticle, due to fungus-mycelium radiating and branching all around the spot (Fig. 344 B). Later, the spots turn grey and necrotic in parts, the latter sometimes dropping out; or, the local effect on the leaf may be a malformation, thickening, and puckering of the lamina around the infected areas which then assume a brown velvety texture. But leaf symptoms vary somewhat on different kinds of apple trees, and sometimes the leaf may be covered in parts with irregular, diffused spots or develop a blistered effect due to inequality of growth below the spots, or the blade may appear as if scorched. Spots may arise at either surface of the lamina, but on the youngest leaves as they emerge from the bud, the exposed lower surface becomes affected first.



FIG. 344.—Apple scab (*Venturia inaequalis*). *A*, healthy (left) and scabbed (right) wood of Wellington apple. *B*, under side of leaf of variety Bismarck, with blotches of the conidial stage on blade and midrib. *C*, scabbed twigs of variety Cox's Orange Pippin (photos by Salmon & Ware, Wye Reports)

A close examination of the tips of some of the branches in spring (sometimes earlier) prior to leafing may show on many varieties of the trees (not on all) small spots on the bud scales and on adjacent parts of the stem itself, and when flower buds are showing, similar spots may be seen on the protecting sepals as well. These bud-scale and shoot lesions play an important part in carrying the disease over from year to year; they are further discussed below. When the trees are in flower, lesions may be seen not only on the sepals but also on the flower stalks, and if severe these may girdle the stalks and cause flowers or young fruit to drop.

The fruit is the part to suffer most from the ravages of scab (Fig. 345) and it may be attacked at any time during its growth (58, 60). Early signs of the disease on young fruit are of smaller, much darker spots than those on the leaves, but they have the same kind of dendritic margin showing under the cuticle. Early fruit lesions usually bring about much deformity of growth, often causing the apple to crack, and the fruit remains small. As the spots on young fruit keep pace with its growth and develop spores, they act as additional sources of infection, and spores discharged from them, or from other parts of the tree, are deposited on other portions of the fruit, causing fresh spots to appear. It is believed that all infections which sooner or later appear on the fruit are contracted whilst the fruit is on the tree. As the fruit ripens the spots vary greatly in appearance, mostly according to the variety of apple, some growing apace and sporulating, but in general, towards the autumn fruit spots remain small, black, or chocolate-coloured, and the cuticle covering them usually remains unbroken though may sometimes be slightly abraded (51). After harvest there is no further spotting of the fruit, and what are



FIG. 345.—Apple scab. A number of young fruit and a mature one severely affected (photos by Salmon & Ware, Wye Reports)

apparently fresh spots which break out after picking, or in storage, result from late infections which had already been contracted on the tree. Lesions which appear on infected apples placed in storage assume most diverse forms; they are generally small, pitted or saucer-shaped, brownish black, black, or dull grey, and are often collected on that half of the fruit near the stalk, few appearing towards the calyx end, the explanation being that the stalk-half, being uppermost on the pendent fruit on the tree, is liable to hold drops of water favourable to the retention and germination of any spores that may have collected there. Whilst early lesions on fruit on the trees usually occur near the calyx end, and are naturally the first to become healed over with cork after sporulation has finished, other stalk-end infections usually occur too late before picking to be checked by cork formation, with the result that they continue to grow in storage [51].

Apple scab is caused by the Ascomycete *Venturia inaequalis*, a member of the Sphaeriales; there are two stages in the life-history, viz. the more important conidial phase (*Fusicladium dendriticum*) on all the affected living parts of the host, and the less significant perithecial, *Venturia*-stage occurring on over-wintered leaves on the ground. The conidial stromata are sub-cuticular (Fig. 346 B); the brown, wavy conidiophores, 50 to 60 by 4 to 6 μ, abstrict from the apex, ovoid, olive or olive-brown conidia 28 to 40 by 7 to 10 μ in culture, as many as 4 or 5 conidia may arise at or near the top of a conidiophore, but on the host they are developed one at a time [8, 11]. Conidial pustules on the fruit show the dark olive conidia surrounded by a fringe of white, torn cuticle.

Before leaf-fall, the mycelium of the sub-cuticular stromatic tissue that gave rise to conidiophores and conidia on the leaves extends itself into the mesophyll (Fig. 346 A), where, chiefly in parts of the spongy parenchyma it proceeds to lay down primordia destined to become perithecia, but the latter do not complete their full development until the leaves have over-wintered on the ground (Fig. 346 C). Perithecia are fairly common, and though known elsewhere for a long time, were first found in England in 1923 at Maidstone [46]. On leaves kept in the dark, they are said to develop abnormally [19].

These fructifications appear as small, dark-brown or black pimples embedded in the leaf, opening by a short beak towards either side of the leaf which happens to be uppermost on the ground [11, 20, 26]; they are spherical, 90 to 170 μ in diameter; the 8-spored asci lengthen considerably when mature and so carry the uniseriate ascospores well up into the ostiole for discharge; the spores are pale-green, 2-celled when mature (the upper cell is the smaller), and measure from 12 to 15 by 6 to 7 μ.

V. inaequalis is a complex of numerous strains highly variable in morphological and physiological characters [40]. Monoconidial cultures collected from a number of apple varieties cultivated under uniform conditions showed a striking degree of pleomorphism as exhibited by the type and rate of mycelial growth, its colour, degree of sporulation, and appearance of mutant forms [44]. Furthermore,

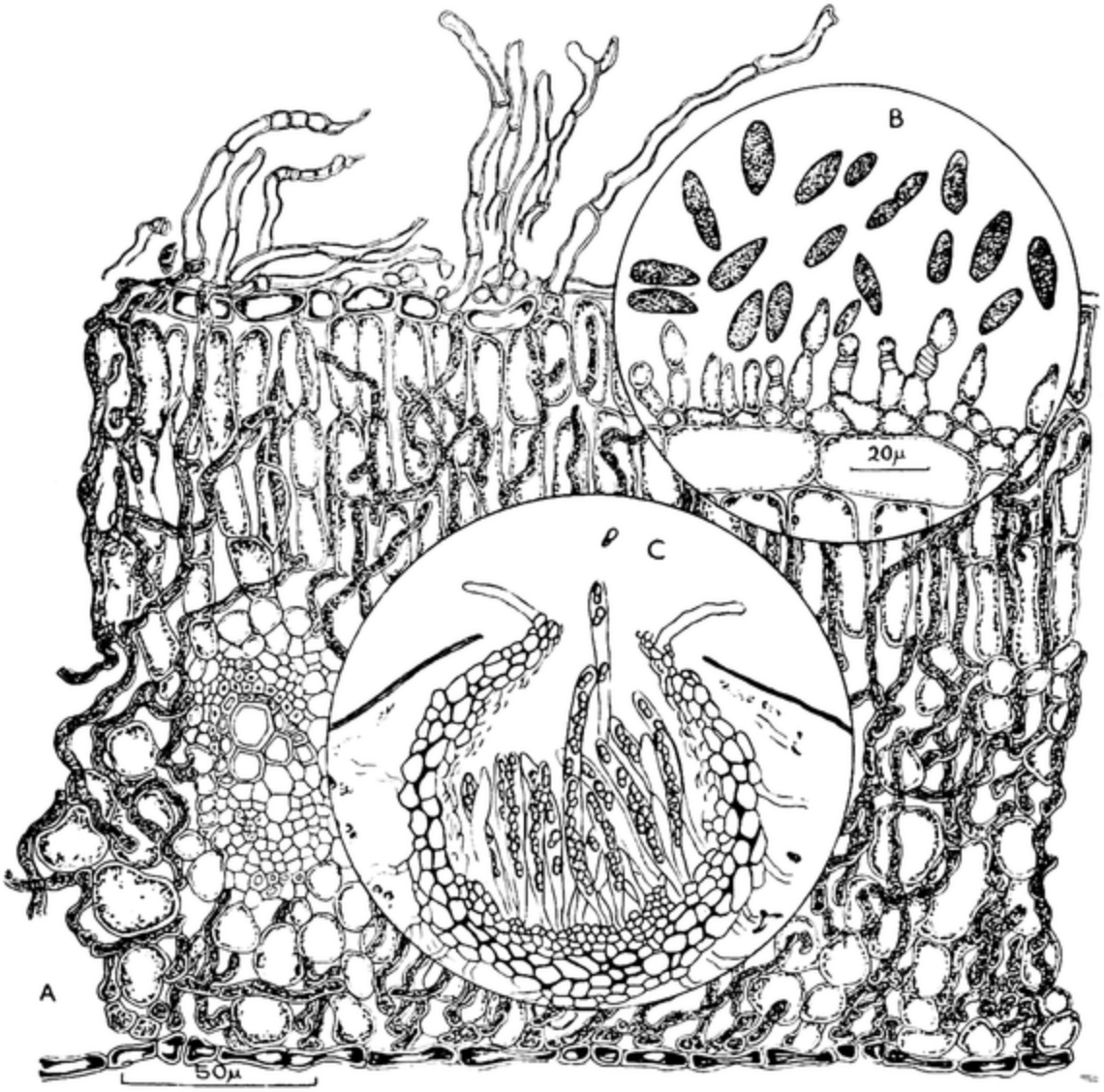


FIG. 346.—*Venturia inaequalis*. *A*, transverse section apple leaf, soon after leaf fall, showing a number of defunct conidiophores, but with the mycelium now invading the mesophyll, occupying the intercellular spaces chiefly but some of the cells as well, in preparation for hibernation on the ground. *B*, portion from a leaf section showing the sub-cuticular mycelium at time of active formation of conidia, whilst the leaf is still on the tree; at this time the mesophyll is practically free from mycelium. *C*, section of a perithecium, within a decayed leaf on the ground, showing asci and spores. (*A*, *B*, orig., *C*, after Wallace, *Cornell Univ. Bull.* 335)

numerous isolates of ascospores which were carried through repeated monoconidial transfers showed the existence of ' groups ' of strains ; some groups were sexually compatible, others self-sterile, others intra-group sterile or inter-group fertile, and other groups differed from their parent isolates in degree of pathogenicity, and one type even produced white lesions on the leaves [2, 24, 25, 48].

Conidia and ascospores are capable of causing primary infections of the trees in the spring. In the British Isles it is the general observation that the conidial pustules which remained protected over winter on the young one-year-old shoots, or on the bud scales, provide the earliest sources for the spring infections [32, 33, 47] (Fig. 93); in some localities primary infections have been found well established long before the perithecia on the ground had begun to discharge their spores [7, 15, 28, 29]. But the ascospores also give a high percentage of primary infections in many localities and in certain seasons, according perhaps, to meteorological conditions [27, 46, 57] they are the principal source of infection [21, 23, 41, 53, 57]. In certain parts of America [12, 20, 23, 55], Germany [27, 57], and New Zealand [41] the critical periods of infection coincide with the incidence of ascospore discharge. Drought in early spring checks the development of the perithecia on the ground, and the disease under such conditions may break out later than usual ; on the other hand, it may appear earlier if the weather is wet in early spring, though intermittent periods of wet and dry are equally favourable to perithecial development and discharge of ascospores. Moreover, depending on the mildness of autumn weather, or on varieties of the trees such as retain their foliage longer into the autumn than others, a delay in the fall of the leaves, involving therefore a delay in the ripening of the perithecia, may in some cases account for a later appearance of the disease in the spring [55]. It is obviously of importance in connection with the forecasting of ascospore discharge and the right time for spraying against scab to determine the first ' puffing ' of the spores from the perithecia. (This can be detected if the fallen leaves are breathed upon and examined, back to the light, with a good hand lens.) Matured ascospores protected within the perithecia can tolerate dry conditions and retain their vitality for several months [53].

Sporulation (whether of conidia or ascospores) is generally active during April and May, when the buds are unfolding. According to the locality and weather conditions, however, the over-wintered pustules on scabbed twigs and bud scales may produce conidia as early as February, and the bud scales may continue to be infective until they drop off in early summer.

The conidia are easily dislodged from their pustules on twigs or bud scales and may be distributed in splashes of rain, or the young leaves may become infected from them by direct contact with the pustules ; aphides are also suspected of carrying the conidia on their hairy legs [7]. From the primary lesions eventually set up, mainly on the leaves of flowering spurs, secondary infections of foliage and fruit take place during wet periods on a wide scale.

Conidia are capable of germinating over a range of temperature from 2° to 30° C., with an optimum at 22° ; the ascospores have a narrower range of 10° to 18° C. [1, 55] ; the optimum for infection with conidia lies between 8° and 10° C., and for ascospores about 19° C. [57] ; ascospores were found to mature slowly in leaves kept at low temperatures of 4° and 7° C., developed better at 12° and 16° C.,

with an optimum near 20° C., but at 24° C. their formation ceased; the perithecia may, however, discharge their spores over a wide range of temperature, from 0·5° to 32° C. [23].

Histological studies of early infections on leaf and fruit have been carried out with conidia and ascospores [39, 56]. Only young leaves are susceptible, and late-season infections have practically no effect on mature leaves. The young leaf must be thoroughly wet, prior to infection, and penetration, which is direct, may occur at either surface. The germ-tube forms a flange-like appressorium which is held fast to the leaf by a mucilaginous sheath, while a narrow penetration-hypha from it is pushed through the cuticle. When the entering hypha is checked by the epidermis it becomes expanded to form an irregularly shaped primary hypha [39]; from the latter there soon develops a small stroma in contact with the epidermis below, and numerous hyphae spreading in all directions from the stroma advance by wedging between and lifting the cuticle from the epidermis (Fig. 110 F-K); then, the mesophyll tissue immediately below the point of entry, apparently to check further invasion, cuts off a layer of cells in the manner of a cork cambium so as to delimit the lesion, and finally with further elaboration of the stroma, conidiophores and conidia are developed as already described. Fruit infections take place in much the same way, the conidial stromata being laid down below the cuticle (Fig. 110 A-C).

Provision to tide the fungus through the winter soon becomes established at the tips of the current season's shoots and, later, on the buds in the axils of the leaves. For the formation of these over-wintering lesions, conidia or ascospores settle on the cuticle and penetration is effected in much the same way as described above for leaf infections, and the process must, of course, be initiated before the epidermis of the young shoot begins to form cork, for the development of primary cork in the apple starts in the epidermis. As in the leaf, a sub-cuticular stroma is laid down in the twig, but here the epidermis appears to collapse and suffers more injury than in leaf infections, so that in the twigs the cells of the epidermis become more or less filled with the fungus. During the winter the mycelium in the twigs continues to spread between the collenchyma and the bark to form a series of subsidiary infections (Fig. 122). All the pustules on the young twigs are thus entirely protected throughout the winter, and as above stated, are of great importance in the initial outbreaks of the disease in the spring. Like the leaf lesions they are delimited by a cork layer, and eventually are sloughed off [32]. There are, however, varieties of apple trees which appear not to be susceptible to twig infections, and it is probable that these kinds receive their first infections from ascospores. Bud-scale infections are initiated towards the end of the growing season. Young buds become infected by conidia from affected leaves being washed down the leaf stalks to collect in the axils, and towards the autumn the tender scales are penetrated in the same way as a leaf or shoot, the stromata being developed mostly in the outermost scales, and even extending into the soft tissues of the bud axis at the base, so that these infections bear close similarity to twig infections. Bud-scale infections remain protected usually until March and open to emit the conidia during April and May when leaves and blossom are unfolding.

Humid, cool weather in spring and early summer is highly favourable to the

incidence of scab disease. Densely foliaged trees are frequently heavily infected because leaves and fruit remain moist longer [52]. Fruit infections take longer to incubate than leaf infections, and longer periods are necessary as the fruit develops. In storage a high degree of humidity is the most important factor favouring increase of disease [3]. A temperature of 0° C. does not inhibit the growth of the fungus in infected apples in storage; at 5° to 8° C., in an atmosphere of 80 to 90 per cent. relative humidity, infection was shown to be equally intense on paper-wrapped apples as on exposed fruit [10, 50]. No definite case has been established that sound apples in contact with scabbed fruit contract the disease, and it is not believed that fresh infections can occur from the presence of conidia in the air of the storeroom though some assert that these possibilities should not be excluded [43].

Several complex factors appear to influence the relative resistance of the host to this disease. Some of these involve the relative thickness of the cuticle, rapidity of cork formation in the young shoots and below the lesions, chemical nature of the sap, concentration of tannin, influence of root-stock and scion, manurial treatment, strain of fungus, etc. [21, 22, 27, 35, 37, 49]. While it is well known that different varieties of apples vary considerably in resistance to scab, this property is not constant under all conditions, and all varieties may contract the disease with variable results, in bad seasons [45].

Since primary attacks are often traceable to scabbed twigs these sources of infection should be removed before the pustules break open in the spring [30]. Fallen leaves should be raked together as soon as possible in the autumn and destroyed by burning or ploughed in deeply to prevent spread by ascospores; spraying the leaves on the ground with 1 per cent. helion, or 8 per cent. carbolineum is said to kill the perithecia [19a, 54]. Dust preparations in the form of colloidal copper and sulphur, wettable and flotation, are variously recommended, but in general are not of greater acceptance than Bordeaux mixture and lime sulphur though the latter, being less injurious, is now largely replacing Bordeaux mixture [4, 9, 13, 14, 17, 34, 36, 37, 38, 38b, 42]; in some instances the dusts are claimed to be less injurious than lime sulphur, for varieties which are sensitive to sulphur but in other cases reports of leaf-shedding and fruit-dropping have followed their use [31]. Lime sulphur is also said to reduce photosynthesis [18]. Bordeaux mixture, at a strength of 4 lb. copper sulphate, 6 lb. hydrated lime, to 50 gallons of water, should be applied lightly as a misty spray. A common schedule in Britain, using Bordeaux mixture and lime sulphur, includes four applications at least: (*a*) at the ' green bud ' stage when the flowers are in bud, with Bordeaux mixture; (*b*) at the ' pink bud ' stage when the petals are showing, but the buds not yet open, with the same mixture, or, $2\frac{1}{2}$ per cent. lime sulphur may be used for *a* and *b*; (*c*) ' petal fall ' stage, with 1 per cent. lime sulphur, except for Stirling Castle, Lane's Prince Albert, Beauty of Bath, Newton Wonder, Rival; but this application is safe for Allington Pippin, Worcester Pearmain, Early Victoria, Blenheim, Annie Elizabeth, Lord Derby, Grenadier, Bismarck, and Bramley's Seedling; (*d*) a second, ' post-blossom ' stage, as for ' petal fall ', 2 weeks later. In addition to spraying practice in the orchard, it is advisable to remove all surface moisture from the fruits before storage [59]. In some countries it is recommended that apple trees should be dipped into Bordeaux mixture before export or distribu-

tion to the grower [5]. Spraying operations should be carried out thoroughly and at the right time, this being considered more important than the selection of any of the more promising fungicides [16]; the interval between consecutive treatments should not be more than a fortnight [38 b].

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White Root Rot of Apple, *Rosellinia necatrix* Prill.

This disease is highly destructive to the roots of apple and pear trees, and by early infection of the finer, fibrous rootlets brings about a slow death of the plant. It also attacks the underground parts of potato, narcissus, arum, iris, gooseberry, strawberry, peach, almond, cherry, and other cultivated crops [5, 9, 13].

In Britain, this root rot exists chiefly on fruit trees, and was first described here in 1896 [6]; it was found again in this country in 1913, near Canterbury, on apple trees in a plantation from which old cherry trees had been grubbed up [12]. In the warmer parts of the British Isles it has probably a wider distribution than already known, since it may frequently be passed unrecognised over a long period after attack, before any symptoms of disease appear above ground [8]. The disease has been known in Europe for many years, and was first studied, in 1883, in Germany [4], and later, in 1891, in France [14], in the latter case, on the grape vine on which it still appears to be destructive in vineyards on the Continent. It also occurs in America where it has been found in a number of apple orchards in Santa Cruz County, California, and in at least one apricot orchard in Alamada County [2].

White root rot is caused by *Rosellinia necatrix*, a member of the Pyrenomycetes, but has long been familiar under the name *Dermatophora necatrix* [1, 7, 10].

Infection is confined to the roots, the fungus forming on the finer terminations, strands of snow-white mycelium (Fig. 347). On the older, main roots, however, the fungus is brown or olive green in colour, more matted, forming in parts short rhizomorphs which remain narrow and succulent, but never attaining the firm, shoe-lace type of rhizomorphs seen in root-rot disease caused by *Armillaria mellea* (p. 907). The white mycelium may also extend from affected roots out into the soil, and in this way may spread to the roots of healthy trees growing near. The mycelium consists of branched, septated hyphae which are of a nodulose nature, the constituent cells becoming swollen just below the septa. After the death of the tree the mycelial web covering the roots practically disappears, and its place becomes occupied by a number of small black sclerotia. In artificial culture, but apparently rarely in nature, the fungus gives rise to conidia, but apart from one report [14] that these spores are germinable, the conidia appear to play no part in spreading the disease [3, 4]. The perithecia arise in dense, black clusters on the dead roots, but they are not common and, so far, have not been observed in Britain [8]; they are round, black, brittle, and furnished with an ostiolate neck which disappears after the perithecia have discharged their spores [3]. The ascospores are spindle-shaped, somewhat dorsiventral, showing a distinct longitudinal groove along one of the flat sides through which one or more germ-tubes emerge at germination [3]. The ascospore dimensions, according to various authors, are 40 by 7 μ [14]; 43 to 47·5 by

9 μ [11]; and 31·1 to 47·6 by 7·1 μ [3]. Apparently the ascospores germinate with difficulty, and it seems doubtful whether these spores again, like the conidia, play any part in the dissemination of the disease. They have, however, been seen to grow on potato dextrose agar, at 22° to 24° C., after a preliminary treatment with lactic acid, and produced the conidial stage; when these cultures were employed to infect the roots of young apple trees, the characteristic symptoms were obtained, and within 6 weeks of inoculation the trees died [3].

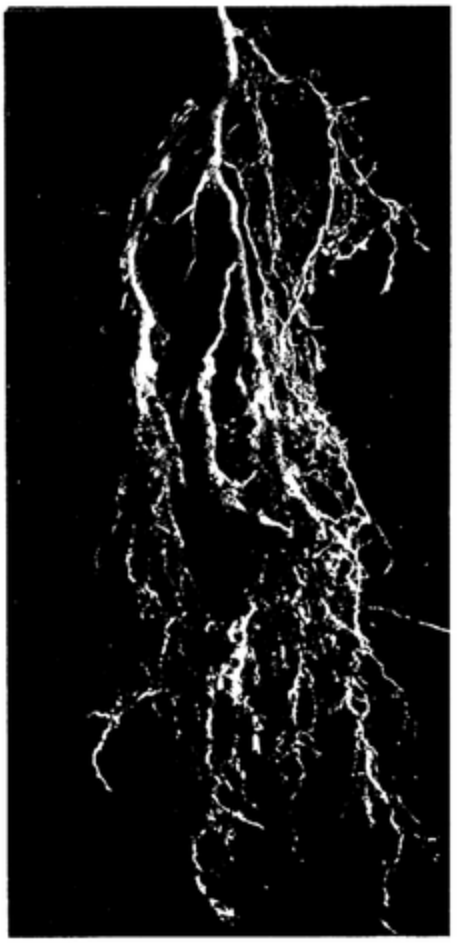


FIG. 347.—White root rot of apple (*Rosellinia necatrix*). A rooting system covered with white strands of the fungus (photo by Nattrass)

The fungus survives on old roots left in the ground, and infections have been known to take place after young trees had been planted in land which had previously supported gooseberry and cherry trees suffering from white-root rot. In Britain, at least, the fungus also survives from season to season in the form of sclerotia in the soil or adherent to root debris. During wet seasons mycelium is early developed, especially in heavy soils of high temperature [6]. The mycelium developing in the soil consists of very fine, narrow hyphae ('exploration hyphae') which travel through the soil and in this way may infect the roots of susceptible trees; in the soil too, may be found greenish-grey to white rather dense strands of the rhizomorphic type of mycelium, sometimes spreading out in fans, these dense aggregations apparently having congregated around bits of old roots or some other organic material in the soil, and the fungus mycelium may also frequently be discovered to occupy minute cavities in the soil, being particularly abundant in worm burrows [8].

The disease may attack the roots of young seedling trees as well as those of mature trees. The parts first attacked are the extremities of the youngest rootlets, and after penetration the fungus travels under the bark into the larger branches of the root by destroying the cortical tissues in its path. The deeper tissues are not invaded, but after the death of the roots the fungus breaks out of the cortical region to the exterior to form the characteristic white mycelium which later gives place to sclerotia, and on rare occasions, near soil-level, to conidia [2], and finally to perithecia. When old trees are attacked, the trouble may exist in the roots for two or three years before any signs of disease are manifest above ground. At first the symptoms are confined to one side of the tree, a sure indication that infection arose from contact of the roots on that side with those of a neighbouring tree, or with infected root debris buried at one spot. The leaves on the affected side of the tree turn yellow and begin to fall off early, and each succeeding season is marked by fewer and fewer leaves, with the result that the fruit fails to mature,

and many branches may also be seen to suffer from a die-back condition. Infection may thus be localised for a long time, and, if the trouble is detected early, young trees may be saved if affected roots are trimmed back and the trees planted in clean ground [8].

To control the disease in the orchard, all badly affected trees should be pulled up and destroyed, care being taken that no bits of diseased roots are left behind. The application of carbon bisulphide to the soil is claimed to be successful as a measure for the control of the disease in vineyards on the Continent.

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Blossom Wilt and Spur Canker of Apple, *Sclerotinia laxa* (Aderh. & Ruhl) forma *mali* (Wormald) Harrison

'Blossom wilt' attacks apple trees soon after they come into flower. Young as well as established trees are liable to the disease, and entire inflorescences with their adjacent green leaves may be killed before the flowers set fruit (Fig. 348). Moreover, in severe attacks the trouble may spread in the same year into the tissues of the short branches (spurs) bearing the withered flowers, killing them and causing a cankerous condition of the bark; if the canker girdles a branch all the length of shoot with the spurs above the canker dies [2]. The same disease attacks the pear, the ornamental red-leaved crab-apple (*Pyrus purpurea*), the white beam (*P. aria*), and the Japanese quince (*P. japonica*), but in no instance as severely as the apple. Stone-fruit trees are also susceptible, the principal hosts being plum (Fig. 348 inset) and cherry, and some ornamental types related to them, namely the Japanese flowering cherry (*Prunus serratula*), the Chinese flowering cherry (*P. tomentosa*) the sand or dwarf American cherry (*P. pumila*), and the bird cherry (*P. padus*).

Early signs of disease are a wilting of the flowers and leaves at the base of a flower cluster, the flowers turning brown as if scorched, and the leaves as they wither curling up at the margin, thus exposing the grey, hairy under surface, and giving the appearance of being covered with mould. But the conidial fructifications of the fungus associated with this disease are usually found on the dead flowers and flower stalks, and not until late in the autumn, or in the spring following infection, are the spores to be found on spurs and cankers. Withered blossom may be retained on the trees throughout the winter, and as the leaves on infected branches are killed before the absciss cork layers are developed in the usual manner

at the base of the petioles, they too may hang on the tree for a considerable time. During the winter, therefore, affected trees are often rendered conspicuous by the presence of withered trusses and leaves left behind long after the healthy trees have cast them off. Affected spurs allowed to remain on the tree for a third year usually dry out, and any cankers associated with them have by that time healed up with callus and rendered innocuous.



FIG. 348.—Blossom wilt and spur canker of apple (*Sclerotinia laxa* forma *mali*). Note the canker on the stem and a dead spur on the right bearing the conidial *Monilia* fructifications (photo by Wormald, *Ann. App. Biol.*). Inset, right, showing 'wither tip' of plum, caused by *S. laxa* forma *pruni* (photo by Foister & Noble)

Blossom wilt of the apple, in Britain, is caused by the fungus *Monilia cinerea*, the perfect, ascigerous stage of which is the Discomycete *Sclerotinia laxa* [1, 8]. The organism exists in various forms; one form attacks the apple, another, the plum; other forms, again, affect other hosts, so that the full designation of the fungus on the apple is *Sclerotinia laxa* forma *mali*; and the form infecting plum trees is *S. laxa* forma *pruni*. The perfect stage has not been seen on apple in Britain, but has occurred on one occasion on dried, mummified plums which had over-wintered on the ground (Fig. 349); it is described below. Some confusion has arisen in later years over the designation of the fungi responsible for brown-rot diseases of fruit trees in Britain and America [12, 13]. The position has now been clarified by the researches of Wormald [14-26] on these diseases in Britain. In this country, the fungus generally attacking the blossom of all hosts above mentioned is *Monilia cinerea*, and the species attacking the fruit is *Monilia fructigena* [3] (described below), but the former has also lately been found to cause a black rot of apples and pears [26a]. The American fungus is deemed to be a different species, namely, *Sclerotinia fructicola* (briefly described below); it does not occur in Britain.

Numerous strains of *M. cinerea* and *M. fructigena* arise in artificial culture, and while striking differences may be seen even between strains of the same organism, such differences are not constant; but strains of *M. cinerea* have been isolated which were biologically distinct from strains collected from the stone-fruit hosts, and strains from the latter were obtained which did not produce blossom-wilt in the apple [11, 21]. Moreover, *M. cinerea* from pear spurs infected apple blossom and also plum fruits, and the same organism isolated from plum twigs attacked pear flowers; and isolates from cherry fruits infected pear flowers with production of blossom-wilt [3, 9, 11, 21]. Differences in mode of growth and in degree of sporulation between *M. cinerea* and *M. fructigena* may be seen when cultured on prune-juice agar or steamed potato; it is noteworthy

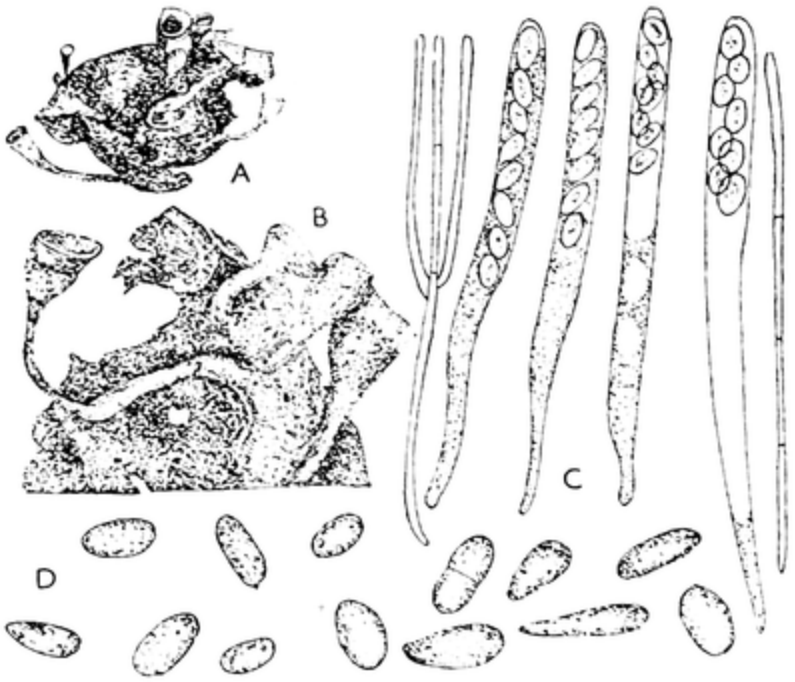


FIG. 349.—*Sclerotinia laxa*. *A*, mummified plum bearing the stalked apothecia of the perfect stage, two-thirds natural size. *B*, four apothecia, *in situ* (× 2). *C*, asci and paraphyses (× 330). *D*, ascospores showing variation in shape and size (× 700) (after Wormald, *Ann. Bot.*)

that while neither of these two organisms produced normal conidia on prune agar, both produced clusters of minute microconidia on this medium, but on the potato medium both fungi produced the normal conidia.

Conidial pustules of *M. cinerea* on the wilted flowers, especially in damp weather, are very luxuriant, consisting of much-branched conidiophores bearing long chains of glistening, bead-like, or lemon-shaped conidia which measure, on an average, 16 by 12 μ [24]. As already mentioned, not until the winter or the spring following infection are the conidial fructifications to be seen on dead spurs and cankers (Fig. 348), when they appear in crevices of the bark as grey, or slightly yellow pustules, about 1·5 μ wide, in which the conidia are smaller than those on the flowers, ranging from 11 to 12 by 8 to 9 μ.

The apothecial fructifications of *S. laxa*, found, as stated above on mummified fruits (Fig. 349) of the plum, are egg-cup shaped, brown, and elevated on long stalks; the hymenium consists of elongated club-shaped asci, from 121 to 188 by 7·5 to 11·8 μ, each containing a row of unicellular, oval, colourless ascospores measuring from 7 to 19 by 4·5 to 8·5 μ [2]. The *Monilia*-stage alone is responsible for the blossom wilt in Britain, and the fungus is carried over from year to year by its survival in spurs and cankers.

Initial infections always take place through the flowers, not through foliage leaves. A flower becomes infected when conidia are deposited on the stigmas and, favoured by damp weather, the growing fungus travels down the styles destroying the tissues in advance, and within a fortnight whole flower clusters with their adjacent green leaves may be browned and killed. From the flowers the fungus passes into the stalks and thence into the woody spur, the tissues of which are again destroyed and browned in advance of the invading fungus, and a brown discoloration may extend even into the branch carrying the infected spurs.

To prevent the invasion of the spurs and wood, and so deprive the fungus of its method of survival over the winter, infected trusses should be removed as soon as they show signs of wilting, but usually this is left too late, and it becomes advisable to cut out all infected wood and cankers, as far back as any trace of brown discoloration remains, making the final cut through clean wood. Where such treatment is impracticable owing to the size of the trees, spraying with lime sulphur, 1 in 50, or with Bordeaux mixture 6 : 6 : 100, just before the blossom

appears, is found beneficial. Other fruit trees and ornamental bushes susceptible to blossom wilt should not be planted close to apple trees. Of the ordinary commercial varieties of apples, Bramley's Seedling is rarely attacked, but the varieties Lord Derby, Cox's Orange Pippin, James Grieve, Rival, and Ecklinville Seedling are amongst the most susceptible kinds [14].

Brown Rot and Spur Canker of Apple, *Sclerotinia fructigena* Aderh. & Ruhl.

Brown rot of the fruit of pomaceous and drupaceous (stone fruit) trees is of common occurrence in the orchard and on stored fruit. It is not a common rot of pomaceous fruits in the United States, but is frequently found there on peaches, plums, and other stone fruits [13].

Apples may be attacked at any age on the tree, and the disease may cause them to fall before ripening [4], or convert them into dry, mummified fruits which may remain on the trees throughout the winter. Serious losses may thus be incurred annually from premature fall of fruit, and in the store the disease may spread, under humid conditions, from affected to sound fruit by contact. The same trouble may be experienced with plums, pears, quinces, and cherry fruits, but apricots and peaches are only occasionally affected.

This disease begins on the fruit, whether on the tree or on the ground, as brown spots which may spread to destroy the whole fruit. During the progress of the rot, small yellow pustules arise under the browned skin, which break through as yellowish or buff-coloured cushions of conidia (Fig. 350). The pustules are usually arranged on the affected part in concentric zones and, presumably stimulated by exposure to light, the fungus produces a fresh zone of spores every day. The disease is not of the nature of a wet rot, and as it advances the fruit becomes wrinkled and mummified. While a great proportion of infected fruit is blown down with the rot continuing to develop on the windfalls, other dry, mummified fruits may hang on the trees through the winter, and both these as well as the fallen fruit may serve as sources of infection of young fruit developing in the summer. Apples, to all appearance sound when placed in store, may develop the rot at the stalk end and turn black, and though the fungus is present in the pulp, only very rarely are the conidial fructifications developed on stored fruit.



FIG. 350.—Brown rot and canker of apple (*Sclerotinia fructigena*). *A*, the rot on the fruit and the canker stage on the stem (photo by Wormald). *B*, conidial *Monilia* pustules of the fungus on the stem of plum. *C*, a mummified plum. (*B*, *C*, photos by Foister & Noble)

Brown rot of apple fruit in Britain is caused by *Monilia fructigena*, the conidial stage of *Sclerotinia fructigena*, but this

perfect stage has, so far, not been found in this country. The buff-coloured conidial tufts, from $\frac{1}{6}$ to $\frac{1}{8}$ inch in diameter, are bigger than those of *M. cinerea*, and the oval conidia are also larger, varying from 12 to 34 by 9 to 15 μ, the average being 22 by 13 μ. The apothecia, asci, and ascospores are similar to those of *S. laxa*; asci range from 112 to 180 by 9 to 12 μ, the ascospores measure from 9 to 12·5 by 5 to 6·8 μ [23]. Mummified apples in Denmark, which had been buried in the soil for 20 months, produced stalked, yellow to grey-brown apothecia, 2·5 to 7 mm. in diameter; the asci measured from 115 to 170 by 7·5 to 11 μ (average 156 by 10 μ), the ascospores, unicellular and ellipsoid were from 11·0 to 13·2 by 5·7 to 7 μ (average 12·2 by 6·5 μ); the conidia measured from 15 to 22·9 by 8·6 to 14 μ (average 19·6 by 11·8 μ) [28]. The fungus tolerates a wide range of temperature, the optimum being about 25° C., and though there is marked reduction of infection below 22° C. [10], it is not checked at low temperatures of cold storage [23]; ordinary summer temperatures are therefore favourable to the development of brown rot.

Infection of fruit is believed to occur through the lenticels in the skin, but chiefly through wounds caused by various insects, or through the action of hail or frost, or by means of cracks due to unequal growth. The sources of infection are the mummified fruits left on the trees, or the fallen fruits left to decay on the ground. The fungus on these revives the next season to form the conidial fructifications which, as already indicated, provide for the primary infections in Britain. Young fruit on the tree may become infected (Fig. 351) by direct contact with a mummy [5], and, doubtless, the conidia are also carried from rotted fruit on the



FIG. 351.—Brown rot of apple (*Sclerotinia fructigena*). Showing infection extending from a mummified apple at *A*, to others *B* and *C*, in contact (photo, Adv. Leaflet 155, by permission of Minis. Agric.)

ground by marauding insects which infect the fresh fruit by biting through the skin.

As in the case of blossom wilt, cankers on the twigs may also be found in association with fruit rot, the fungus growing down through the stalk and thence into the spur and stem. The resulting cankers appear much later than those caused by *M. cinerea*, usually in late autumn.

Infection of apples in storage may take place through wounds in the skin or through the stalk end when the fruit is picked without the stalk. A general browning of the flesh takes place at the seat of infection and the fungus may spread quickly under moist conditions in storage or transit. Affected specimens in storage are usually smooth and black, but hardly ever develop conidia unless the environment is unduly moist, in which case they become covered with mycelium which, moreover, supplies an additional means of infection to sound fruit in contact with it.

Apples possessing high resistance to brown rot are said to have fruit containing sap of a higher *p*H value than that of susceptible varieties ; but with the progress of the rot within the fruit the *p*H was found to increase [10].

Since mummified fruits and infected windfalls are frequently the sources of fresh outbreaks of brown rot, all such materials should be removed from the orchard ; and all infected spurs and cankered wood should be cut out and destroyed.

In America, Australia, and New Zealand [6], brown rot of fruits is caused by a different species of *Sclerotinia*, namely *S. fructicola* (Wint.) Rehm ; the British and European type *S. fructigena*, above described, has so far not been found in association with the disease in the countries named. *S. laxa* does, however, occur both in the United States and Canada, and both *S. fructicola* and *S. laxa* are responsible for blossom-wilt and twig-blighting, chiefly of stone-fruit trees, and may under certain conditions cause fruit rotting as well [7]. The *Monilia*-stage of *S. fructicola* was successfully produced after infection of peach blossom with the ascospores. The apothecia of *S. fructicola* may be found in abundance on mummified peaches hanging on the trees, or on the fallen fruit, and sporulate just about the time the trees come into bloom, but to what extent the ascospores are responsible for setting up primary infections is not known. The asci of this species range from 102 to 215 by 6 to 13 μ, and the ascospores from 6 to 15 by 4 to 8·2 μ. The conidia range from 10 to 28 by 7 to 17 μ, with a mean of 10·46 by 16·24 μ [23]. Microconidia have been observed on mummified fruit, coincident with the development of the apothecia ; they may probably serve as fertilising cells [27].

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Bitter Pit of Apple (*non-parasitic*)

' Bitter pit ' of apple is a common functional disorder of the fruit, and is not attributed to any plant pathogen. First described as ' stippen ' in Germany [34] in 1869, the disorder has long been known as ' Baldwin spot ' or ' brown spot ', and the name ' bitter pit ' was first applied to it in Australia where the trouble is very prevalent, especially in South Australia and New South Wales. It is also well known in the United States, South Africa, and other countries [8, 9, 10, 11, 14, 31].

The trouble appears to arise as a result of picking before the fruit is ripe, and develops more rapidly the more immature the fruit [31]. While the general observation is that bitter pit is a storage disorder, the trouble really begins in the orchard, the symptoms which break out on the stored fruit being traceable to a physiological disturbance during the growth of the fruit [27]. Numerous observers have noted that large-sized fruit from a light crop are more susceptible than smaller fruit from an average-sized crop, but others maintain that the relative severity of bitter pit cannot be explained on a basis of size of fruit alone [1, 27]. It was observed in British Columbia that fruit from trees bearing less than one-third of a normal crop proved to be much more liable to the disorder than fruit from trees carrying more than this proportion [5]. Bitter pit is very erratic in its appearance, for in some seasons trees known to have suffered from it may be quite free, and in others to develop it severely, and trees of the same variety growing close together may develop extensive pitting of the fruit at harvest-time, and little in storage, but, in general, the symptoms appear more rapidly during the first two or three months of storage [13].

Bitter pit is characterised by the presence of a few to numerous depressions on the surface of the fruit, chiefly towards the calyx end (Fig. 352). The spots are circular, sub-circular, or streaky, and may sometimes be barely visible, but commonly measure from 3 to 6 mm. in diameter. Immediately below the spots, brown necrotic areas arise in the pulpy tissues (Fig. 353), but a browning of the flesh, arising as it does from within the fruit, may sometimes not be accompanied by any external pitting. According to the natural colouring of the particular variety

of apple, the pitted spots may be grey-green, olive-green, light brown, dark chocolate-brown, or almost black [9]. Observations in Australia [9] on Cleopatra apples showed fairly constant differences in the symptoms, one form, described as *mild pit*, consisting of small, rather inconspicuous superficial lesions in both cold and ordinary storage, and another form, *severe pit*, characterised by large depressions of a dark colour, developing only in cold storage.

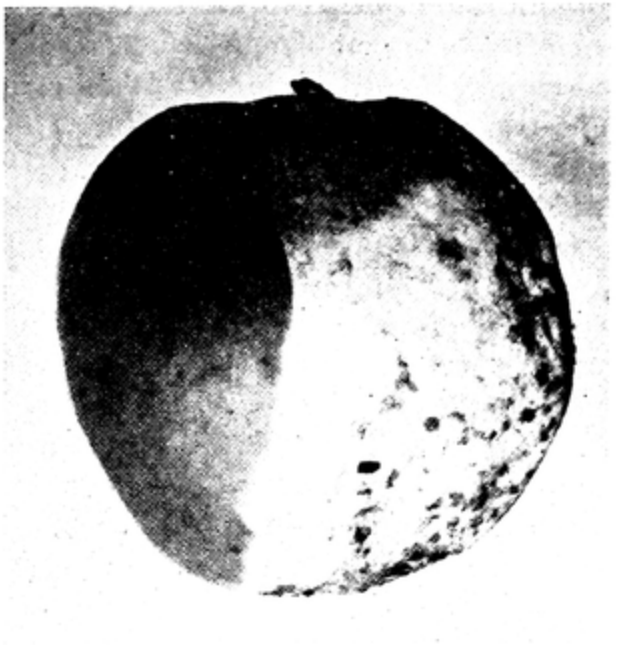


FIG. 352.—Bitter pit of apple. Note the dark spots on the affected right half of the fruit (photo by Foister & Noble)

Numerous theories have been put forward from time to time which seek to explain the cause of bitter pit in apple fruit [3, 6, 9, 12, 17, 18]. Its etiology is still obscure, however, and much remains to be done also on the problem of its control [13]. The probably correct view is that the trouble is due mainly to a disturbance of the water-balance between the leaves and the growing fruit [27] (cf. *Blossom End Rot of Tomato*, p. 678). It has been shown that when single branches of a tree were defoliated, the fruits were less susceptible to pitting, since the competition for water would thus be lessened. The view is expressed that susceptibility to bitter pit is increased by any treatment that raises osmotic values in the leaves, at the expense of those in the fruit. Experiments performed on the trees, such as 'ringing' of the limbs after blossoming, girdling of the flower stalks, drastic thinning, etc., all showed that susceptibility was increased when the osmotic concentration of the leaves was raised at the expense of the fruits [26]. Further, a disturbance of soil-moisture conditions, whereby increased intake of water would occur, especially towards time of fruiting, is also suggested as being a possible cause of an over-stimulation of the fruit during growth [6]. Excessive transpiration during the day and a sharp reduction or cessation of the process during the night, while intake at the roots remains unabated, is also put forward as a contributory factor which, by increasing internal pressure in the fruit, would cause injury to the cells [14]. In addition to an irregular water-supply, an unbalanced nutritional condition may also be conducive to the disease [13]. It is asserted by some that the trouble is associated with the retention of starch in the areas affected, the necrotic spots on the fruit resulting from excessive translocation, followed by



FIG. 353.—Bitter pit of apple. Section of cleared apple tissue showing pits (dark areas) under the skin and their relation to the vascular bundles (after Smock, *Cornell Univ. Bull.* 234)

osmotic action between the starch-filled cells and those in which the starch had been largely or completely changed to sugar [8]. Others have found, however, that starch may be present in both healthy and necrosed cells in affected fruits [20, 25]. The trouble has also been attributed to widely fluctuating temperatures during night and day [17], but this view has not received general support [24].

The necrotic lesions, at first, consist largely of groups of collapsed cells situated beneath the hypodermal layers of the fruit; later, the cell walls become torn. Amongst the disorganised tissues starch grains may be found, and these persist in the affected spots during storage. When the necrotic spots are traced inwardly into the flesh, they are found to be associated with the vascular bundles in the pulp (Fig. 353). In the early stages of the formation of the spots, the overlying epidermal and hypodermal layers remain intact, but later they too collapse when larger portions of the flesh become involved in the lesion. As the disorder works outwards from the pulp to the surface of the fruit, the browned areas may sometimes not be visible from outside, but when the fruit is cut they are found to be associated with the browned strands in the pulp. These histological features are the same in fruits still on the tree or on those in storage [25, 27].

Bitter pit is aggravated by dry conditions and high temperatures during early summer, followed by heavy rains [6, 13, 28, 33]. A great deal still remains, however, to be learnt about weather conditions in relation to this disease. Observations on the effects of light intensities appear to be conflicting. In British Columbia [5] it was noticed that the trouble was severe on Newtown apples in seasons enjoying more than the average amount of sunshine, and was not so pronounced, for the same period, when the amount of sunshine was less. On the other hand, excessive shading of the fruit during growth is said to increase susceptibility to pitting in storage; fruits picked from the centre of the tree, which were much shaded proved to be very susceptible [27].

Susceptibility to bitter pit appears to increase with excessive applications of nitrogenous manures during the growing season [18, 27, 34].

Wastage from bitter pit may largely be obviated by delaying the picking until the fruit is ripe, and the fruit should be stored immediately at a low temperature; storage at 32° F. delayed the appearance of the symptoms and sometimes reduced the development of the trouble [5, 23, 31]. Lightly cropping trees should be picked about a fortnight later than heavily cropping trees [1]. Bitter pit is not associated with any mineral deficiency in the soil, and is not relieved by the application of boron [7, 24, 30].

The varieties Baldwin, Northern Spy, Rhode Island, Greening, York Imperial, and Gravenstein are listed as being very susceptible to bitter pit [27].

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Pear Scab, *Venturia pirina* Aderh.

Scab disease of pears is equally as common as apple scab, which it closely resembles in many features. The fungus causing it is almost identical with that on the apple but the two fungi are distinct species and confined to their respective hosts.

Pear scab is widely distributed in Europe and in the United States [2]; in California [17] it is the most important disease of this host, and in Australia is reckoned to be the most serious factor limiting the export of the fruit [5].

In comparison with the organism of apple scab, the fungus of pear scab is more aggressive, penetrating the host more quickly and more extensively. It also has a greater capacity for spore production, which, moreover, is spread over a longer period than is ordinarily observed with apple scab [9].

The general symptoms of scab on this host are very similar to those on the apple but are more clearly defined. The leaves, twigs, bud scales, flower stalks, sepals, and sometimes the petals and the main axis of the inflorescence, as well as the fruit (Fig. 354) are all attacked in much the same way as above described for apple scab [15]. Twig infections, however, are far more extensive on the pear than on the apple (Fig. 355).

Pear scab is caused by *Venturia pirina* [1], the name assigned to its perithecial stage on the over-wintered fallen leaves ; the conidial stage (*Fusicladium pirinum*) is the more important, parasitic stage on the tree. The conidiophores are brown, septated, and are more zigzag than those of the apple fungus because in addition to a terminal conidium, a number of lateral conidia arise in sympodial fashion, whereas in the apple the conidia are



FIG. 354. Pear scab (*Venturia (pirina)* (photo by Wormald, *Diseases of Fruits and Hops*, Lockwood)



FIG. 355.—Pear scab on the wood. Pustules of the fungus are breaking through at points indicated by the three lines; the base of scale leaf (top line, right) is also cracked open by the pustules (photo by Salmon & Ware, *Grdnrs'. Chron.*)

formed in succession from the apex of the conidiophore. The conidia are dark brown or olivaceous, ovoid, unicellular or 1-septate, 15 to 40 by 6 to 10 μ, in nature; in culture somewhat longer, 15 to 60 μ [3]. The perithecia on over-wintered leaves on the ground were found in England, in 1924, at Wye, on blown leaves sheltering under a hawthorn hedge [14]. These fructifications are smaller than those of the apple fungus, being just visible to the naked eye as dark points, scattered or sparsely grouped at either or both surfaces of the leaf; the asci contain 8 spores; the ascospores, similar to those of *V. inaequalis*, are transversely septated into two unequal cells, but in *V. pirina* the larger cell faces the top, not the base of the ascus as in the former species; the germ-tube is formed from the larger cell. The perithecia usually discharge their spores towards the end of April; a wet spell followed by dull weather with occasional sunshine, and a temperature of 49° to 55° F. are recorded to be the best conditions for good spore dispersion [14].

For the primary spring infections of the leafing trees in England, conidia from the over-wintered twig lesions play a more important part than the ascospores discharged from the leaves on the ground [9]; in other European countries, in the United States, California [17], and in Australia [1], early infections as in the

case of apple scab are said to be caused by ascospores. But in England, so prolific are lesions on the twigs in their production of conidia in the spring that many of the opening buds after a shower of rain are found to be bathed with conidia; as the water dries off, these spores may be seen not only on the bud scales but actually sucked in amongst the young leaves and flowers of the opening buds. First infections are, therefore, usually found on the under surface of the leaves, and on the sepals of the flower buds [3].

V. pirina embraces a number of physiologic races [18] some of which are reported to produce chlamydospores in culture, and mutant forms are also said to occur [6]. Here too, as in the case of the fungus of apple scab, some isolates of the pear-scab fungus turned out to be intra-group sterile, or inter-group fertile, but pairing of the isolates revealed only two compatible groups [8].

All varieties of pears are liable to this disease. The common Fertility is highly susceptible, as also are Doyenne du Comice, Williams' Bon Chrétien, Clapp's Favourite, Beurré Bosé and Marie Louise, but B. d'Amanlis, Conference, and Dr. Jules Guyot are less susceptible. There is evidence that the particular type of 'stock' on which the tree is worked has a bearing on the degree of susceptibility of the scion to scab attack [11].

Pear scab may be controlled on the same lines as indicated for apple scab. Pears are not so liable as apples to suffer surface injury from the application of fungicides, but Doyenne du Comice is sensitive to copper, though slight scorching of the foliage causes little injury to the crop itself [9]; Williams' Bon Chrétien may also suffer injury from lime sulphur [14]. Since sporulation of pear scab continues over a longer period than is usually observed with apple scab, additional sprayings with Bordeaux mixture are recommended, as many as six applications being advised for the most susceptible varieties [9, 10, 12, 13, 16]. While no method of winter spraying can destroy the protected stromata on the twigs, the summer sprayings, by killing the pustules on the leaves, help to keep down infections on the new wood. If practicable, since the twig lesions are such an important source of infection in the pear, all scabbed wood should be cut out before the buds open.

At Long Ashton, Bristol [9], a useful dual programme has been devised for controlling apple and pear scab together. It comprises two pre-blossom sprayings, the first for the pear, the second for both apple and pear; then follows the important 'pink blossom' spray for apple, and then the first 'post blossom' spray for pear, a week later. For this programme lime sulphur is employed at two strengths, 1 in 30 and 1 in 60, thus:

30th March to 4th April; for the pear, with 1 : 30.
10th April to 30th April; for apple and pear, with 1 : 30.
25th April to 4th May; for apple, with 1 : 30.
1st May to 15th May; for pear, at 1 : 60.

The weaker fluid is advised for later applications to both the trees, and to varieties of apples which are not sensitive to sulphur [9].

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Bacterial Canker, Leaf Spot, and Wilt of Plum and Cherry, *Pseudomonas mors-prunorum* Wormald & *Pseudomonas prunicola* Wormald

Plum and cherry trees in Britain are subject to severe forms of bacteriosis which affect all parts of the trees except the roots. These diseases have been extensively studied in this country by Wormald [13-27]. Bacterial diseases of stone fruit trees are also well known in Germany [1] and in various parts of America [4, 11, 12]. It is probable that the organisms associated with these diseases, variously named by different authors in these countries, are very closely related to, if not identical with, one or other of the two organisms *Pseudomonas mors-prunorum* and *Pseudomonas prunicola* discovered and named by Wormald, to be the causal parasites in Britain.

Stems, branches, shoots, buds, leaves, less frequently flowers and fruits, are all susceptible to attack. The disease affects the trees in three ways. The most serious phase is the development of cankers on stems and branches (' bacterial canker ') which may result in a die-back of all parts above the canker if the latter goes right round the stem or branch ; another phase of the disease causes young shoots to wilt (' bacterial wilt ') ; and the other form of the disease, a spotting and perforation of the leaves (' leaf spot ' and ' shot-hole '), accompanies the canker and wilt stages.

(a) *Bacterial Canker*

Both plum and cherry trees affected with canker show, in the spring, yellow, narrow, and curling leaves, and the shoots, especially those of the terminal branches, are stunted ; sometimes there is considerable loss of foliage from such branches (Fig. 356). Young trees recently planted up are the worst to suffer but established trees are also attacked. If the stem of a maiden tree of the sweet cherry is girdled with a canker the buds above the lesion are checked in their development, and though the tree may come into leaf the leaves above the lesion soon turn yellow and wither (Fig. 357 c). The buds below the canker may, however, grow into healthy shoots, but their leaves may later become spotted as a result of bacteria being splashed on to them from the cankers. Sometimes individual buds may be killed from the presence of a canker around the base of the bud itself, while other buds

not so affected grow out into healthy branches. Infected buds may also occur on spurs on older branches, and infection may pass from them into the axis of the spur, in which case all the buds on the spur may be destroyed. Lesions from affected spurs may extend so as to set up canker formation on branches and even main stems may become infected from spurs [22].

The extent of yellowing and withering of the leafy crown of an established tree will often indicate the degree of cankering in branches or stem. If the withering is general over the whole tree the canker has probably girdled the stem, but if the discoloration of the foliage shows only in a part or parts of the tree the indication



FIG. 356.—Bacterial canker and shoot wilt of stone fruit (*Pseudomonas mors-prunorum* and *Pseudomonas prunicola*). *A*, cherry tree with branch lesions. *B*, with bacterial canker on stem ; inset, portion of a branch with masses of yellow-brown exuded gum. *C*, young cherry tree with stem lesion, dead buds on the lesion, and curled leaves above it ; compare latter with the flattened leaves below (photos by Wormald, *J. Pomology*)



FIG. 357.—Bacteriosis of stone fruit (*continued*). *A*, leaf infection of cherry showing 'shot-hole' effect. *B*, lesion on the shoot, killing the terminal portion. *C*, severe leaf infection, causing withering of the foliage (photos by Wormald, *J. Pomology*)

is that only one or a few branches have been girdled, the rest of the branches being unaffected and capable of producing normal fruit. Plum trees suffer more than cherry trees from the effects of bacterial canker, because plum cankers arise mostly on the main stem, so that an entire plum tree may be killed from the presence of a girdling canker, whereas cherry trees are more prone to develop cankers on branches than on main stem, and healthy branches are in no way hindered from making normal growth. Cherry trees thus only partially affected may be saved if infected branches are cut away well below the cankered part. But cherry trees, like those of plum, do sometimes contract cankers on both stem and branches. A striking feature accompanying cankers on the stem and branches of the cherry is the copious exudation of a gummy substance from the cankers, often in such

quantity as to run down the stem (Fig. 356 inset). The exudate is also produced by cankers on the plum but is not nearly so copious as on the cherry. Though the gummy substance itself contains none of the pathogenic bacteria, its presence nevertheless causes much interference with the normal functions of the host tissues, and constitutes a serious hindrance to the translocation of food substances from the leaves to those parts of the plant below the cankered portion. A canker which completely girdles a branch or stem has much the same effect on the adjacent host tissues as ' ringing ' of the bark, for by destroying the cells of the cambium and phloem a canker sets up a distinct swelling of the part just above the lesion, from which there is much discharge of gum, especially in the cherry. The parasitic bacteria are to be found only in places where diseased tissues abut on the healthy, in the cambium, cortex, and phloem, and if a branch is cut through at a canker, the bark and woody tissues are seen to be discoloured brown from the staining effect produced by the bacterial secretions infiltrating into these tissues from those diseased. The bacteria die out of the cankers during the summer [5, 5a].

The bacteria, washed down from leaf spot infections (described below), gain access to stems or branches through wounds in the bark, and infection of these parts takes place only during the autumn, just before leaf-fall. In the vicinity of the wound the bacteria gradually attack and destroy the cortex, phloem, cambium, and sometimes the newly formed xylem as well. Even from a small abrasion in the bark an infected area may extend as a long narrow strip, sometimes for several feet up and down, or may go entirely round so as to girdle the tree. Despite such infection, however, there may still be present in the affected parts above a canker a sufficiency of food reserve to allow for growth of fresh foliage in the spring, and sometimes even undersized fruit may be produced, but, in general, the new foliage begins to lose condition fairly early, and the parts above the lesion die if the canker goes right round the stem. If the cankers do not encircle the stem the tree may grow normally without showing any leaf discoloration, but the canker may still spread, evidence of which may be seen by a progressive ridging of the bark. A bacterial canker of the plum and cherry does not, however, get bigger from year to year, like an apple canker (*Nectria galligena*, Fig. 339), because the bacteria die out of the cankers during the summer, and thereafter, since they have every chance of healing up like ordinary wounds, by formation of callus, the trees bearing them are no more subject to fresh infections than healthy trees, since new stem or branch infections, as above mentioned, probably originate from without, from bacteria washed down from the leaves.

(b) *Bacterial Shoot Wilt*

During a wet season, usually towards end of May, young green shoots of plum and cherry make very rapid growth, and on account of their increased succulence become very susceptible to bacterial infection. Such infection is believed to take place from direct penetration of the shoot through the lenticels, the ultimate effect being that the terminal leaves, though still remaining green, lose their turgidity and wilt. On the shoot itself a number of isolated blackened spots which become elongated in the direction of the axis arise and soon appear as sunken areas 2 to 3 mm. long, surrounded by a paler zone which again is bordered by a darker

margin. These narrow lesions may sometimes be several inches long, and if confined to one side, cause the shoot to curve over, but if the shoot is completely girdled the part above the lesion wilts and dies (Fig. 357 B). Shoot wilt is almost invariably accompanied by leaf spotting.

(c) *Leaf Spot and Shot Hole*

The cankers on stems or branches, and the spots on the leaves, are two distinct phases of this disease. The bacteria pass the winter and spring in cankers on stems and branches and die out in the summer but not before the leaves become infected from them. It is not exactly known how the leaves become infected in the spring. It is possible that the organisms are carried from the cankers to the leaves by insects or splashed in raindrops, or perhaps the bacteria pass the winter in or on the buds, infection of the leaves taking place as the buds expand. There is little doubt, however, that much of the leaf-spot infection occurs during wet periods from the splashing of the organisms from cankers to leaves. The spots on leaves of the plum are dark brown, more or less circular, about 2 mm. in diameter, with a narrow pale zone around, but the central tissues of a spot killed by the bacteria soon dry and drop out, leaving a ' shot-hole '. Sometimes young leaves in prolonged wet weather become riddled in this way, but a dry spell soon checks fresh infections. The leaf spots are infected with bacteria, which, however, do not ooze out to the leaf surface unless the leaves are kept continuously wet, and it is probable that when such conditions prevail just prior to leaf-fall the bacteria are washed down the branches and stem when these parts, as stated, become susceptible to canker formation in the autumn.

On the cherry, the leaf-spot symptoms are much the same as those on the plum (Fig. 357 A). The spots are usually numerous and crowded, rather small and angular when scattered, the bigger ones being from 1·5 to 3 mm. in diameter. Sometimes crowded spots join together to form necrotic blotches causing the leaves to become distorted. Isolated spots later dry out and drop out to form ' shot-holes ', as in the plum. One way in which infection of leaves probably does take place is through water pores around the margin, and a general withering of the leaf around the margin is a common feature of infection in the cherry. Sometimes infection also occurs on petioles and midribs. This appears to be brought about by an extension of the disease from an infected spur upwards along the petioles into the lamina, and from the midrib it may enter into the larger veins. This method of leaf infection is, however, by no means common, and seems to take place only in very wet weather in early spring before the leaves are fully expanded, in which event the leaves may be killed before emergence. Leaf spotting is usually most severe in the neighbourhood of branch cankers, which seems to indicate that the germs are washed down by rain to the foliage beneath, and though there is no visible exudation of bacteria on the leaves, yet they may be seen in great swarms if leaf spots are dissected in a drop of water, and it is probable that the organisms come to the surface of the spots when rain collects on the leaves. It is by no means easy to detect the presence of the bacteria in the leaves, except in sections suitably stained and counter-stained to differentiate between bacteria and mitochondria. In Fig. 358 the germs are shown mostly in the intercellular

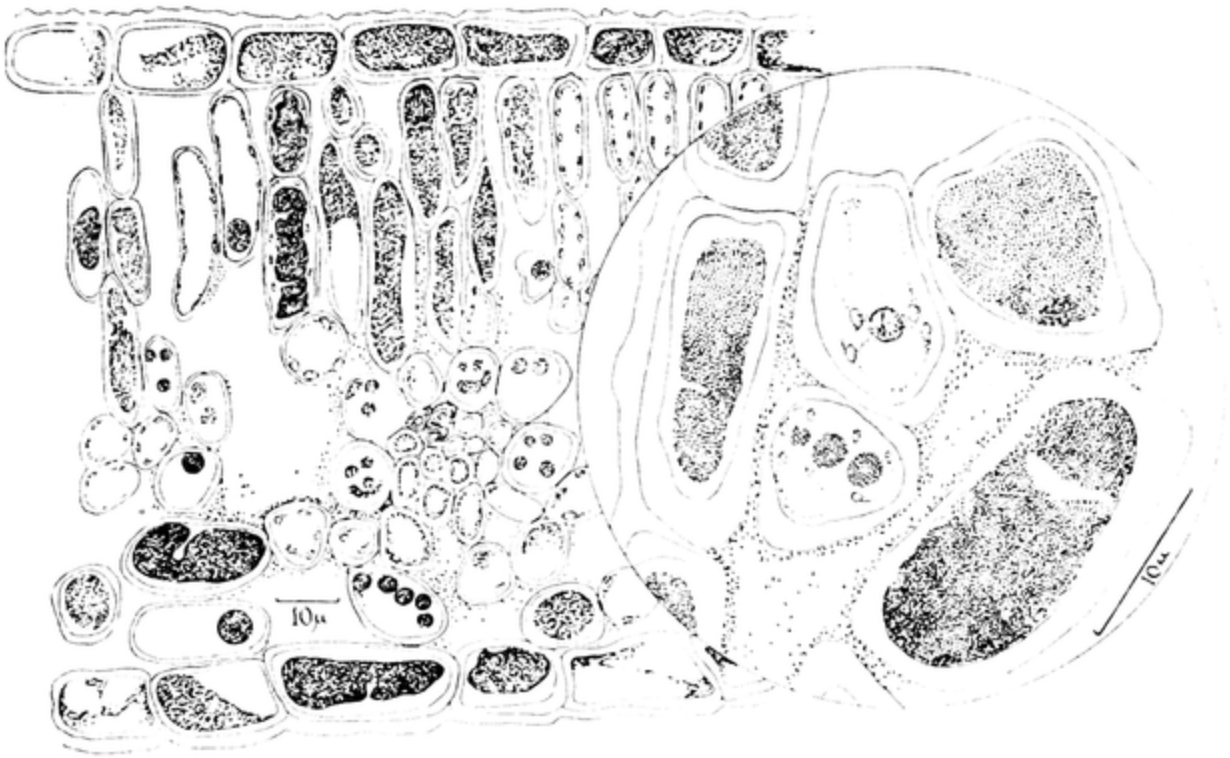


FIG. 358.—Bacteriosis of stone fruit (*continued*). Transverse section leaf of cherry showing the bacteria in the intercellular spaces at time of very early leaf infection; there is considerable swelling of the cell walls, especially of those cells in contact with bacteria as well as in the cells filled with gummy deposit (dark contents). Inset, showing the bacteria between the cells, and masses of amorphous gummy substance in the cells, the cell walls being much swollen. From slides of specially fixed material, microtomed and stained with Milovidnov's method for differentiating between bacteria and mitochondria (from a slide made by Watson)

spaces near the small veins, and they are also seen, in fewer numbers, within the cells of the conducting sheath. Sometimes wilted flowers and young green fruits of cherry may be found to harbour the bacteria in their tissues, but whether such infection takes place from without or from within by invasion from infected spurs is not known. But the disease may spread from the flower-trusses to the twigs, causing a withering of leaves and flowers above the point of invasion, so that twig blight occurs and further infection may appear as the fruit develops, the fruit stalks turning black and fruit formation is inhibited [27].

Wormald found these diseases of plum and cherry in England to be caused by two bacterial organisms which he named *Pseudomonas mors-prunorum* and *Pseudomonas prunicola*. The diseases attack also the Morello cherry, the cherry plum (Myrobolan), almond and peach, as well as some ornamental shrubs. An organism *P. syringae*, reported to cause disease in a number of other host plants, including roses [9] and lilac [18] and *P. citriputeale*, causing citrus blast [10], are believed to be the same organism as *P. prunicola*. Whether inter-infection between these ornamental bushes and stone-fruit trees takes place is not yet known, but the relation is one of practical importance if these additional hosts are allowed to remain in proximity to fruit plantations [22].

P. mors-prunorum is a rod-shaped organism, from 0·8 to 2·5 μ long; rarely forms filaments; stains slightly by Gram's method; one polar flagellum, but frequently 2 or 3;

no endospores ; thermal death point 46° C. ; optimum temperature for growth 25° C. ; colonies on agar, grey or almost colourless ; strongly aerobic ; no gas formed ; survives over a range of *p*H 2·8 to 9·6 [8] ; gelatine dissolved by some strains. (Code number, according to the American system, is 211·2222032.

P. prunicola is rod shaped, from 0·9 to 2·5 by 0·3 to 0·5 μ ; filaments sometimes formed ; stains slightly by Gram's method ; one polar flagellum, but frequently 2 or 3 ; no endospores ; thermal death point 45° C. ; optimum temperature for growth 25° C. ; colonies as above ; liquefies gelatine ; strongly aerobic ; no gas produced. (Code number, 211·2322033.)

The two organisms can be distinguished from each other by growing them in a nutrient broth containing 5 per cent. saccharose ; transfers should be made from 3 days' old colonies. When tubes of the nutrient broth with sugar are a few days old they are examined by reflected light against a dark background ; the cultures of *P. prunicola* show a slight yellow tinge and are more or less translucent, while those of *P. mors-prunorum* are white and somewhat opalescent. One or both of these pathogens may be present at any phase of the disease, but *P. mors-prunorum* is the one most commonly associated with the canker phase in plums, and in the canker of the cherry either or both organisms may be present, but *P. mors-prunorum* is the more common, though the other is also responsible for the wilting of young, green plum shoots [8b]. There is some evidence of host specialisation in respect of the two organisms, for strains of the pathogen taken from plum are more virulent on that host than on cherry, and the same is true of strains taken from cherry when inoculated into cherry.

No conclusive results have been obtained that the disease can be controlled by application of artificial fertilisers. Sand-culture experiments showed that potash in no way reduced the amount of disease on the highly susceptible Victoria plum (worked on a resistant stock) ; in fact, high potash and phosphate content of the soil had an adverse effect, and low phosphate content reduced the amount of cankering ; it is suggested that the harmful effect of phosphatic manuring is due to its action in reducing the availability of iron salts [3] ; lime does not help to increase host resistance, proving in fact detrimental when used in combination with a balanced dressing of ammonium sulphate, superphosphate, and sulphate of potash [23].

Of susceptible varieties of plums, Giant Prune is the worst to suffer from canker and leaf infection, followed by Victoria, Czar, Prince of Wales, and Bradley's King Damson ; Purple Egg and Denniston Gage are rarely severely affected and the variety Utility is definitely resistant [7, 19]. Of susceptible cherries, Bigarreau de Schrecken, Cluster Black Heart, Black Tartarian, Emperor Francis, Bradbourne Black, and Black Eagle are very commonly attacked, while Frogmore and Governor Wood are less susceptible [22].

Since the canker phase of the disease on plums attacks chiefly the main stem and is less commonly found on the branches, one successful method of reducing the disease on the plum is to build up trees on resistant stems, the method being known as ' framework grafting '. For this purpose, some stems, such as those of Purple Pershore and, in particular, Myrobolan B, give better results than others such as Pershore, Denniston's Gage, and Victoria, all of which are very highly open to stem canker, while those of President, Utility, Warwickshire Drooper, and Purple Egg are intermediate [2, 6, 8a]. Although varieties of cherry, in general,

are more prone to contract canker on branches than on main stem, there is hope that, for cherry trees as well, resistant stocks will be available for working on to them the more valuable susceptible varieties [7, 22].

Since cankers are developed on the trees through wounds during the autumn and winter, no pruning should be done during that time, and it is advised to remove all leader shoots, broken branches, and feather shoots during the growing seasons, preferably during May and June, at which time wounds are less susceptible to infection, for the organisms in the cankers die out during the summer and by midsummer most cankers tend to become healed without attention; any open wounds left after cutting should, if practicable, be protected by the application of antiseptic paste such as that employed in the treatment of 'silver-leaf' of plums (p. 246).

As the bacteria attack branches and stems in the autumn, and the foliage in spring and summer, spraying programmes are recommended on the following lines: for stem cankers, spray about the third week in October with Bordeaux mixture 10 : 15 : 100; this, however, has not yielded as good results on plums as with cherries, and except in severe outbreaks it may be omitted, in favour of spraying both plums and cherries with this strength just before leaf fall; and for leaf spots on cherry, Bordeaux mixture 6 : 9 : 100 with a 'spreader' at time of bud-bursting, and again with 4 : 6 : 100, applied 3 weeks after the petals have fallen. For plum foliage the latter may be used, under similar conditions, and 2 or 3 weeks later, using a colloidal copper preparation containing 0·025 per cent. metallic copper [8a, 8b]. Some growers have reported harm to certain varieties of cherries and cropping plum trees (but not to nursery or young orchard trees) from spray treatment, but the addition of cotton-seed oil (0·75 per cent. by volume) obviates this damage [2].

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Plum Rust, *Puccinia pruni-spinosae* Pers.

Plum rust is fairly common in the south of England in wet seasons during late summer ; it is not frequent in Scotland where it has only recently been reported [13]. It is widely distributed in Europe, North America, Australia, New Zealand, Tasmania, Egypt, South Africa, and other areas. The rust also occurs on apricot, almond, prune, peach, and nectarine, but is not common on the cherry [3, 7, 10, 12].

Stone-fruit rust is caused by *Puccinia pruni-spinosae* (*Tranzschelia pruni-spinosae* Fischer). The fungus is heteroecious, having the uredospore and teleutospore stages on the stone-fruit *Prunus* host, and the aecidial stage on various species of *Anemone*, chiefly *A. coronaria* (Fig. 359). In the United States, two distinct forms of the parasite are recognised, one on wild plums and the other on cultivated plums, the latter apparently being the prevalent form in Britain. The differences between the two forms are based chiefly on teleutospore characters, no constant differences being shown by the uredospores on any of the hosts above mentioned. At least two physiologic races of the rust exist [4, 8, 9, 17, 19].

The rust on the plum occurs mainly on the leaves, its effect being to bring about partial or complete premature defoliation and a general loss of vigour in the tree. It may occur on badly cultivated soil in dry localities [6], or in low-lying moist situations [10]. In Palestine, on practically the entire range of hosts above mentioned, the disease may develop locally in all parts of the country during the



FIG. 359.—Plum rust (*Puccinia pruni-spinosae*). The aecidial stage on *Anemone coronaria* (photo by Dennis)

winter, and in summer only areas with a relatively low temperature and high atmospheric humidity are affected [16a]. While in many localities, as in England and the south-eastern parts of the United States, the rust is to be found only on the leaves, in other areas, as in the western parts of the United States, Australia and New Zealand, lesions and cankers may also develop on the young twigs, and blemishes and cracks may also develop on the fruits, making the latter unfit for canning and export [3, 9]. The rust first appears as small yellow spots on the upper surface of the leaves, and at corresponding spots on the under side brown pustules of uredospores appear. While the symptoms, in general, are usually the same on all species of *Prunus*, small differences have been observed in the colour, size, and distribution of the spots on other hosts [8]. The lesions on the twigs, common on peach varieties, form small cankered areas, causing the bark to split longitudinally, and in the crevices uredospores are developed. The blemishes on the fruit consist of small, circular, depressed spots or cracks, and mar the appearance of the fruit [7].

The uredosori are hypophyllous, cinnamon brown, pulvurulent; uredospores are oblong clavate, or oblong fusiform, 24 to 42 by 15 to 23 μ [9], or, 20 to 35 by 10 to 18 μ [1], with a wall brownish yellow above but paler below, smooth above but sharply echinulate below, with 3 to 5 equatorial germ-pores; the spores are interspersed with capitate paraphyses [9]; other spore measurements given are, 18 to 28 by 15 to 25 μ, on myrobolan, and 25 to 45 by 12 to 16 μ, on apricot [8].

The two forms of the fungus in respect of the differences in the characters of the teleutospores are defined as follows:

(*a*) *P. pruni-spinosae* forma *typica* (Fischer); on various wild plums. Found in general in North America, Europe, and Asia. Aecidial stage usually found on *Anemone caroliniana* and *A. quinquifolia*. The teleutospore cells are of approximately the same size and colour, the wall not noticeably thickened at the top of the apical cell, and uniformly coarsely verrucose over both cells; oblong or obovate-oblong, 30 to 39 by 18 to 27 μ; wall, chestnut brown, 1·5 to 2·5 μ thick.

(*b*) *P. pruni-spinosae* forma *discolor* (Fischer); on cultivated plums. In Europe generally, with slight exception, on *Anemone coronaria*. Sori on the leaves are pulvurulent, dark, chocolate brown masses forming compact almost black clusters. Upper cell of the spore globoid, the lower cell from globoid to an irregular-shaped form definitely contracted at the base; wall of the upper cell generally, but not always, thickened at the apex and much darker than the wall of the basal cell, coarsely verrucose over its entire surface; wall of the basal cell never completely verrucose, and in some hosts almost entirely smooth. The spores measure from 26 to 39 by 15 to 23 μ; the basal cell is generally narrower than the apical cell [9].

Infected anemone plants present a somewhat stiffer appearance than the normal plants, and bear the aecidial cups thickly aggregated on the lower surfaces of the leaves (Fig. 359). The aecidia (*Aecidium punctatum*) occur chiefly on *A. coronaria*, but may also occur on the wild *A. nemorosa*, the windflower. In Switzerland, infected plants of *A. ranunculoides* showed hypertrophy of the stems and a swelling of the involucral leaflets, with spermagonia and aecidia developing

on the sepals [15]. The spermagonia are chiefly epiphyllous and scattered. The aecidia are hypohyllous, scattered, often opening out into four widely spreading lobes; the aecidiospores are globoid or oblong globoid, 18 to 26 by 15 to 23 μ [6], or 16 to 24 μ [1]; furnished with a thick, verrucose wall, 1·5 to 2·5 μ thick, cinnamon brown in colour. Plum leaves inoculated with aecidiospores from *A. coronaria* developed teleutospores 80 days later [6].

The rust is capable of surviving from season to season in various ways. The fungus may persist as a perennial mycelium in the underground stems of Anemone, and when these are planted in early autumn spermagonia and aecidia are usually observed on the leaves, from March onwards. (The mycelium in the underground stems is said to be haploid, while in the leafy sub-aerial organs a dikaryophytic condition of the nuclei has been found, and there is evidence that the rust is heterothallic [5].) In some localities the rust appears to survive the winter in the form of uredospores which have survived in lesions on the twigs [7, 8, 9], bark, or buds, or even on living leaves which had remained attached to the tree. In some areas, therefore, the intervention of the aecidial host does not appear to be necessary [19, 21].

To keep the disease in check in the orchard it is obvious that anemone plants should not be cultivated in the near vicinity. As a precaution, plum trees should be sprayed with Bordeaux mixture when the fruit is about half grown, and again immediately after picking [6, 17, 22, 23]. A zinc-lime spray, 10 : 5 : 100 was found to be almost as good as a 4 : 4 : 100 Bordeaux mixture [3]. Lime sulphur, 1 in 60, after picking is also recommended, care being taken also to bury deeply all infected leaves [12]. Gently heating the underground stems of anemone up to 34° C. for four days was found to kill the rust in the tissues without injury to the host [5].

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Silver Leaf Disease of Plum, *Stereum purpureum* (Fr.) Fr.

Silver-leaf disease attacks a wide range of fruit trees and bushes, including apple, pear, cherry, peach, gooseberry, raspberry, and currant, and is most destructive of all on the plum. All varieties of plums are subject to this disease, though some varieties are seldom attacked, while some degree of resistance, in certain localities, is shown by Monarch, Purple Egg Plum, and Damsons. A few other broad-leaved trees are also susceptible, and the fungus causing this disease can thrive saprophytically or hemi-parasitically on the stumps of numerous forest trees but rarely on oaks and conifers [5]. The disease has been extensively studied by Brooks in England, [1-9] where it first came into prominence about 1902 [11].

A characteristic symptom of the trouble on plum trees in the orchard is a silvery sheen on the leaves (Fig. 360), a pathological effect due to the presence of fungus mycelium, not in the leaves themselves but in the wood of the branch or branches bearing them, often at considerable distances below their insertion. Silvered foliage shows signs of wilting as soon as a toxic substance formed by the fungus reaches the leaves in the transpiration stream. It has the effect of causing the upper epidermis to separate from the palisade cells (Fig. 361) and with the accumulation of air under the loosened epidermis, a silvery effect results from the play of light on the surface of the leaf. Silvering of the foliage, in any plant, is not always a sign of infection and may arise from various factors disturbing the normal functions, and silver-leaf disease kills some of its victims, e.g. hawthorn, roses, beech, and birch, without producing this feature [5].

Infection always starts from a wound in the bark or any part of the tree, rarely on the roots unless they are exposed, but permeates the entire tree, roots, stem, and branches. In early infection of plum trees, which are usually several years old before they are attacked, the silvery effect may be seen only on a few branches, but the affection spreads gradually until the entire tree succumbs. Sometimes trees lightly affected may recover if induced to make greater vigour of growth, especially during a very dry season ; the disease is difficult to check if a wet summer is followed by a comparatively mild winter [5, 12].

Silver-leaf disease is caused by a Basidiomycete, *Stereum purpureum*, whose bracket-like or imbricate fructifications are common enough on logs or stumps of a wide range of trees. The sporophores are formed in abundance on killed branches of plum trees, and may sometimes be formed on a dead branch while the rest of the tree is still bearing silvered foliage [1]. They show considerable variation in shape and colour ; sometimes they are resupinate, at other times profusely imbricate ; if the dead branch is horizontal the



FIG. 360.—Silver leaf of plum (*Stereum purpureum*). Showing contrast between silvered (left) and healthy (right) leaves of plum (photo by Wormald, *Diseases of Fruits and Hops*, Lockwood) (see also Fig. 32)

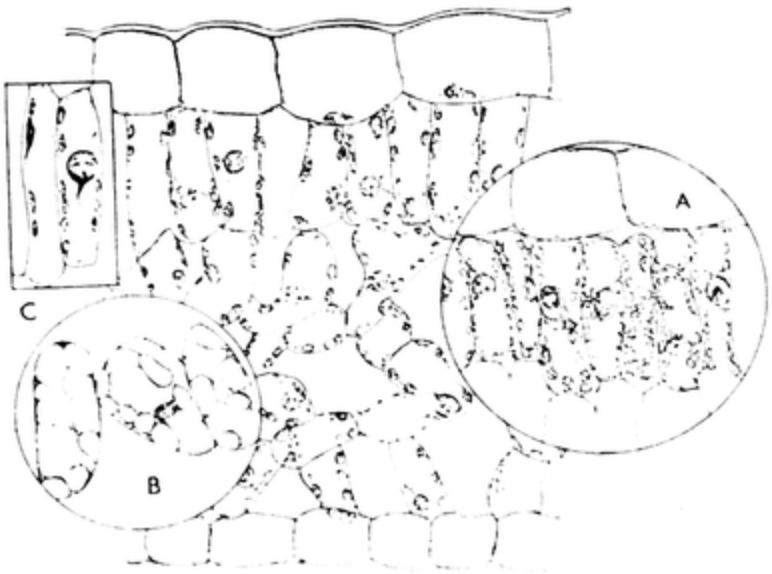


FIG. 361.—*Stereum purpureum*. Section of healthy leaf of Victoria plum (× 640). Insets, *A*, portion of a heavily silvered leaf showing separation of epidermal cells from the mesophyll. *B*, palisade cells from a yellow, heavily silvered leaf showing excessive starch accumulation. *C*, degeneration of one nucleus after nuclear division in a palisade cell (after Tetley, *Ann. Bot.*)

sporophores are usually found pressed down flat on its under surface, but if on the trunk or on a vertical branch they are partially free, bracket-like, and densely imbricated (Fig. 32). Young sporophores are purplish in colour but become dingy with age; the free, sterile upper surface of the imbricate kind is tawny or brown, frequently hairy and zoned; the hymenium on the lower or exposed surface is purplish or lilac and is covered with basidia. Mature sporophores vary from 2 to 8 cm. across; they are firmly built of a richly branched mycelium furnished with a vesicular system of hyphae (Fig. 48) which resemble a 'laticiferous system' for nourishing the hymenium, and under humid conditions there is a constant production of basidia with renewed production of spores in rapid succession. The sporophores appear in abundance after heavy rain during the autumn and have an extraordinary capacity for spore production; even if kept dry for weeks or months they will sporulate freely if moistened, a feature which no doubt accounts for the widespread occurrence of the fungus in nature [10]. Basidia vary from 4 to 5 by 4 μ; they carry 4 sterigmata 2 to 3 μ long, which bear small, oval, hyaline, thin-walled basidiospores 5 to 7 by 3 to 4 μ [5]. The fungus is easily grown in culture, from spores sown on sterilised blocks of plum wood, or from pieces of the infected wood or bits of a sporophore. The organism is common as a saprophyte or hemi-parasite on almost any kind of moribund woody material in nature, on which it is usually the pioneer, and does not seem to thrive if the substrate is already colonised by other fungi [5].

The spores are able to infect any freshly made wound on plum wood; snags or split branches are a favourable means of entry. Inoculations may also be performed by inserting pieces of moist sporophore into T-shaped cuts under the bark and are usually followed by the appearance of silvering. The trees are liable to infection at any time, but are much less susceptible during June, July, and August, so that if any pruning has to be done this operation should take place during the summer. At other times the trees are highly open to infection and spores introduced into a cut are immediately sucked into the wood by the transpiration stream [6]. Numerous hyphae soon become established in the wood, and the developing mycelium extends much more quickly in a longitudinal direction, both up and down from the wound, than in a lateral direction, and may eventually reach the roots, but whether the fungus ever breaks out from decayed roots into the soil to spread infection underground has not been proved. Infection

may possibly travel in this way from tree to tree by contact of roots, but again only through a wounded surface [4]. The bark of a diseased branch does not become affected as soon as the wood, but the fungus advances within it, as in the wood, more rapidly up and down than in a lateral direction [2]. As already stated the fungus does not pass into the leaves, and the silvery effect and death of the leaves are due to the infiltration of a poison produced by the fungus. In affected leaves the nuclei in the mesophyll cells break down early, and the separation of the epidermis from the palisade tissue is due to inhibition of division in the palisade cells, so that after the separation has occurred the epidermis is likely to stretch over the affected parts of the lamina, and the epidermal cells of silvered leaves are often larger than those of normal leaves [14, 15].

In the woody tissues of trees infected by *S. purpureum* there is considerable formation of gum. This substance, when first formed, is of a pale-yellow colour and more or less soluble, but when fungal growth slows down, the gum deposited in the farthest reaches of infection is insoluble and black at the margin and denotes the limits of infection. Beyond the barrier of black gum the fungus is not able to penetrate, and a xylostroma is thus formed within which staling products accumulate. It is probable, therefore, that these conditions occur prior to the development of the sporophores at the exposed part or parts of a xylostroma and in a position from which spores can be dispersed into the open. Furthermore, a gum barrier may delimit a whole xylostroma, thus rendering the contained fungus inert, and the tree may recover. The gum, which is formed by the living cells of the medullary rays infiltrates into the wood and appears to be derived from the action of the fungal enzymes on the starch contents of these cells. Any vascular tracheae in stem or branches occluded with the gummy substance are no longer functional in conveying water to leaves and buds, so that buds which happen to be in the path of advance of the fungus fail to open, and only those buds outside the invaded tissues are able to produce leaves which, however, are not prevented from silvering if the infiltrating toxin can reach them; buds quite outside the track of the fungus may, of course, thrive normally for a long time [5].

Owing to disturbance of the leaf functions consequent upon infection of the wood, silvered leaves contain more starch than normal leaves, and some of the starch remains immobilised however long the leaves may be kept in the dark. Branches bearing silvered foliage wilt rapidly owing to loss of adaptability of the stomata in controlling transpiration, and also to

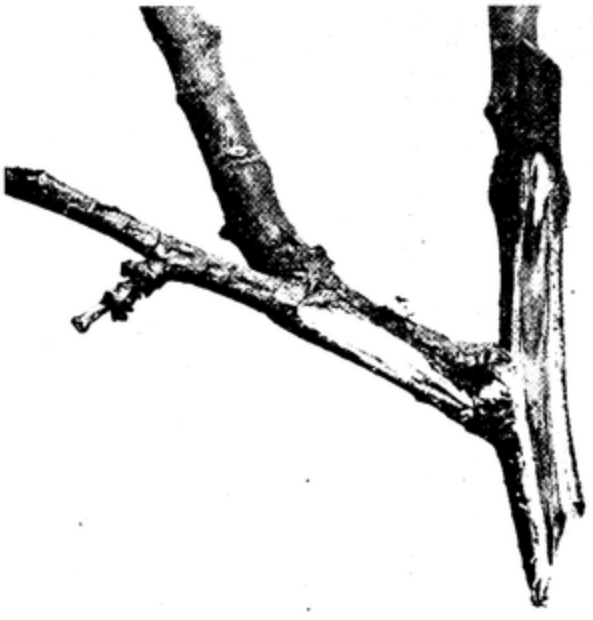


FIG. 362.—Silver-leaf disease of plum (*Stereum purpureum*). The wood exposed to show the dark staining effect which follows upon infection of the tree (photo by Foister & Noble)

direct physiological disturbance of the tissues which assist in this process [5]. These features may be shown experimentally if plum twigs with buds just expanding are placed in an extract of the fungus, or in a culture fluid in which the fungus had grown; wilting and browning of the leaves take place, but no silvering, owing probably to the rapidity of absorption of the poison. Silvering of the foliage and browning of the tissues may be induced by injecting the stem with the same fluid extract and the effects produced agree closely with those observed in natural infections (Fig. 362) [8].

Plum trees appear to be in a condition for maximum infection when the food materials available for the fungus in the wood are at the greatest concentration of soluble carbohydrates. This peak period usually comes in April, after which there is a considerable decline until a minimum concentration is reached in September; this probably accounts for the great success of April inoculations, and of the difficulty of getting the spores to infect wounds when the available carbohydrates are low during the summer.

The character of the soil does not seem to have any bearing on the incidence of silver leaf in plums, nor has the relative intensity of light or shade any influence on the severity of infection. Any treatment that will induce greater vigour of growth will help infected trees to recover; to this end, the application of basic slag, and to a lesser extent kainit, is recommended [6].

The nature of the root-stock on which a variety of plum is worked is said to influence relative resistance to this disease; thus when the very susceptible variety Victoria was worked on 'Myrobolan A' stock, susceptibility was greater in comparison with the same variety worked on the common plum [9]. Furthermore, one of the differences between the varieties Victoria and Pershore is the capacity of the latter to secrete more gum, thus rendering such varieties more resistant to attack [5]. Owing to comparative immunity of the trees from infection in the summer, silvered branches should be removed before mid-July and all prunings destroyed by burning; if it is found impossible to cut back healthy wood, general infection is likely and the tree must be pulled up. Since wounds caused by pruning and thinning are slow to heal in plum (and apple) it is advisable to cover them immediately with soft grafting wax or preferably with a white-lead paint (see p. 246). No infected prunings or logs on which the fungus could fructify should be left about the orchard, and since the fungus can thrive on the decayed stumps of trees and develop its sporophores, close watch should be made of any trees bordering on the plantation, particularly of birches, willows, and poplars, which are very susceptible to silver-leaf disease

By the Silver Leaf Order of 1923, fruit growers are compelled to destroy all dead wood of fruit trees killed by *S. purpureum* before 15th July every year.

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Fomes Disease or Heart Rot of Plums, *Fomes pomaceus* (Pers. ex S. F. Gray) Lloyd

Old plum trees in plantations, hedgerows, and neglected orchards are sometimes attacked by the Basidiomycete *Fomes pomaceus*, infection having entered through wounds and in some cases causing a die-back of the branches. Cherry trees are also sometimes attacked by it [4]. The fungus is widely distributed in Europe and America, and has also been reported in Australia [1]. The organism is, however, only a feeble parasite, and progress of the rot in the trees is usually very slow.

The disease has the effect of converting the centre of the heartwood into a white crumbling mass, the affected part being separated from the healthy wood by a brown gummy zone (Fig. 363). There are two stages in the progress of the rot, a 'gummy' and a 'white rot' stage, the latter being marked by an increase in hyphal development, especially in the occupation of the medullary rays. The gum is present in the vessels and in the medullary rays and is formed at the expense of the starch present in the ray cells. When the gum disappears in the later stages of decay the wood is rendered white and soft, a characteristic feature of the disease being a reduction in the thickness of the walls of the fibres. This is effected by a removal of the lining layer from the walls of the fibres, leaving a framework of lignin. The medullary ray cells, which become emptied, are the last to be attacked by the parasite. Plum wood in the final stages of white rot decay



FIG. 363.—*Fomes pomaceus*. Section of wood of Robin greengage decayed by the fungus (× $\frac{2}{3}$). (Reproduced by permission of Director, Forest Products Research Laboratory, *Ann. App. Biol.*)



FIG. 364.—Fomes disease of plum (*Fomes pomaceus*). The hoof-shaped fructifications on the stem ¼(· $\frac{1}{10}$). (Reproduced by permission of Director, Forest Products Research Laboratory, *Ann. App. Biol.*)

is very densely occupied by the fungus. The softening of the wood is apparently not due to delignification, for the damaged wood still gives lignin reaction, but to the weakening of the fibres due to the removal of the layers lining these elements.

The sporophores may be found from November to May, covering usually only one side of the tree-trunk (Fig. 364), and there is a copious discharge of spores during the last week of November, though scanty deposits may also be found practically throughout the winter [2]. The hard, woody fructification when perfectly formed is a thick, hoof-like bracket measuring about 5 cm. across, but more commonly it is resupinate or half-resupinate, forming a plate about 1 cm. thick, adhering to the bark. If any of the upper surface is exposed, the colour is an ashy grey, turning a brownish grey in the older parts; the porous hymenium is at first ashen then finally dark cinnamon brown. The hymenial pores are minute, about 4 to 5 to the mm. The spores are hyaline, almost spherical, about 6 μ in diameter; the flask-shaped cystidia are dark brown and measure on an average 18 by 8 μ [1]. The basidiospores remain viable for long periods (24 weeks) and the fungus may be cultured from them or from diseased wood, on malt or Dox's agar, the optimum temperature for growth being about 30° C. The dense growth of mycelium is colourless at first, darkening later to a cinnamon buff; it retains its infectivity for a long time [2, 3].

Once the tree becomes affected, this disease is very difficult to control, and early removal of branches which exhibit symptoms of die-back is advisable, the wound being suitably protected by an antiseptic coating [1]. The variety Pershore is rather more susceptible than the Victoria plum [2].

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Cherry Leaf Scorch, *Gnomonia erythrostoma* (Fr.) Auersw.

This disease of wild and cultivated cherries was first observed in Germany about 1880 [3], and in 1900 was reported to be present in numerous districts in south-west England [2, 5].

Trees affected with leaf scorch retain their withered leaves for an abnormally

long period, often throughout the entire winter and sometimes into the spring. Withered leaves may even be found when the trees are producing new blossom [5]. The disease has the effect of inhibiting the formation of the normal absciss layers at the base of the petioles, thus preventing the fall of the leaves (Fig. 365).

Early symptoms of 'scorch', seen on the leaves about the beginning of July, consist of yellowish patches along the margin or near the midrib; the fruit may also be attacked about the same time and may either drop off early, or not be formed at all, or it may become malformed and develop hard spots on the flesh [5]. With the spread of the disease in the leaves and its extension into the leaf stalks there follows much destruction of the leaf tissue; the trees become so impoverished that little fresh wood is made; those infected for a number of years show weak growth and finally die. Owing to lack of nutrition from the poor reserves laid down in the wood, the twigs of the current year remain stunted, the internodes scarcely developing at all, and the leaves are small and crowded together.



FIG. 365.—Leaf scorch of cherry (*Gnomonia erythrostoma*). Top, a cherry branch with withered leaves of last season still firmly attached at points indicated by the three pointers. Below, old, dead leaves, seen below the flowers, have remained attached to the branch throughout the winter and bear perithecia (photos by Salmon, Wye Reports)

Leaf scorch is caused by *Gnomonia erythrostoma*, a member of the Pyrenomycetes. Towards the end of August the spermagonial fructifications may be seen in great number on the under side of the leaves, forming tiny circular spots on the yellowed parts, and soon the whole leaf turns brown. The spermatia are straight or curved, 8 to 18 by 0·5 μ, and are extruded through an ostiole, embedded in a short, mucilaginous coiled thread [1]. Perithecia continue to develop in the leaves from November to March; they are furnished with a long, protruding neck; the ascospores, 16 to 18

by 5 to 6 μ, are septate, and unequally 2-celled; there are no paraphyses. The perithecia in the leaves usually reach maturity in the spring and the ascospores are ejected when the new leaves appear. Penetration is direct [1, 5] and the intercellular mycelium, devoid of haustoria, becomes established mostly in the spongy mesophyll. During the summer the fungus passes from the lamina into the leaf-stalk, killing the cells right down to the branch without, however, actually entering the branch itself, but preventing the formation of the abscis cork layers.

The disease is favoured by a moist environment, and its rapid spread is often encouraged by overcrowding in the orchard.

The varieties Florence, Waterloo, Frogmore Bigarreau, and Early Amber are very susceptible; Blackheart and Elton Heart less so; while Turk, Crown, and acid cherries are resistant [1, 2, 4].

To check the disease, the trees should be sprayed with Bordeaux mixture (4 : 4 : 50), a first application being given just before the flowers open (in Kent about 12th April), and a second soon after the petals have fallen (about 10th May). If trees are few, the dead leaves should be collected and destroyed [4, 5].

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Peach Leaf Curl, *Taphrina deformans* (Berk.) Tul.

Leaf-curl disease is common on peach trees and also attacks almond and nectarine [26]. It is far more prevalent in the orchard than under glass.

There is no definite knowledge of the place of origin of peach-leaf curl, but it is believed to have existed in Britain as far back as 1841 [28], and was recognised in 1862 as being due to "a minute fungus, *Ascomyces deformans*" [5]. The disease is now widely distributed in Europe and America, in parts of Asia, notably China and Japan, and is reported also from Africa, Australia, and New Zealand [2, 3, 9].

The symptoms of leaf curl on peach trees in the spring are very striking. Soon after the leaves are well out of the bud some of them appear to be distorted, folding over so that the tips are directed backwards while others more heavily infected curl so badly that the whole lamina with the exception of the tip looks like a partially inflated paper bag, but other leaves again, while still being more or less dorsiventral, may be distorted from tip to base (Fig. 366). The blistered portions are thicker and softer than the normal parts of the leaf blade, and while the affected areas may remain green for a while, they gradually change to yellow and finally to a reddish purple tint which renders the affected leaves very conspicuous against the green colour of the healthy foliage. The reddish velvety surface of the lamina soon becomes covered with a whitish-grey bloom which may appear at both surfaces of the leaf, but chiefly the upper. Lesions on the fruit are not common.

Affected leaves fall off early, and in heavy infections the trees may suffer severely from premature defoliation in late spring. There is, however, an apparent

recovery during the summer by the production of a fresh crop of leaves, and as this new foliage is obviously developed at the expense of a further drain on the already depleted resources of the plant, recurrent attacks of leaf curl from season to season are very weakening and the fruit deteriorates in bulk and quality every year.



FIG. 366.—Leaf curl of peach (*Taphrina deformans*). The affected leaves are thickened (see Fig. 143) and of a reddish-purple colour; note the blistered and crumpled condition of the smaller leaves

Taphrina (*Exoascus*) *deformans*, the fungus causing leaf-curl disease, is a member of the Ascomycetes, but in its type of fructification and behaviour in artificial culture it is not at all typical of that group of fungi. Thus, no true conidia are formed, and actually the grey bloom on the leaf surface consists of a great number of asci which are not developed and protected within a fruiting body at all, but merely break through the leaf cuticle under which they arise from a mycelium within the leaf, to form a hymenium of indefinite extent, so that the old generic name *Exoascus* (still in use by many authors) is descriptive of the final position of the asci exposed on the surface of the leaf (Fig. 143 D). The asci vary in size from 25 to 40 by 8 to 11 μ, and appear more like exposed terminations of hyphae than the usual club-shaped cells typical of asci. Each ascus contains 8, or fewer, globose ascospores which measure from 3 to 4 μ in diameter. A characteristic feature of this fungus is that the ascospores frequently bud, in yeast fashion, whilst still within the ascus to form minute secondary spores in such profusion as to fill the entire ascus. These tiny spores, which may be called 'conidia', are capable of growing on artificial media by budding; they are slightly oval in shape and measure from 2·5 to 6 by 4·8 μ [12]. The ascospores, which are discharged mostly in early summer, are ejected forcibly from the asci, adhering together in tiny spore balls which may be carried by wind or splashed in raindrops, and in water the ascospores float apart. In culture it is remarkable that the ascospores do not develop a definite mycelium and the colonies consist purely of conidia budded from the spores; some of the conidia, which are furnished with thicker walls than others, appear to be of the nature of resistant resting cells and contain denser cytoplasm than the remaining thin-walled conidia [18, 21]. The resting conidia germinate, not by immediate budding, but by producing very short germ-tubes of a

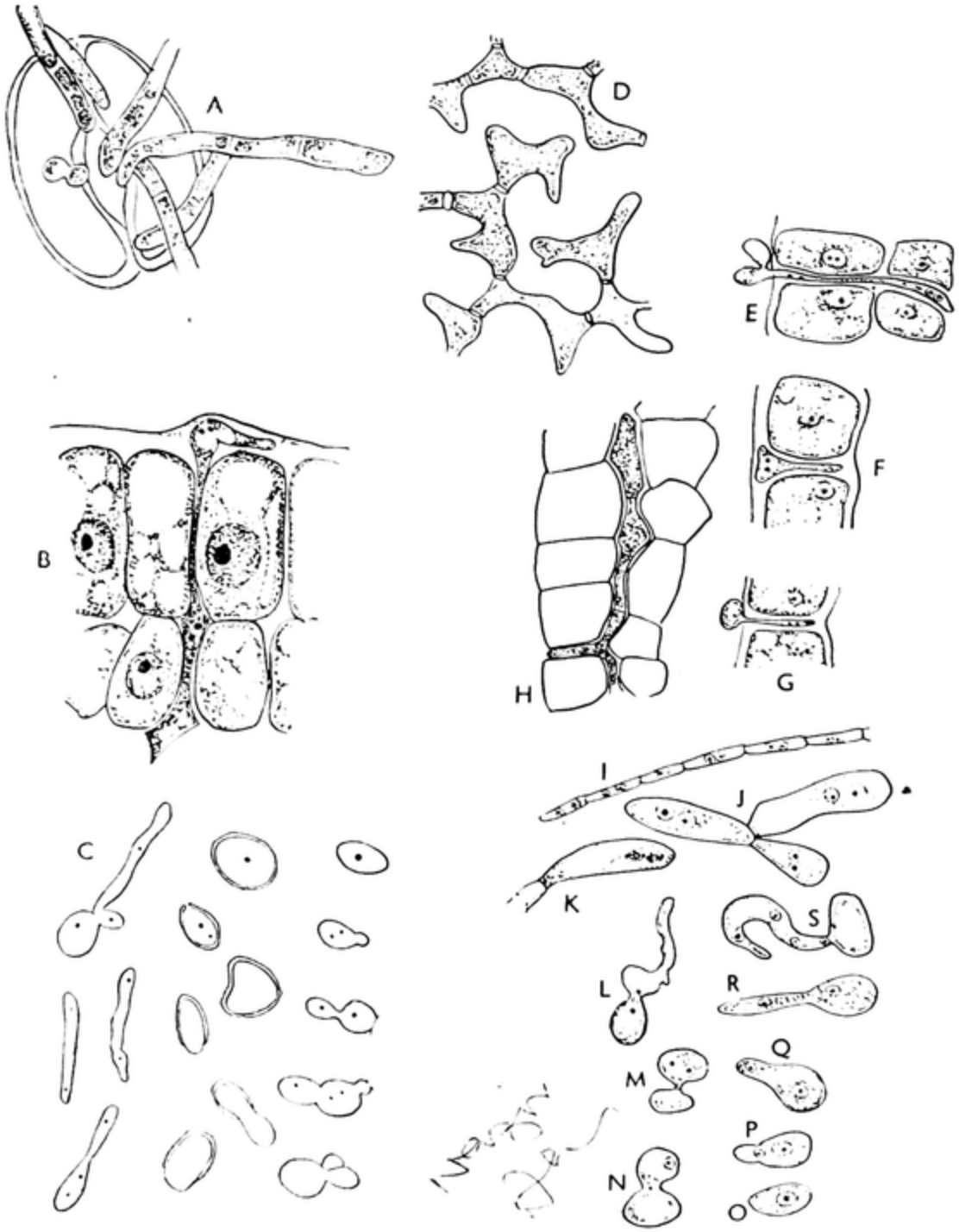


FIG. 367.—*Taphrina deformans*. *A*, showing stomatal infection (after Martin, *Phytopath.*). *B*, section of leaf showing binucleate condition in the intracuticular and initial intercellular mycelium. *C*, various types of sprout conidia (after Fitzgerald, *Sci. Agric.*). *D*, cells of sub-epidermal mycelium, viewed from above. *E*, a case in which copulation of conidia has apparently preceded penetration. *F*, *G*, hyphal penetration between epidermal cells. *H*, the sub-epidermal mycelium. *I*, *J*, *K*, types of binucleate cells. *L*, *M*, *N*, copulating conidia, from a culture. *O*–*S*, budding conidia (after Mix, *Phytopath.*)

swollen, vesicular nature, and from these, by a further process of budding, thin-walled conidia are formed again [18] (Fig. 367).

It is still not clearly known in what form the fungus of leaf curl survives from one year to the next. For a long time it was believed that the fungus existed as a perennial mycelium within the shoots [25], the fungus attacking the leaves again at the opening of the buds. It was maintained that the young wood became infected by the fungus passing into it from affected leaves, the mycelium surviving in the wood to renew infection in the spring, but this is now believed to play but a small part, if any, in perpetuating the disease [21, 23, 24]. Some state that the fungus is continuous from lamina to petiole, though in decreasing quantity towards the base of the petiole [24], but others are emphatic that the mycelium in the leaf never reaches the wood at all [23]. The general opinion would appear to be that primary infections which occur at the opening of the buds in spring are effected by spores, probably resting conidia, which have been sheltered on some part of the host during the winter [12, 23, 24].

Infections take place almost entirely in the spring, and when the dead infected leaves fall off prematurely in June and July and new foliage is developed in the summer to make good the loss, the trees to all appearances are free from disease for the rest of the season, for the new leaves are rarely attacked. But it is not clear what happens to the ascospores or the budded conidia which remain in the hymenium on the fallen leaves, and there is no evidence to show that they are capable of living through the winter on the decayed leaves on the ground, or in the soil, or elsewhere [12]. Since the ascospores do not reach maturity and are not discharged until early summer, their part in starting primary spring infections would appear to be ruled out.

The new foliage developed in summer to replace the prematurely fallen infected leaves apparently remains quite clean to the end of the season, and this is difficult to understand since the ascospores are in full discharge from the fallen leaves at that time. It has been observed, however, that late infections do actually occur on the new summer foliage but the lesions remain undeveloped, and, as they are so small and scattered over the leaf, they usually escape detection. Whether these light and late infections on the summer foliage are directly due to ascospores or to secondary infections from the spring lesions is not known, but it is remarkable that the primary spring infections on the young expanding leaves are so successful while infections on young leaves produced in the second crop in the summer are hardly visible, if not entirely absent. The probability is that the disease fails to develop on the second foliage owing to the higher summer temperatures, and these belated summer infections are so light that affected leaves are not lost to the trees prematurely like those at the spring infections, so that during the summer the trees to all appearances are free from disease.

At the time the ascospores are being discharged in the summer, the young buds in course of development in the axils of the leaves are protected by bud scales of such smooth texture that it is hardly possible for the ascospores to become attached to them and remain viable for a long period. It is, however, not improbable that if the ascospores germinate forthwith on the bud scales to form conidia,

the latter by virtue of their mass production and yeast-like consistency are enabled to adhere to the buds, especially as the bud scales become more and more pubescent towards the winter. Conidia produced from ascospores in artificial culture are capable of withstanding desiccation and low temperatures, and it is probable that the fungus is capable of surviving the winter in the form of agglutinated masses of conidia on bud scales or twig surfaces on the host [23]. In old cultures where budding is less frequent, the conidia are often quite thick-walled and the fungus may possibly be able to tide the winter on buds or twigs in this form. It appears, however, to be a matter of some difficulty to collect and isolate conidia from any parts of the host, even by washing, during the winter. Finally, there is the possibility that the organism may live over adverse periods as a saprophyte in the soil [12] or in substances naturally occurring on the bark of the peach, in the same way as wild yeasts are known to thrive epiphytically on their various hosts [25]. Moreover, sporulation has been found, in California, on peach fruits [31].

T. deformans is easily grown in artificial culture on a variety of media, even on those of low food value such as a dilute solution of peach gum. As stated, the growth from ascospores consists entirely of budding conidia forming colonies of a creamy consistency, like yeast. On potato dextrose agar [23] the lobed colonies are thick and convex, at first white, later becoming pink ; a well-developed mycelium, as already stated, is not produced in culture, but when the growth passes over eventually to the production of thick-walled conidia, the latter may sometimes be seen to germinate (Fig. 367 L, M, N) by the formation of very short hyphae which, however, resemble short chains of oval cells more than hyphal threads with definite cross walls [12, 21, 29]. On a 2 per cent. dextrose broth to which 0·1 tryptophane, or 1·0 per cent. peptone, was added, the maximum amount of growth by budding occurred over a range of temperature from 12° to 16° C.[19]. The organism has a wide range of tolerance towards acid and alkaline conditions ; on potato dextrose agar the best growth occurred at a reaction of pH_5[21].

Inoculations on peach have been performed successfully by smearing the buds with the yeast-like masses of conidia from pure culture, or by spraying twigs and buds with a suspension of conidia in sterilised water. Strong evidence in favour of the view that the fungus is capable of tiding over the winter in the form of conidia on the surface of the host was obtained from the observation that conidia sprayed on to the tree survived on the twigs from July to the following spring and were still able to cause infection culminating in the symptoms characteristic of leaf curl. Moreover, shoots that had been specially protected by covering them with bags to prevent the deposit of conidia showed complete freedom from disease, which would not have been the case had infection been present as perennating mycelium in the tissues of the shoot. Despite the observations that mycelium from the leaves may sometimes travel down the petiole into the twig of the current year, in some cases setting up distortion and hypertrophy of the shoot, there is no clear evidence that primary infections can be initiated from perennating mycelium emerging from the wood [23, 24].

It is noteworthy that the development of a true mycelium in the history of peach-leaf curl takes place only on the living host. Conidia sprayed on to young leaves continue to grow by budding, and long, dichotomously branched, septated hyphae soon make their appearance on the surface of the leaves. The tips of some

of these hyphae enter the leaf by cuticular penetration [12, 23], but stomatal entry also occurs (Fig. 367 A) [18]. Since early infections take place as soon as the buds break into leaf, infections occur largely at the lower leaf surface, but later, when the leaves are expanded, the upper surface may also be entered. The intercellular mycelium travels across the mesophyll and proceeds to spread out chiefly between the palisade layer and the upper epidermis, where it develops in greater abundance than in any other parts of the leaf. Infection of the young leaf does not retard its growth ; on the contrary it has the effect of stimulating cell division in the mesophyll cells to such an extent that the intercellular spaces become almost obliterated and there is finally little distinction between the cells of the palisade and spongy mesophyll, and the cell walls are also considerably thickened (Fig. 143). All starch contents in the leaf are used up by the fungus, and with the gradual degeneration of the chloroplasts a rich purple pigmentation appears in the enlarging vacuoles of the infected cells [10].

In preparation for the reproductive stage, the branches of the mycelium which became established between the epidermis and mesophyll now make their way between the epidermal cells to form a network of short cells beneath the cuticle (Fig. 367 B). When the latter is raised up and finally ruptured, these cells, each furnished with a pair of nuclei, elongate somewhat and form a more or less continuous hymenium over the blistered area. The paired nuclei in each cell fuse [10], and on the division of the fusion nucleus one of the two nuclei moves to the distal portion of the cell while the other nucleus remains in the basal part, which is finally cut off by a cross wall to form a small stalk cell to the upper and larger cell which becomes the young ascus. The nucleus in the latter undergoes a reduction division and eight spores are formed, but sometimes fewer, frequently only six, develop ; each ascospore is uninucleate (Fig. 143 D) [17].

The incidence of leaf curl is usually associated with comparatively low temperatures such as occur in the spring. The minimum temperature for the growth of the fungus is below 10° C., the optimum about 20° C., the maximum lying between 26° C. and 30° C. The inability of the fungus to thrive at comparatively high temperatures is perhaps one reason why the new leaves developed in the summer do not become infected despite the active discharge of ascospores during that period. The trouble in the orchard is especially severe if cold, wet weather prevails at the time the trees are bursting into leaf [22] ; in Germany in 1927, after a mild winter followed by an early spring, the disease was abundant, but in 1929, following a corresponding, comparatively very cold period, it was practically absent [11].

In view of the wide acceptance that the fungus causing leaf curl tides the winter in the form of conidia or their derivatives (the ' yeast ' conidia) lurking between bud scales or in crevices in the bark of shoots and branches, a considerable amount of investigation has been done to control the disease by spraying. Obviously the treatment must be carried out before the buds open, and in Britain the trees should be sprayed about the end of February. Good results have been obtained with lime sulphur, Bordeaux mixture, and Burgundy mixture [13, 15, 16, 30], the last named in the proportion of $9\frac{3}{4}$ oz. copper sulphate, 11 oz. soda, in 3 gallons of water being found very effective [15, 30]. A dormant spray of Bordeaux mixture of 6 : 10 : 100, with lubricating oil as an adhesive in the proportion of 3 gallons of

oil to each 100 gallons of the mixture, is also recommended [1]. A winter spraying during December or before mid-January with 6 : 6 : 50 Bordeaux mixture before the buds burst, or with 1 in 8 lime sulphur before the winter buds swell, has also been found beneficial [6, 13, 20]. Others recommend a very alkaline Bordeaux mixture incorporating casein as an adhesive, applied at the beginning of winter [4, 7]. A spring application of 0·75 per cent. copper sulphate when the buds are at the swelling stage is also advised [27]. Though not in itself a sufficient means of checking the disease, it is profitable to cut out infected shoots and see to the removal of all infected leaves in the vicinity of the trees, destroying all such material by burning.

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Shot-Hole Disease of Peach, *Clasterosporium carpophilum* (Lév.) Aderh.

' Shot hole ', or ' gum spot ' disease attacks stone-fruit trees such as almond, apricot, peach, plum, cherry, and some other related kinds. It is of common occurrence wherever these fruits are cultivated, existing in the warmer parts of Europe, and extending across the fruit-growing districts of Russia into Central Asia, where it causes most severe losses to the apricot industry (export of dried fruit) [21]. It also occurs in Australia [15, 17], and New Zealand [3, 4] whence it has driven out of cultivation the once very common Maori peach. It is prevalent too in Algeria and Tunis, and is responsible for serious epidemics on apricots and peaches in certain parts of South Africa [14]. In the United States the disease occurs

chiefly on apricot and peach [18, 23]. The serious losses suffered annually in some areas are due not so much to the actual destruction of fruit as to the extensive premature defoliation brought about by the disease, the result being that fruiting twigs are weakened and fail to develop fruit properly [23].

The first discovery of shot-hole disease was made in France in 1843 [13], and its occurrence in England dates back to 1864 when it was observed on a variety of peaches grown in Flintshire [2]. In America it was first found in 1894, in Michigan County, also on peach trees [20].



FIG. 368.—Shot hole of peach (*Clasterosporium carpophilum*). *A*, the shot-hole effect in leaf of peach. *B*, *C*, lesions on the fruit. *D*, on the stem (photos *B*, *C*, *D*, by Louw; reproduced by permission of Editor of *Farming in South Africa*)

The disease is caused by a member of the Fungi Imperfecti, *Clasterosporium carpophilum*, which now replaces the older name *Coryneum beijerinckii* [1, 9, 13], though the latter is still used in America. The systematic position of this organism has, however, not been definitely established, and it possesses some features which appear to indicate close affinity with the genus *Helminthosporium* [17]. The only fructifications formed by the fungus consist of coremia of conidiophores (Fig. 369) arising on leaf spots, and in black, sunken lesions on stem and fruit.

In artificial culture, as on corn meal or quaker-oats agar, spore formation usually occurs from a mycelium which gives rise to single conidiophores, but on the host, the conidiophores are developed in groups or coremia; saltation is frequent [17]. The conidia are elongate-fusoid, 23 to 62 by 12 to 18 μ, pale olivaceous, 3 to 5, mostly 3-septate, with slight constrictions at the septa. They are viable for at least 15 months, and on germination give rise to hyphae coated with mucilage by which the developing mycelium is enabled to adhere closely to the substratum; sometimes the germ-tubes develop appressoria, especially when germination takes place in the light [17].

Symptoms of the disease vary somewhat according to the type of host and locality. In America the disease takes the form of an affection of the twigs, called 'twig' or 'winter blight', whereas in Europe and Australia the predominant phase consists of perforated leaves and early defoliation, hence the common name of 'shot hole' given to this malady, on account of the small round holes left in the leaves after the affected spots drop out (Fig. 368). Lesions on the stem are far more common on peach than on apricot trees, while almond trees do not usually become affected with the twig-blight phase of the disease at all. But a serious feature common to all these three hosts is the blighting of dormant buds, and in

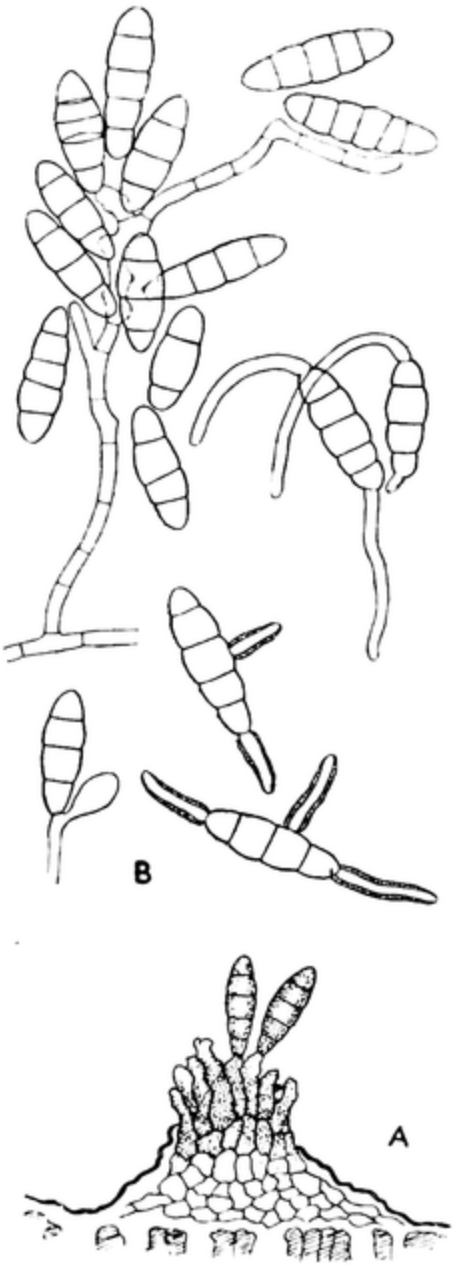


FIG. 369.—*Clasterosporium carpophilum*. *A*, section of a small acervulus showing conidia. *B*, various modes of development of conidia and their germination (after Samuel, *Ann. Bot.*) (see also Figs. 138-140)

this respect apricot seems to suffer most. Infection of the foliage, with the shot-hole effect, and later, defoliation, may again be common on all these three hosts. Infection of blossom and fruit is more prevalent on apricot and almond than on peach (Fig. 368 B, C).

Infected peach trees examined during the winter or early spring show on the young wood small, dark, irregular cankers which may vary in size from a pin's head to lesions $\frac{1}{2}$ inch long or more, and from which there is usually a copious exudation of gum. If these cankers girdle the young branches, a die-back condition may occur, and the loss of young wood is perhaps the most serious phase of shot-hole disease; in bad attacks all the previous season's growth may be killed during the winter. In the spring, at the opening of the buds, the disease becomes active, and while many buds may fail to open, others lightly attacked make variable attempts to develop shoots, which may subsequently become blighted, appearing as if scorched [14]. When the trees are in leaf, infection of the foliage consists of small round yellow spots with a purplish margin, but the yellow centre soon turns brown from the death of the tissues below, and then usually drops out, leaving a hole right through the leaf. On young leaves diseased spots may increase in area rapidly, and a fair portion of the leaf blade may turn brown and be killed without developing the shot holes characteristic of the smaller, localised lesions. If leaf infection is heavy, severe defoliation may ensue, leaving the trees in a weakened condition [23].

It is not usual for the disease to attack the flowers of the peach, but floral infection is common in the almond. Lesions on the fruit of the peach are, however, fairly common, and, like those on the leaves, are purplish at first, later turning brown but having a reddish margin; they finally become sunken into the tissues and filled with dark-coloured, sticky conidia. Not infrequently, diseased fruits may dry out, becoming mummified, and such fruit, by remaining attached to the tree over winter, may act as a source of infection in the following spring [7]. But the fungus is believed to survive the winter chiefly in twig lesions and in blighted buds, both as mycelium and spores. In the buds the spores are held and protected largely between the bud scales, and may be found in a viable condition at all times of the year. The fungus

remains alive in twig lesions of the peach for at least a year, again furnishing spores for leaf infection in the next season.

The spores are scattered chiefly by rain splashing on the trees, and, by virtue of the mucilaginous character of the spore-coat, once in contact with the host the spores are not easily dislodged [7]. Infection of the leaves during rainy periods may start as soon as the leaves project beyond the bud scales, and may continue on the expanded leaves for a long time, but the foliage is more resistant as it gets older. Infection may take place at either surface of the leaf, direct through the cuticle; in the leaf, the sparingly branched mycelium fills the intercellular spaces of the mesophyll and may also enter the cells. The host reacts to infection early and the activity of the fungus is definitely checked when the host cells around the margin of the lesion, by becoming lignified stop further progress, and as the cells just beyond the lignified zone divide to form a suberised zone (like the absciss layer at the base of a leaf), the central part of the spot becomes isolated from the rest of the leaf and drops out, but the margin around the shot-hole is protected by the cork border, so that there is no further spread of infection into the leaf [17] (Figs. 138–140).

The young twigs appear to become infected while they are still covered with epidermis, that is, before cork formation has begun. Penetration takes place in the same way as in the leaves, and the young fruits are believed to be attacked in a similar manner. In some cases buds may be killed when the twigs are blighted, either by direct attack, or perhaps by the fungus within the twig entering the bud at the base. In other cases, when the trees are in leaf, buds in the leaf axils become infected from spores washed down to them from lesions on twigs and leaves, and the spores retained within the bud scales remain thus protected and viable until the buds open in the spring. A good number of the early spring infections are seen to break out on the lower parts of the trees, close to the ground, caused probably by the splashing of spores from over-wintered leaves and fruit left lying on the soil [7]. Warm, rainy periods are greatly favourable to the incidence of this disease, and relatively long and continuous rain during the preceding winter is invariably followed by heavy loss of fruit.

As the fungus is capable of survival, especially in blighted buds and twigs, for periods of more than a year, methods of controlling this disease should be directed towards checking bud and twig infections. A spraying programme carried out with Bordeaux mixture, with or without lime sulphur, in sequence, is fairly popular among growers of stone-fruit trees [11, 14, 19]. In South Africa, Bordeaux mixture alone, in the proportion, 4 : 6 : 50 is recommended for spraying peaches, the first spraying being done before the arrival of the winter rains, or before the trees have lost all their leaves, a second application being given just before the buds open [14]. As this mixture is said by some to injure the foliage, lime sulphur is sometimes used and found to give equally good cover [5, 6, 10]. Thus a 20 per cent. application is made at the dormant period, followed by three treatments, using a weaker strength of 2 per cent., before blossoming, the other two being given at intervals of two or three weeks [22]. Some recommend the addition of lead arsenate to the spraying fluids to ensure increased adhesiveness [5, 12, 16]. A dormant application of carbolineum followed, before blossoming, by a copper oxychloride spray is also recommended [8].

No varieties of any of the trees above mentioned have proved to be immune from shot-hole disease; and some varieties of peaches and plums which have shown some measure of resistance to twig infections are very susceptible to fruit infections.

All infected wood and mummified fruits should be removed before the winter, and if practicable all infected debris on the ground gathered up and burned.

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Red Core of Strawberry, *Phytophthora fragariae* Hickman

This root disease of strawberry is believed to have first appeared in Scotland about 1920 and is commonly known as the Lanarkshire strawberry disease [19]. Since that date its spread has been rapid and cultivation of this fruit in the Clyde area is now on a much reduced scale. The trouble is widespread throughout Scotland [15-17, 19-22] and though somewhat less prevalent for a few seasons previous to 1943, during that year it again caused heavy losses in many plantations [17]. It occurs also in several parts of England [7, 13, 23], and the disease known in the United States as ' red stele ' is identical with the one now described [6a, 8a, 14, 18] though there has been an element of doubt about the identity [6, 10, 11]. Its first occurrence in the United States, under the name of ' black stele ', was in Illinois in 1935 [5].

Strawberry plants usually show the first signs of disease during the first fruit-bearing season and there is no definite evidence of its presence in the first year of growth. It breaks out in late spring or early summer in more or less circular patches in the crop, affected plants being small and squatted, the leaves of a dull, leaden-green colour becoming browned and sometimes reddened around the margins. The outer leaves are the first to perish though they are frequently retained for a long time. Affected plants may either fail to develop fruit at all

or die before the first berries ripen. In general, the effect of the disease is to check growth so that the plants produce few or no runners, ultimately wilt and die. After fruiting is over, lightly affected plants may recover to some extent by the development of new roots to replace those lost through disease. The recovery, however, is only temporary, for the new roots, in turn, become infected, and though root renewal tends to produce long woody stocks and additional crowns, the rooting system is so damaged that it fails to cope with the new growth.

The disease is greatly influenced by weather conditions, and prolonged wet periods during late July and August result in considerable spread of the trouble in the beds with consequent high mortality during the autumn and winter. Many selected varieties of strawberries which had hitherto shown good resistance to the disease have partially broken down under very wet soil conditions [17].

The contrast between the roots of affected and healthy plants is very striking. While the roots of sound plants are of firm texture, with an abundance of fibrous rootlets, those of diseased plants are soft and greatly deficient in fine roots and rootlets. Here actually is the source of the trouble, for the destruction wrought

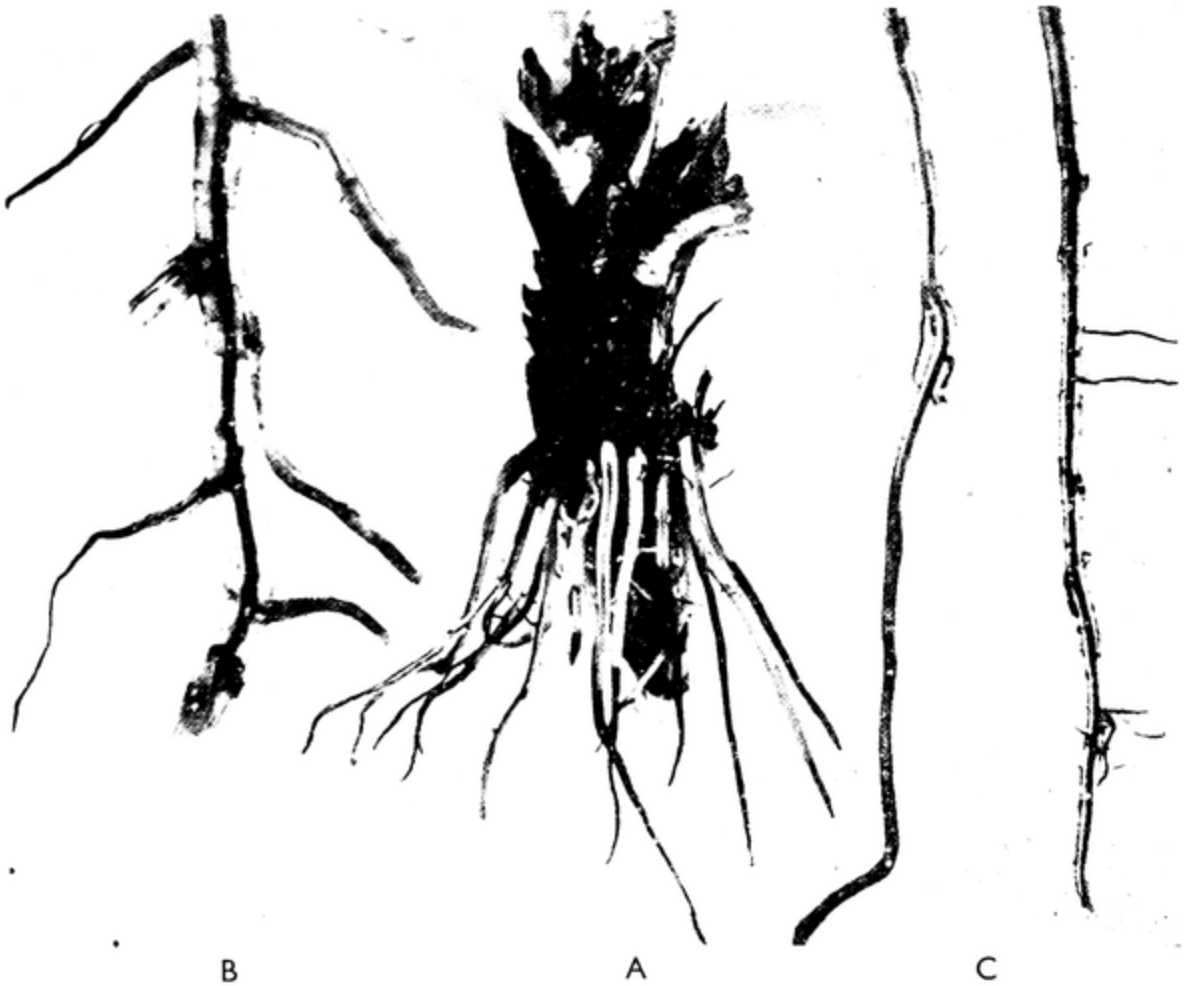


FIG. 370.—Red core of strawberry (*Phytophthora fragariae*). *A*, plant showing diseased roots. *B*, *C*, roots split to expose the red-discoloured steles (photos by Hickman, by permission of Long Ashton Res. Station, *J. Pomology*)

on the finer branches of the rooting system is responsible for all the symptoms of starvation shown by the parts above ground [19].

In the early stages of disease the roots are soft, of the consistency of india-rubber, and the tips of the roots become browned and rotted (Fig. 370). Under wet conditions, as the disease advances from the root tips, the cortex gradually becomes separated from the vascular cylinder and an appreciable length of cortex can be slipped off a young root, like a tube, leaving the vascular core or stele projecting like a coarse hair from the remaining stump of the root. On exposure to the air the vascular strand turns red, this discoloration being a characteristic feature of the disease, which gives it the name of ' red core ' or ' red stele ' [18]. A root rot of strawberry, known as ' black lesion ', common in Europe and America, is caused by a number of fungi, and appears to be distinct from red-core disease [8].

Whether all the symptoms of red core are caused by one or more fungi is not clear. Earlier investigators found a number of fungi in the diseased roots but conclusive evidence has now established a new species of *Phytophthora*, named *P. fragariae* [12, 13], to be the true parasite. This fungus has two stages of reproduction, an asexual sporangial and an oosporic, and both have been found to develop under natural conditions in the rotted roots (Fig. 371).

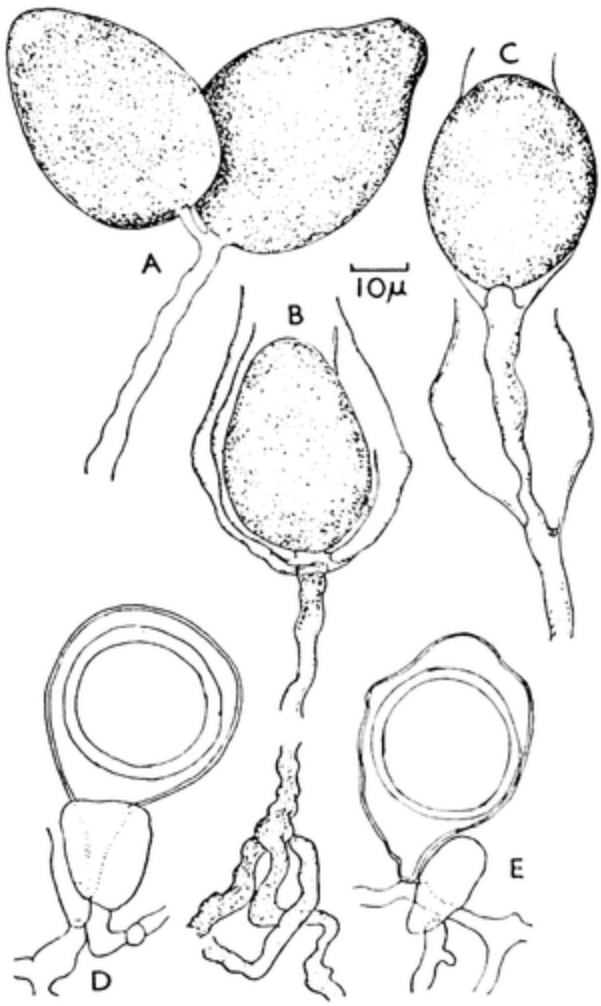


FIG. 371. — *Phytophthora fragariae*. *A*, sympodial development of the sporangia. *B, C*, sporangia produced by proliferation. *D, E*, oogonia with amphigynous antheridia (after Hickman, by permission of Long Ashton Res. Station, *J. Pomology*)

In damp soil, the sporangia, borne terminally on short, undifferentiated hyphae, emerge to the surface of recently infected roots chiefly from the root tips and from the cortical tissue close behind them. The sporangia are inversely pear-shaped, ovoid or ellipsoid, and measure from 32 to 90 by 22 to 52 μ (average, 60 by 38 μ); they are hyaline and non-papillate (Fig. 371). Germination is indirect and some 40 to 50 zoospores may be developed, either within the sporangium or in an extruded vesicle, and further sporangia may arise by proliferation. Sporangia and zoospores can be produced in abundance if bits of the roots are placed in water at room temperature, or inserted into a portion of the fresh fruit. Melted snow proved excellent for inducing production of sporangia and zoospores [6a]. Sexual organs, oogonia, and antheridia (amphigynous, less commonly paragynous) are produced abundantly in the central cylinder of the root, in the cortex of the smaller laterals, and near the tips of recently formed main roots. The

oogonia (Fig. 371 D, E) are lateral or terminal, 28 to 46 μ (average, 39 μ) in diameter, smooth and thick-walled, and of a golden brown colour; the oospores with a 3 μ thick smooth wall are hyaline, and vary from 24 to 44 μ (average, 33 μ) in diameter [11]. They have not been observed to germinate and their length of life in the soil has not been determined [6a]. New strains of the fungus are believed to exist [17a].

Though the germination of the oospores has, so far, not been observed, there is abundant evidence that decayed roots and rootlets harbouring oospores serve to bring about new infections in clean soil. Whether the fungus is capable of a saprophytic existence in the soil is not known, but it is reported to be still viable after a lapse of five or six [24], even eight years from strawberry planting [3]. The fungus has been cultivated on Lima bean agar, and portions of the culture in water produced zoospores [11a]. Quaker-oat and French-bean agars gave good growth at an optimum temperature of 20° C.; the sexual organs developed sparingly in pure culture [13].

Young strawberry plants in contaminated soil become infected through the root tips. After penetration of the delicate tissues of the root tip the fungus passes into the vascular cylinder, entering first the region of the pericycle, then the phloem and medulla, less frequently the protoxylem. The mycelium occupies the cells as well as the intercellular spaces. As secondary thickening sets in and the root gets older, the fungus concentrates mainly in the tissues of the phloem, with the result that roots are early killed and secondary organisms from the soil soon find access into the dead root tips. Various fungi, *Pythium* in particular, follow very closely in the wake of the true pathogen. Travelling along the vascular cylinder, the organism passes into the larger roots, killing the stelar tissues which become gradually reddened. This discoloration is probably due to oxidation of the fungal by-products and to their staining effect on the lignified elements of the stele. With gradual progress of the fungus towards the root-stock the pace of infection slows down, and the tissues of the crown are not occupied to any great extent, if at all, the infection being practically confined to the roots. There is, however, a certain amount of red discoloration in the root-stock and much of its parenchyma may be filled with granular material not found in the healthy tissues, and the lignified elements are often stained and plugged, but all these features are due probably to the infiltration of some substance produced by the fungus in the roots [13].

There is no definite evidence to the effect that any particular type of soil gives encouragement to this disease, but on this point as well as on the behaviour of the fungus in acid and alkaline soils further information is desirable. The disease is favoured by damp climate, high soil moisture, and a moderately low temperature. The sporangia germinate to best advantage, with a maximum production of zoospores, between 14° and 18° C., but grow poorly at 4° and 22° C. [6, 13]. The progress of the disease in the field is fairly slow, but on sloping ground, infection appears to follow the flow of water, and the trouble is much worse on the low-lying parts than on higher ground, or in any part where drainage is impeded.

For the eradication of this disease of strawberries long rotations are essential. Though the trouble is not known to affect any other plant, it is observed to be worse after potatoes than any other crop and is least virulent when the crop follows cereals [2]. The most prolific means of spreading the disease has been proved to be

the planting of ' runners ' from infected stock, and the rapid manner in which the trouble has spread throughout Scotland has no doubt been due to the distribution of apparently healthy plants from a few infected sources [1-3]. Soil disinfection with a view to destroying resting oospores or mycelium has not been helpful since the contamination has been found as low down as two feet or more. The disease appears to yield little to any changed methods of cultivation, and special manurial treatments have, so far, not been encouraging [2, 16]. But highly successful results have already been attained in Scotland, through selection and breeding, and though the work has been greatly hampered by complications arising from virus troubles, good progress has been made in the production of varieties resistant to the disease [16, 17]. In Britain, the varieties Huxley, Tardive de Leopold, Western Queen, Sir Joseph Paxton are the most susceptible kinds ; Royal Sovereign, The Duke, Bedford Champion, Aberdeen Standard, John Ruskin, Overtoun, Scarlet Queen are intermediate, while Pillnitz, Perle de Prague, Cambridge Early, Oberschlesien, and Auchincruive Climax [17a] show considerable resistance. In the United States new resistant kinds have been found, among them Pathfinder [24] and a variety called American Aberdeen [18], which, though of poor fruiting qualities, has proved over a number of years to be resistant both in Britain and in the United States [9], and results have already shown that its high resistance has been transmitted to a large proportion of its progenies derived by crossing with susceptible varieties of good quality [4, 17, 18, 25] ; but the resistance of these and of other promising varieties is apparently not maintained under abnormally wet conditions of the soil [16] and under a wide range of environmental conditions ; but, so far, Climax has resisted all infection [17(a)].

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Leaf Spot of Strawberry, *Mycosphaerella fragariae* (Tul.) Lind.

' Leaf spot ', though a common disease of the strawberry, is not very harmful as it usually affects only the outer leaves and petioles. The spots are small and round, about $\frac{1}{8}$ inch in diameter, reddish or purple at first, later turning grey or almost white, but may still retain a dark-red colour around the margin. They are distributed irregularly over the entire surface, and may sometimes be so numerous as to reduce considerably the assimilatory area of the leaf, with the result that the fruit remains small and the crop generally poor (Fig. 372). On plants under glass, spots may also be seen on the stalks of the flowers or those of the fruits [8]. Sometimes when the disease takes a more severe but unusual form, large areas of the leaves turn brown, in which case entire leaves are killed [6]. A peculiar form of this disease has also been reported from Maryland and North Carolina [1], the tiny achenes on the berries being found to turn black (' black seed ' disease); this feature detracts considerably from the appearance of pale-coloured varieties of the strawberry, but as it occurs only on one or two parts of the berry the fruit appears to suffer little harm.

Leaf spot of strawberry is caused by *Mycosphaerella fragariae*, an Ascomycete of the group Pyrenomycetes ; the conidial stage has been variously placed in the genera *Ramularia* and *Phyllosticta* [3]. Conidia may be found in abundance on leaves, petioles and flower stalks. They arise on simple conidiophores which are developed in tufts on small, ill-defined stromata between cuticle and epidermis ; the conidia are elliptic or cylindrical, hyaline, and measure from 20 to 40 by 3 to 5 μ, and may have 1 or 2 septa ; they germinate



FIG. 372.—Leaf spot of strawberry (*Mycosphaerella fragariae*) (photo by Wolf)

easily in rain water or acidified tap water [5]. The perfect *Mycosphaerella*-stage develops its tiny, black perithecia in the autumn on spots on old leaves, but they are not common ; the asci contain 2-celled, hyaline ascospores which measure 15 by 3 to 4 μ. In place of, and somewhat resembling, perithecia, the leaf spots may bear small black sclerotia which are capable of surviving through the winter on the fallen leaves to give rise to crops of conidia for renewal of infections in the spring. But it is not clear, apart from the rare occurrence of perithecia, that the production of sclerotia is the only means whereby the fungus can tide the winter. Entirely new spots have been observed in early spring to break out on the over-wintered dead leaves on the ground in so short a time after they are brought indoors as to preclude all possibility of their having originated from newly formed conidia, from which it would appear that these new spots on the old leaves developed from fresh growth of mycelium that had survived in the leaves [2, 4].

Infection may occur at either upper or lower side of the leaf and may be direct through the cuticle [2], or stomatal [4], the fungus travelling between the cells of the mesophyll without forming haustoria [4]. When the fungus appears on the tiny achenes, as above mentioned, infection travels in through the stigmas at flowering time, the fungus thereafter finding its way not only into the tissues of the unripe achenes but penetrating also the soft adjacent tissues of the pulpy receptacle. Infections of young berries were obtained in the greenhouse both with conidia taken from leaf spots and ascospores from perithecia, as well as with inoculum collected from the blackened achenes, and the typical symptoms on the leaves were also reproduced following the inoculation of young leaves from the same source [1].

As the fungus appears to survive over the winter on dead leaves on the ground, these should be gathered and burned ; in large plantations, after the fruit has been picked, straw may be spread and burned on the soil so as to kill any infected material still left on the ground. If the disease breaks out early the plants may be sprayed with 4 : 4 : 40 Bordeaux mixture, as soon as growth is well started, that is, when the leaves are about half grown, and the operation may be repeated frequently until the fruit is about half size [7]. For fresh planting, runners should be selected free from disease, and should any outer leaves become spotted their prompt removal and burning will help greatly to prevent the trouble from spreading.

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Yellow Edge and Crinkle Diseases of Strawberry

' Yellow edge ' and ' crinkle ' diseases of the strawberry are of considerable economic importance in Britain, and account for much deterioration of the crop, here and abroad. While they are generally recognised as two more or less distinct

diseases attributed to different viruses, or virus complexes, there is still a good deal of uncertainty about the separate identity and properties of the component infective principles. Since both of these diseases appear to have ' mild ' and ' severe ' phases, it may well be that they are caused by virus complexes. Moreover, it has been asserted that no case of yellow edge is ever free from taint with crinkle, and it is by no means certain that many reports of the occurrence of strawberry diseases, referred to specifically as yellow edge or crinkle, are actually not mixed infections of the two together.

(a) *Yellow Edge*

Yellow edge first came to prominence in England in 1930 [9, 10], and has since been acclaimed in many parts of Britain as being the main cause of deterioration in the varieties Royal Sovereign, Sir Joseph Paxton, and others. The disease is present in Europe, Australia, New Zealand, and the malady known in Canada and the United States as ' xanthosis ' is now believed to be identical with yellow edge [2, 3, 6, 7, 8, 22, 26, 27]. In some areas, as in Queensland, it is reported to be more prevalent than crinkle [2], while in other parts, as in Rhodesia [15], it is frequently found in association with a severe form of the latter. In Britain, yellow edge and severe crinkle, in bad seasons, may reduce an average crop of strawberries from 2 tons per acre (3 to 4 tons are not unusual) to little more than 15 to 25 cwts. per acre [31]. In 1937, in the Isle of Ely, a normal yield of 6 tons per acre was reduced to barely a ton [4].

Strawberry plants affected with yellow edge show the symptoms to an increased degree as the plants get older, and the symptoms are most marked from mid-September to the end of October. During this period the outer leaves remain more or less normal, the symptoms being confined largely to the central leaves, which show the characteristic feature of the disease as a yellow discoloration extending all around the marginal areas of the leaflets (Fig. 373). This marginal chlorosis is the principal feature whereby yellow edge can be distinguished from crinkle, in which the chlorosis is unevenly distributed in small areas all over the lamina [12]. Plants affected

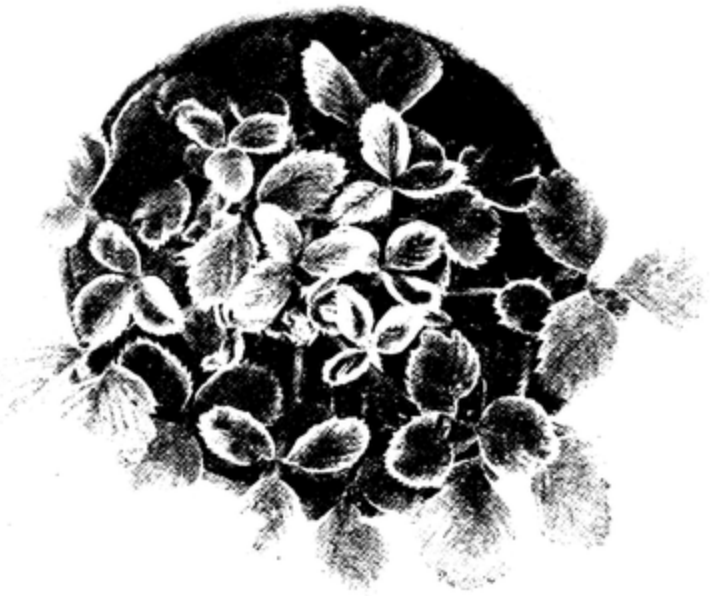


FIG. 373.—Yellow edge of strawberry. Plant of variety Royal Sovereign showing symptoms of yellow-edge virus infection (photo by Rogers, King, and Massee, *Sci. Hortic.*, by permission of the Editor, and East Malling Research Station)

with yellow edge also show an irregular, generally upward curling of the leaf margins, a downward curling of the midrib, and frequently a distortion of the entire leaf. There may also be a considerable reduction in the size of the laminae, and the leaf stalks are much shorter than those of healthy leaves and the usual red pigmentation is reduced or absent [10]. Moreover, affected plants have a reduced capacity for forming runners. The fruit is not directly affected. But these symptoms are by no means general. Some plants in their maiden year exhibit the symptoms in a *mild* form, and apparently may continue to do so indefinitely, while other plants get progressively worse, presenting a distinctly *severe* phase of the disease. It appears that once a plant shows mild or severe symptoms in one season, it tends to repeat them in the following year, and while sometimes they may become slightly worse, they remain, however, in the same category. These variations in symptom-expression among individual plants are not associated with nutritional changes, no differences being observable in healthy or infected plants when grown in different soils; for instance, of greenhouse compost or woodland soil or of peat, greater variation in fact being seen in any one soil than between plants in different soils [19].

Since the diversity of symptom-expression presented by plants affected with yellow edge is so pronounced, it has been suggested that yellow edge may be the work of two or more virus strains in the plant; experiments at East Malling [16, 19] have not confirmed this hypothesis, but recently give support to the view that both yellow edge and crinkle may be caused by virus complexes. These investigations (concerned with an enquiry into the method of transmission of these diseases) have led to the suggestion that a common infective principle, capable of transmission, exists in yellow edge and in both types of crinkle, mild and severe, this component being probably the virus of mild crinkle. Recent observations at East Malling [25a, b] now show that yellow edge is caused by the combined action of two distinct viruses, namely the mild crinkle virus already referred to, and another, called the ' mild yellow edge ' virus. But this does not exclude the possibility that other viruses or combinations of viruses may also cause yellow edge. Thus, a virus isolated from plants affected with ' severe crinkle ', in combination with the virus of ' mild yellow edge ', also produces severe yellow edge. The obligate nature of the association frequently occurring between crinkle and yellow edge has, therefore, been demonstrated.

Observations of the progress of yellow edge on such varieties as Royal Sovereign have shown that the incidence of disease depends on the operation of a variety of factors. At times the symptoms are well marked, while at other times they are masked. This suppression of symptoms is apparently related to the interaction of three factors, namely, seasonal weather conditions, soil conditions, and age of infected plants. Furthermore, it has been demonstrated that the symptom-picture varies in its intensity according to temperature, and especially soil-moisture conditions, being enhanced in damp weather and inhibited or appreciably reduced in hot, dry weather [15, 19, 31]. Favourable conditions for the disease generally prevail, in the south of England during September and October, sometimes also in May and June, and are most marked under conditions of abundant moisture with an air temperature of over 60° F.; in south Queensland, the symptoms of

yellow edge and crinkle were more pronounced in the cooler than during the warmer months, while at temperatures of over 80° F. the symptoms of yellow edge were masked [2].

The virus of yellow edge has been variously named : *Fragaria virus 1* (K. M. Smith); *Strawberry yellow edge virus* (Harris 1933); *Strawberry yellows virus* (Plakidas 1926); *Strawberry xanthosis virus* (Plakidas 1927); *Strawberry virus 1* (J. Johnson Classification). The virus is transmissible by grafting, and is not conveyed in the seed. Attempts to transmit the viruses of yellow edge and crinkle by mechanical inoculation have failed [15]. Strawberry viruses are not sap-transmissible and the separation of the component viruses from the mixtures in the plants is accomplished by the aphid vector. The principal insect vector for both these diseases in Britain appears to be *Capitophorus fragariae* [20, 20a], in which the virus of mild crinkle persists for only a few hours, whereas that of mild yellow edge remains for several days [25b]. This differential persistence in the aphid vector has been put to actual practice as a means for separating the component viruses from mixtures present in the plants [25a]. Observations in Bangor, North Wales [29], showed this vector to tide the winter on strawberries, in the apterous viviparous state, but viviparous alatae and nymphs with wing buds have been found in late autumn and winter ; the alatae appear to migrate over a distance of at least a mile and there is also considerable movement of apterae within a crop. This vector, moreover, will exist on silver-weed (*Potentilla anserina*), *Potentilla sterilis*, wild strawberry (*Fragaria vesca*), and *Fragaria moschata* although it has never been observed on natural cultures of *F. vesca*, and only rarely on the other species and genera. Of other aphides found in the same locality *Macrosiphum solanifolii* was most abundant and bred very readily on strawberries in the spring and early summer, and was able to survive the winter in the crop, but in other areas it has not been observed to be a vector of strawberry diseases. In 1940 and 1941 the virus of strawberry crinkle was transferred from Royal Sovereign to *Fragaria vesca* by the vector *Pentatrichopus* (*Capitophorus*) *tetrarhodus*, producing symptoms identical with those set up by *Capitophorus fragariae* [32]. In New Zealand, yellow edge and both types of crinkle were successfully transferred by *Capitophorus potentillae* [1].

(b) *Crinkle*

Crinkle first came to prominence in England in 1934, on the variety Royal Sovereign [21]. It was first recorded in America, in Oregon in 1925 [35], and is widespread in the Pacific West on certain varieties of strawberries, notably those of the Marshall group. Though the disease causes considerable deterioration in the crop, the plants are not killed and may continue to yield, but at a reduced rate.

As already stated, the characteristic symptom of crinkle is a localised chlorosis of the leaf (Fig. 374), in contrast to marginal chlorosis presented by yellow edge. At first the chlorotic areas are very small, merely pin-point flecks, later increasing somewhat with expansion of the lamina. A rugose condition of the leaf seems to follow the chlorotic areas, but with no very definite pattern. The leaf spots show to better advantage viewed by transmitted light, and often the very pale centre of



FIG. 374.—Crinkle of strawberry. Plant of the variety Royal Sovereign showing symptoms of crinkle virus infection (photo by Rogers, King, and Massee, *Sci. Hort.*, by permission of the Editor, and East Malling Res. Station)

these yellowed spots becomes, first red, and then brown and necrotic. Besides this mere stippling effect, some leaflets exhibit a more uneven type of chlorosis. The yellowing may extend in streaks along a few veins towards the midrib, or the veins, for the most part, may be cleared. The above observations, made in Oregon in 1932 on plants suffering from crinkle alone, are supplemented by the statement that the leaflets, as a rule, were yellower towards the margins. Nevertheless this new discovery of crinkle in Oregon was believed to be distinct from the already known ' yellows ' (xanthosis) but which appeared to be restricted to California [36]. From what has since transpired about the complex nature of these strawberry diseases it is not clear whether the symptoms attributed to crinkle alone are not those of mixed infection of yellow edge and crinkle. But another quite distinctive symptom of crinkle is a definite change in the pattern of the marginal serrations, the teeth being bigger, the whole leaf margin presenting an unnatural, lobed appearance. In general, at any season of the year, infection with crinkle imparts to the host a lighter shade of green than normal, and in the autumn, whether in field or greenhouse, the plants lose much of their erect growth. During favourable growing periods affected plants may throw off all these symptoms, but they can still be picked out from healthy plants by their lack of uniformity of colour and absence of a smooth leaf surface characteristic of normal plants. Finally, there is a tendency in affected plants to the development of a downward curling of the lamina or a cupping upwards of the leaf margins [36].

The virus of crinkle disease has been named *Fragaria virus 2* (K. M. Smith);

Strawberry crinkle virus (Zeller & Vaughan 1932); *Strawberry virus 4* (J. Johnson Classification).

The wild strawberry of the woods, *Fragaria vesca*, is never found to be naturally infected, but affords a useful ' indicator ' host in the greenhouse and experimental plots, for it readily submits to infection, either by grafting of plants affected with yellow edge or crinkle, or with both together, or by exposure to the viruliferous vectors above named. On this differential host the symptoms transmitted from these three sources are highly variable and difficult to analyse, and only a few salient features can be given. The symptoms transmitted from plants affected with yellow edge alone are not easy to define; neither is it easy to distinguish plants of *F. vesca* with crinkle alone, from those infected with both viruses. When *F. vesca* is grafted to a plant with yellow edge, the most distinctive feature developed is *leaf curling*, and this has not been seen in plants affected by crinkle alone. Grafted to a plant affected with crinkle alone, the salient feature is a *chlorotic speckling*, while *F. vesca* grafted to a plant affected with yellow edge and crinkle exhibits most diverse features which have been grouped into three stages, the salient differences being (*a*) no reduction in leaf size, midrib curled backwards and downwards; leaf with a rounded outline, with prominent dark-green, raised-up areas; sometimes chlorotic speckling present, attributed to crinkle: (*b*) leaves reduced to one-half or quarter normal size, asymmetrical, not curled, local laminal puckering, chlorotic spottling, and a reddening may also be present: (*c*) leaves extremely minute, laminae greatly reduced or wanting; sometimes these leaves do not even show typical crinkle symptoms, but remain very small, smooth and green, except for a reddening in the region of the veins. At any stage bending and twisting of the tips of the young developing stolons may be present, and young runner plants produced by these stolons are severely affected [16].

Samples more or less free from these diseases have been found among certain selections of the susceptible varieties Royal Sovereign, Sir Joseph Paxton, and King George. These selections showed no visible reaction in *F. vesca* and were therefore considered to be virus-free. While selections of other varieties showed no symptoms in themselves, when grafted to *F. vesca* they transmitted the disease to the indicator host, and were therefore symptomless carriers. But one strain of the variety Huxley Giant appeared to be free from any virus, and this is being propagated at East Malling for further trials. It is clear that strawberry species and varieties differ to a great extent in their tolerance to the viruses here discussed, all grades from very susceptible intolerants to symptomless carriers being found [16, 34]. In New Zealand, in 1940, Royal Sovereign proved very susceptible, and 70 per cent. infection was shown by certain lines of Marguerite, while Laxton's Noble and Captain Cook showed moderately high infection [33]. At Wisley, in 1940, Aromatic, Campbell's Seedling, and Redbourn showed definite symptoms when infected, while Corvallis, Duke of Kent, Huxley Giant, Ettersburg 121, Oberschlesien, Pillnitz, and Tardive de Leopold showed no symptoms, being all symptomless carriers and, therefore, potential carriers of infection [24].

For the control of strawberry virus diseases it is clear that growers must select from the virus-sensitive, so-called *Fragaria virginiana* or British group which includes the susceptibles, Royal Sovereign, Sir Joseph Paxton, Stirling Castle, etc.,

and not from the tolerant *F. Chiloensis* or Continental group, which includes the symptomless carriers, such as Madame Lefebvre, Madame Kooi, Brenda Gautrey, Western Queen, and others already mentioned [31]. In the matter of cultivation, special beds for propagation from runners should be situated at least half a mile from all other areas under strawberries. New fruiting plantations must also be isolated from older beds, and all heavily infected, older fruiting beds should be ploughed up after bearing, say, three crops [28]. A preliminary roguing of established runner beds should be carried out in June and the final inspection and roguing made in September and October ; new plantings of runners should be similarly inspected and rogued [19, 23]. Since an increasing number of runners succumb to yellow edge as the plants grow older, the plants should be grown for one year only and propagated annually, observing good cultivation methods so as to avoid overcrowding [24]. Although the vector *Capitophorus fragariae* is common on strawberries in most districts, it does not appear to migrate widely in the same way as, for instance, *Myzus persicae*, and isolated plots in non-strawberry-growing districts are likely to remain free from infection through its agency for long periods [29]. Such plots of healthy plants for runner production may be protected against the greenfly vectors by spraying (nicotine 8 oz., soft soap 8 lb., water 100 galls.) first in April, secondly before flowering, and again after picking.

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Crown Gall of Raspberry, *Bacterium tumefaciens* S. & T.

Crown gall disease attacks a wide range of plants which include herbs, shrubs, and trees, but is of little economic importance except on fruit trees. These belong mostly to the *Rosaceae* family, namely apple, pear, quince, plum, cherry, apricot, peach, raspberry, blackberry, loganberry, and many from diverse groups, e.g. grape vine, hop, beetroot, chrysanthemum, dahlia, and others (Fig. 142).

As the name suggests, the galls develop mostly at the ' crown ', that part of the stem at, or just below soil-level, but in a few hosts, as in the red raspberry (the European species *Rubus idaeus*), they may also occur at various places on the long shoots, well above ground (Fig. 375). Crown galls should not be confused with the so-called ' burr knots ' frequently found on fruit trees ; these abnormal knots are often covered with incipient buds and roots and, unlike true crown galls, are not always of parasitic origin, and do not develop like crown galls.

This disease is often troublesome to nurserymen and fruit growers, because of its occurrence on stocks which are propagated by stools and layering, and particularly so when incipient stages of infection are very difficult to detect, and which may, therefore, be carried by apparently healthy plants. Crown galls vary considerably in size, according to the host, from that of a pea to lumps several inches in diameter, and are naturally a heavy drain on the food reserves of the plant ; they interfere greatly with the proper development of the host tissues in their vicinity, causing a decided check to normal growth, affected plants being often stunted and deformed.

The study of crown gall first came into prominence, in 1907, in the United States, where it was discovered that galls on a species of chrysanthemum (' Paris daisy ') were due to bacterial infection [31]. From the galls an organism was isolated, named *Bacterium tumefaciens*, which produced similar galls when inoculated into healthy plants of the same host. This association of tumour-like growths with bacterial infection naturally gave rise to much investigation and considerable speculation as to whether the origin and mode of development of bacterial galls in plants would throw any light on the cause of animal cancer. But the

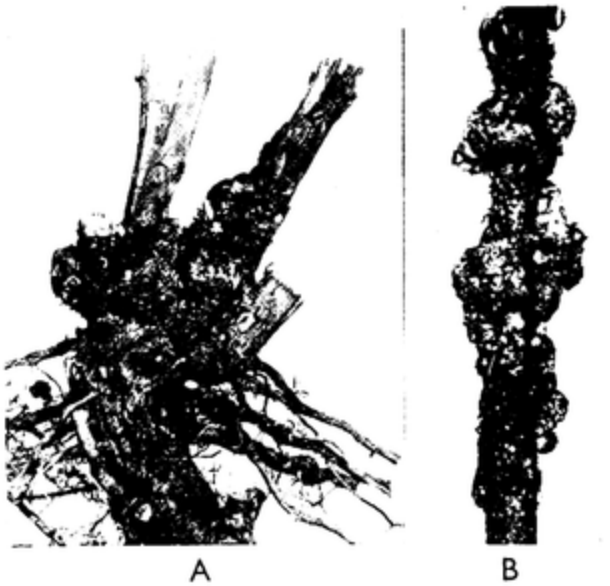


FIG. 375.—Crown gall (*Bacterium tumefaciens*) of red raspberry. *A*, the gall at the base of the plant (photo by Foister & Noble). *B*, the galls on the canes, above soil-level (photo by Wormald)

evidence so far produced is not convincing that the pathological anatomy of crown gall justifies any parallel being made between this disease and animal cancer [9, 17, 18].

Crown gall has probably been in existence in Europe for over eighty years [18]. First report of the occurrence of the disease in Britain was made in 1921, again on the Paris daisy [36], and extensive studies have since been carried out in this country [29, 37, 38, 39], in the United States, and on the Continent, especially in relation to its attacks on fruit trees [1, 2, 3, 4, 17, 18, 21, 25-28 b].

While the choice of any particular host for the description of crown gall is of little significance, the selection of the red raspberry here is made because of the undoubtable association of the galls on this plant with the organism *B. tumefaciens* and of the indisputable evidence of the pathogenicity of the organism isolated from the galls on raspberry, to other hosts, such as apple, tomato, etc. With the adoption of this host it is important to note, however, that crown gall of red raspberry in Britain appears to differ somewhat from the disease on this particular host in America [1, 2, 3]. These differences are discussed below, and whether they are due to genetical differences in the variety of the host is not known, but it is probable that the specific origin of the European red raspberry is not the same as that of the American plant of that name.

On English wild, as well as on the cultivated red raspberry, galls may be seen to develop throughout the growing season at two places on the plant, namely at the crown, and at variable points along the canes. The galls formed at the crown, at first a dirty-white colour, later become a brownish colour, and are very variable in size and shape, usually about the size of a walnut. On the growing canes they arise at variable points, and resemble more or less elongated warts of firm texture, some appearing to join together, while others remain isolated; stem galls may also arise so early at the extreme tip of some of the canes that growth is entirely checked. While all these symptoms are also reported to be the same on black and purple kinds of raspberry in America, on the red variety in that country, crown gall appears to be confined to the roots. It is true that galls may also arise on the roots of the red raspberry in England but they are different from crown galls, being much harder and of a more woody texture than true crown galls, and all attempts to isolate an organism from them has failed and, so far, the cause of root galls on English raspberry is not known. Similar galls have also been found on the roots of red raspberry in Germany but apparently they cause little damage [11].

Bacterium tumefaciens is a short, rod-shaped organism measuring, according to Riker [25], from 1·0 to 1·6 by 0·4 to 0·7 μ, as compared with the dimensions given by Smith and Townsend [31], from 1·0 to 3·0 by 0·4 to 1·8 μ. It occurs singly or in pairs, has 1 to 3 polar flagella, is aerobic, non-acid fast. Gram-negative; does not reduce nitrates; gelatine is not liquified; on agar the colonies are small, circular, and white. On nutrient agar, with 1 per cent. dextrose, at *p*H 6·8, the optimum temperature for growth was 22° C., growth being considerably reduced at 30° C. [27]. The organism isolated from galls on black raspberry canes in Ohio and other States, appears to differ consistently from the crown gall strain originally isolated from apple, and it is suggested that the two are different, the raspberry cane organism being a variety of *B. tumefaciens*, if not actually a separate species [2]. But numerous strains of the crown gall organism exist which vary in their ability to infect certain hosts [13, 39].

The presence of the bacteria in the interior of crown gall tissue has never been satisfactorily demonstrated, and the organisms have been observed mostly on the outer surfaces of the galls. In fact, the diminishing population of the bacteria, as the inner parts of the galls are reached, has given rise to much discussion and speculation as to the true nature of crown gall disease. When the galls finally disintegrate, the bacteria, during wet weather, are released and washed down the stem or crown into the soil and under conditions of a humid atmosphere continue to be formed within the living gall remnants, and with further growth of the gall the organisms on the exterior are again released from the gall surface [3, 29]. The germs are reported to live in the soil for variable periods, from a few months to over a year, and to survive the winter in the field [1, 2, 21, 24].

There is general agreement that the organism of crown gall enters the host only through wounds, such as would be caused by pruning, grafting, removing cuttings, etc., or through punctured tissues. Such wounds may be caused by insects or their larvae, or through breakage of epidermal hairs or prickles, or by a splitting of the bark [4]. In the United States, arthropods are said to cause most of the injuries to the underground parts of the raspberry through which infection occurs [2, 3], and the profusion of insects which followed a rotation of 'blue grass' was believed to be responsible for high incidence of crown gall on raspberry in Minnesota [12].

After the entry of the bacteria into the wound, the tissues of the host are stimulated to localised growth around the wound (apparently a period of four days must elapse before the host cells are actively stimulated [7a]), but, as already stated, the bacteria are apparently confined to the outer surface of the hypertrophied tissues, and it is very difficult to follow them into the inner parts of the galls, and increasingly so into the adjacent tissues of the host. Some suggest that this diminishing appearance of the bacteria as the internal tissues are reached may be due to the action of a bacteriophage [10, 15].

The changes which take place in the host cells during the development of a gall, as already mentioned in Chapter VI, are very similar to those which occur in the normal formation of callus in wounded tissue [35]. A few days after the inoculation of a wounded surface, the bacteria may be seen to have entered the vessels and sieve-tubes that may lie in the path of injury, and to occupy the intercellular spaces of the thin-walled parenchyma such as that of the cortex, and cells in actual contact with the bacteria early develop necrotic changes similar to those which follow on wound reactions. There is, however, no production of cork on the surface of the galls and as the dead cells on the surface of the galls continue to be thrown off owing to the meristematic activity of gall cells below them, vast numbers of the bacteria are carried with the sloughed tissue into the soil.

Gall formation in the genus Rubus has been studied to better advantage in *R. procerus*, the Himalaya blackberry, than in the red raspberry in which, however, the salient features are much the same. Cell multiplication in the host, leading to gall formation on the stem or cane, starts here in the pericycle adjoining the vascular bundles (Fig. 148), and consequent upon this meristematic activity a swelling of the wounded region occurs. Within the hypertrophied tissues some of the cells continue to be meristematic, adding new parenchyma, both internally and externally, to the dividing cells; some of the new cells lose their contents

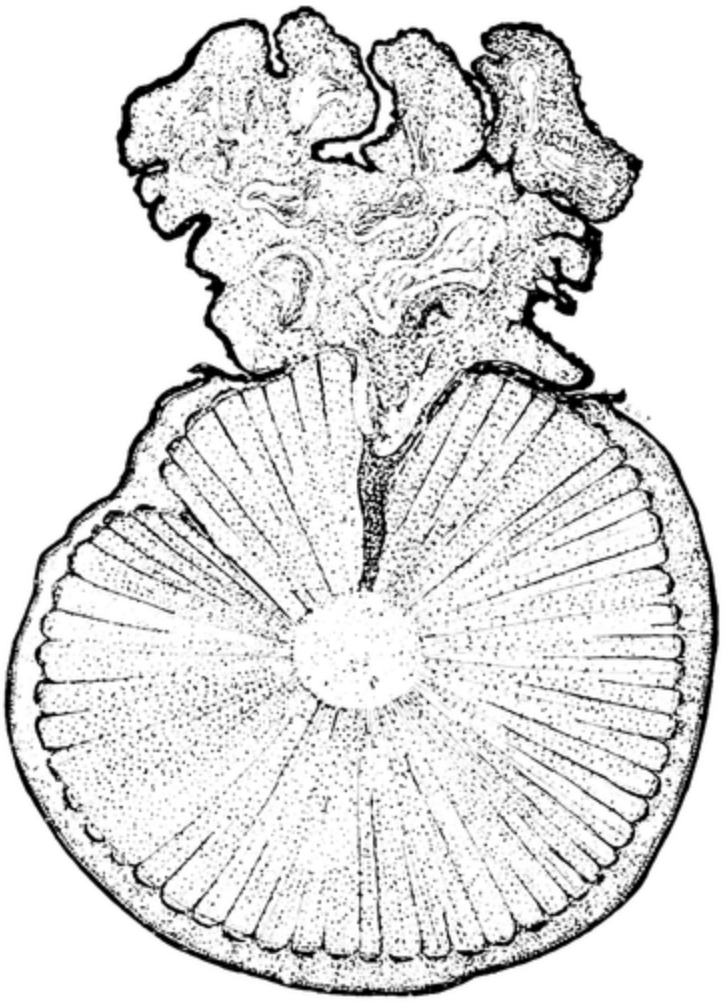


FIG. 376.—Crown gall (*Bacterium tumefaciens*) of English red raspberry. Cross-section of a cane showing the gall arising from the tissue outside the vascular bundles, and disrupting the bark (×4) (see also Fig. 146 D)

and become transformed into lignified tracheids (Figs. 146 D, 148, 149, 376). Within the growing gall, the meristematic cells are now seen to be scattered at various points and separated by secondary parenchyma derived from them. As these various meristems continue to divide actively they contend against each other, with the result that there is much crushing and distortion of the gall parenchyma formed by them. As some of the parenchyma becomes converted into tracheids, the vascularisation of the gall presents a most irregular pattern, the tracheids appearing, here and there, in groups or bundles of most fantastic shapes. In this host there is no extension of gall activity into the pith of the cane, and the vascularisation of the gall depends entirely upon the conversion and lignification of groups of the secondary parenchyma into tracheids (Fig. 149) [16a].

While an infection at a single point on the plant is usually followed by the development of a single gall as above described, it is by no means clear how a single infection can account for a *series* of galls, separated from each other, and at a distance from the point of inoculation. The series of aerial galls common on the canes of the red raspberry presents us with this problem for the elucidation of which reference must be made to a number of studies of crown gall on Paris daisy, Nicotiana [29], and sunflower [26]; in these, stress is laid on differentiating between these aerial, so-called secondary galls and true secondary galls; the latter have, however, only been produced under artificial conditions, and have not, so far, been observed under natural conditions. The naturally-occurring, so-called secondary galls are better referred to as aerial galls. They occur only when the inoculation is made into *very young* tissues, such as in the growing parts of the shoot. Thus, following upon a single horizontal needle-thrust inoculation through the tender apical bud of Paris daisy, a case is cited where six galls, developing at the base of the punctured bud-leaves, eventually became widely separated from each other by the elongation of the internodes in the normal manner of stem growth. These galls had all arisen as a result of the *single*

inoculation having caused six wounds along the track of the needle in the rudimentary leaves, whilst still enclosed within the bud, and which became separated by the subsequent growth of the shoot. Actually, therefore, these six galls, being traceable each to a separate wound, are, all of them, independent galls. Moreover, each of the six galls carried, and further developed, its own load of bacteria, and from which they were readily isolated [29]. Similarly, in the sunflower serial galls on the stem occurred only when the original inoculation was made into a stem *actually growing*, or close behind a bud about to elongate [26]. True secondary galls produced, as already stated only under experimental conditions, arise *at a distance* from one or more primary galls which alone are associated with actual wounds. That is, true secondary galls *arise from within* the injured shoot. Moreover, such galls are smooth, not rough like the aerial galls described. Only when the smooth secondary galls, such as those observed on Nicotiana [32], break out to the surface from pith or cortex, does the surface become rough and, as in the other galls, the bacteria are again largely confined to the rough surface. A true secondary gall begins with the subdivision of a number of cells adjoining a part of the protoxylem in which bacteria had collected, and which were released into the intercellular spaces of the pith when the walls of the protoxylem cells became torn during the elongation of the shoot, this breakage being a more or less normal occurrence during growth [34]. In order to accommodate meristematic activity, stimulated by the presence of the organisms in the pith, there results a slight general displacement of the original pith tissues but there is no intrusion of the new cells between the old, at least not for any appreciable distance. It would appear, therefore, that these internal galls arise as the result of the diffusion into the area where they are formed, of some cell-stimulating substance sent out from the infected protoxylem. In the case of secondary galls on the stem of Nicotiana, such galls arose around definite centres of bacteria in the interior of the structures in which they appeared, without evidence of any invasive growth of tumour tissue from a distance having taken place [29]. In the unusually wide intercellular spaces of this plant, the bacteria were found in definite aggregations or zoogloeae, or of zoogloeal strands, advancing through these spaces in the pith. Similar features have also been found in inoculated tissues of the tomato (Fig. 146 A), the bacteria being found in cell spaces below the epidermis and adjacent chlorophyllous tissue [13, 14, 16]. The bacteria are readily isolated from the interior of the secondary galls, and their migration from the original point of infection for very considerable distances through the plant has, in some cases, been conclusively demonstrated. Further experiments with galls on sunflower [7] have shown that pronounced swellings arose on stems that had already elongated, at points several inches above and below the point of inoculation ; in this case the bacteria were found to have travelled through the xylem vessels for a considerable distance, and while it is not claimed that the bacteria may actually move out of the xylem tracts, it is suggested that the bacteria-filled vessels give out cell-stimulating substances which diffuse into the adjacent living parenchyma, there to set up hypertrophy culminating in gall formation.

Following upon the above theory that the appearances generally are not inconsistent with an influence diffusing from bacteria-containing tissues, as for

instance from protoxylem to pith, numerous studies [8, 19, 20, 23, 28] have opened up enquiry as to whether the galls arise as the result of the infiltration of a ' growth substance ' from the bacteria into the host. Numerous experiments have shown that plant galls, similar to crown galls, may be produced following inoculation with various growth promoters, in the absence of bacteria. For instance, galls on broad bean, which were practically identical with the natural ones, were obtained merely by inoculating the hypocotyl with a 3 per cent. heterauxin paste [19], and others succeeded in inducing the formation of galls on the same host by the application of indole acetic acid paste, from 1 to 15 secondary galls arising at some distances from the point of application [8]. The histological appearances, therefore, are strongly suggestive that secondary galls are due to the secretion by the bacteria in the primary gall, of a cell-stimulating substance which is capable of travelling through the stem for considerable distances. It is stated again [20], that the gall-stimulating substance (perhaps auxin *a* or auxin *b*) may quite as likely be a product of the host cells under the influence of the bacteria as of direct bacterial metabolism. A summing-up of the situation [28, 28a] indicates that, while the presence of some kind of growth substance seems clearly to be associated with the development of crown gall, the host range of crown gall, and that of the positive reactions of any specified growth promoter, are not always parallel, and crown gall may occur readily on various plants on which the growth substance has no effect. As in many other pathological conditions in plants, gall formation is probably the result of the interaction of host and parasite in producing a cell-stimulating chemical which starts the cells on their abnormal career of division. Furthermore, it is suggested that the reaction resulting in gall formation may be due, not so much to the production of a cell-stimulating chemical as to the removal of some substance which controls or inhibits cell division. But it is fairly clear that *Bacterium tumefaciens* releases some influence which stimulates gall formation, and that galls may arise without the continued presence of the bacteria [40].

Chemical analyses of galled and healthy tissues have been carried out only on succulent hosts such as beets, and these have shown that while the tumours had a higher content of crude protein, starch, pectins, cellulose, lignin, and total ash than healthy tissues, the latter had a considerably higher content of sucrose than the tumours [23]. The average infected beet contained only about one-half the amount of sugar as the healthy beet, although it contained more of all the other substances. These experiments with galled beets indicate that the galls divert the sugar to build up their own structure, leaving the rest of the beet, therefore, deficient in protoplasmic and cell-wall materials, the host remaining stunted in consequence.

There appears to be, in general, some degree of correlation between the development of crown gall and prevailing temperature, soil moisture, atmospheric humidity, air supply, and maturity of the host. Few specific hosts have, however, been experimented on in relation to the influences of these factors. In experiments on crown gall of tomato [27, 28b], 22° C. was found to be the most favourable temperature for gall development ; galls produced at 18° and 26° C. were much smaller, though the host itself reached maximum height at 30° C., at which gall development was somewhat checked. While the tomato host grew better when

soil moisture was at 80 per cent. moisture-holding capacity, the galls developed better at a lower value of 60 per cent. There appears to be common agreement that one attack of crown gall in no way influences future gall development, and although certain tests for precipitins and agglutinins in the host tissues near the galls and in the galls proper have given negative results, precipitin effects were observed in the tissues of Paris daisy [29]. In comparison with healthy tissue, gall tissue is much richer in oxidising enzymes, and on the basis of total nitrogen content, gall tissue was found to respire much more rapidly than the corresponding host tissue [22, 28].

In investigated cases of crown gall, the disease has been found to develop more extensively on relatively alkaline soils and on well-drained sandy loam than on acid, heavy soils [2, 30]. The H-ion concentration of plant cell sap most favourable to the growth of *B. tumefaciens* was found to be about *p*H 5·2, a value which was close to that of meristematic tissue where galls originated [6].

There is evidence, in the case of apple trees propagated by stocks, that certain varieties of the latter behave differently towards infection with crown gall. Thus ' Paradise ' apple stocks are very susceptible in this respect, while those of ' Free ' types are more resistant. The type of scion worked on the various stocks may also have some effect on the reaction of the stock to infection.

For the control of crown gall on the raspberry, all infected canes should be removed and burned [38]. For its eradication from the field a rotation of at least three years is recommended [5]. Soil treatment has proved to be of little value to check infection, though some measure of control resulted from the application of sulphur, applied at the rate of 50 to 100 grams per sq. metre [33]. As the disease can be carried on nursery stock without visible signs of infection, information should be sought about the history of the stock. Failing this, the plants should undergo quarantine, and cases are known of plants showing no signs of the disease in cold storage becoming severely diseased after planting out, infection having presumably taken place from bacteria present in undetected galls being washed down into the soil, infection taking place through wounds. Young ' sucker ' plants are recommended to be steeped in a 0·5 to 1 per cent. emulsion of uspulun loam [11]. Since crown galls primarily become established only at wounded surfaces, all precautions should be adopted during and after planting to avoid injuring the bark, especially at soil-level.

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Cane Spot or Anthracnose of Raspberry, *Elsinoe veneta* (Burkh.) Jenk.

Anthracnose or cane spot is fairly common in Britain on red raspberry, and may also be found on the loganberry and sometimes on the blackberry. The malady is widely distributed throughout Europe, Canada, Australia, and occurs in all parts of the United States where it is especially severe on the black raspberry (*Rubus occidentalis*) and other species of Rubus, including the blackberry [1, 2, 5, 6, 7, 8, 15, 16]. In Britain the disease has been extensively studied at the East Malling Research Station, where it has been found to attack many of the standard commercial raspberries [9-13].

In Britain, cane spot disease often occurs along with spur blight (*Didymella applanata*), described below, and in certain parts of the United States cane spot, together with crown gall (*Bacterium tumefaciens*) constitutes a limiting factor in the black raspberry industry [15].

Anthracnose causes a dwarfing of the canes and often a die-back from the tip, and the general result is a diminution in the weight of the crop every year with each successive attack. As the name cane spot suggests, the canes are the parts of the plants to suffer most, but petioles, leaves, fruiting laterals, and berries are also liable to attack (Fig. 377). The canes are more susceptible during early growth in their first year, when about 6 to 12 inches high, than when more or less matured. From infections which occur from April to early July, mainly in June, the stems of young canes are dotted over towards the base with tiny, round, purple

spots which, as the canes continue to grow, get bigger and elliptic in outline. The spots, as they increase in size, change colour at the centre to a silvery white and become sunken, but are still bordered by a narrow margin of purple. Owing to tangential growth of the affected cane these lesions tend to split open and thus become converted into small cankers. Such cankered canes attacked in the first season of growth naturally stand the winter badly and are liable to suffer further damage from frost and thaw.

If the canes are infected early the cankers usually go down fairly deep into the tissues of the stem, often as far as the cambium, but after July, when the canes have made good growth, new infections are not so serious and, unlike the earlier sunken lesions, later lesions about the end of August remain small and shallow. Until October or so, these superficial lesions are of a whitish-grey colour, but after that they turn black from the presence on them of the fructifications of the fungus which causes this disease.



FIG. 377.—Anthracnose or cane spot of raspberry (*Elsinoe veneta*). The spots on leaves, petioles, and stems (photos by Foister & Noble)

Spots which appear on leaves and petioles are small and purple at first and, like those on the canes, develop later a white centre and purple margin. On the lamina the small round spots occur chiefly close to the veins and if very numerous the leaves may curl and drop off prematurely. Sometimes the white centres drop out leaving a shot-hole effect.

By the time the canes are in their second, fruiting year, there may be considerable cracking of the bark due to fusion of numerous cankers developed the previous season, and if the cankers are more numerous along one side of the stem than another a distinct twist is developed in the stem. By this time, too, the canes have changed from green to brown and the individual lesions are no longer purple and distinct. The later-formed, superficial spots have meanwhile become more prominent by virtue of the black fructifications of the fungus standing out on them in clear relief, these now being more evident than they were in the autumn of the first year.

As soon as the buds are due to unfold on the fruiting canes, it soon becomes

evident that some buds will fail to develop at all, or produce only one or two dwarfed leaves, while others grow out into dwarfed laterals which fail to fruit, and often the tips of many of the canes are dead [11]. On fruiting laterals the spots are similar to those on the first-year canes, but usually are not serious enough to stop the formation of fruit entirely.

Injury to the fruit comes about chiefly during rainy periods. From splashing of spores from the lesions on the stem to the berries, only some of the drupels may become infected directly, while others mature normally, so that affected berries often show deformed growth, the normal drupels turning red, the affected ones remaining green, or entire berries may remain small and green. Spots on the flower stalks usually occur just before the blossom clusters begin to open; these spots, though small, interfere with translocation and water-supply to the developing berries, causing them to remain small and dry, and in severe infections of the stalks, fruits may not set at all [5]. Affected parts of the berries frequently develop the fructifications of the fungus.

There are two stages in the life-history of the fungus causing this disease. By far the more common is the conidial stage known as *Gloeosporium venetum*; the perfect, ascigerous stage *Elsinoe veneta* (Plectascales) (once named *Plectodiscella veneta*) is only occasionally met with [3, 4, 14]. The conidia are developed from June to November within acervuli on leaf spots and cane lesions, and on the fruiting canes they may be found at all times of the year until the canes are dead. An acervulus may be subcuticular or may arise within, or below the epidermis. The conidiophores are short, unbranched, and bear unicellular, oblong to elliptic conidia, slightly constricted, 5 to 7 by 2·5 to 3 μ [18]. The ascocarps are developed in large numbers on the small superficial spots on the fruiting canes from February onwards; these fructifications are not usually to be found on the sunken lesions of the first-year canes. They are not easily distinguished from acervuli but are slightly larger and more circular in shape [11]. They arise on stromata under the bark, at the edges of the spots. They are not individually distinct, like typical cleistocarps, but are more or less immersed within the erumpent stromatic tissue; they are deep-brown to black, and the periphery of the stroma breaks up irregularly when the asci come to maturity about the end of March. Asci are globose, thick-walled, 24 to 30 μ wide, scattered irregularly and often separated from each other by stromatic tissue; ascospores 5 to 8, arranged parallel within the ascus, hyaline and 4-celled, are 18 to 21 by 6·5 to 8 μ, slightly curved, with constrictions at the septa. When the outer layers of the stroma break down for release of the spores, the asci become exposed and elongate to about three times their length before the spores are discharged into the air. The expelled spores are furnished with a gelatinous sheath and germinate readily in water by budding to form conidia identical with those found in the acervuli [4]. Growth in culture from both conidia and ascospores is slow, and from ascospores on potato-destrose agar, wrinkled heaped masses of minute sprout-conidia are produced, the colour of the culture varying from light-russet-vinaceous to maroon; when the culture is about a month old there is a sparse development of white downy mycelium; conidia are seldom produced in culture but a sudden change so as to increase humidity induces their formation on small conidiophores covering the colony; they are similar to those found normally on the canes [4]. The conidia germinate readily in water at an optimum temperature between 22° and 26° C. (not below 11° or above 30° C.). Though some conidia merely become septated and others put forth germ-tubes with a small amount of branching, growth in general is by

budding from any of the cells, and there results a mere heaping up of the cells, the whole resembling a stroma, without fructification.

Under natural conditions the fungus survives the winter in the spots on the canes and with the advent of spring revives during wet and warm periods to produce acervuli and ascocarps [5]. In the absence of the latter, infections probably occur through conidia, but whether from conidia or ascospores, infections of young shoots growing up around the old infected canes are preceded by the formation of a mass of sprout-conidia on the moist surface of the cane, thus repeating the behaviour of conidia and ascospores in culture. In this way good adhesion of the inoculum to the host is effected, and from the sprouting cells germ-tubes are produced which by branching and interweaving form a number of cushions from which infecting hyphae proceed to penetrate the cuticle. Penetration of the tender tips of the shoots is effected readily and continues throughout the season if wet weather prevails. As already indicated it is the earlier infections that are accountable for the deeper and more destructive lesions on the canes; lesions occurring later when the canes are well grown are shallow and less harmful to the tissues below them. Infections in England usually start about May; after July or August the attacks are merely superficial, but the amount of damage, gauged by the depth of the lesions, depends on the age of the canes at infection and the state of the weather at that time. In early infections penetrations are soon followed by collapse of small areas of epidermis and cortex, followed by hyperplasia of the subjacent phloem. Later, when parts of the cambium below the lesions are killed, the formation of a small canker is inevitable. When the rest of the cambium proceeds in normal fashion to increase the girth of the stem the lesion becomes more and more sunken as the host tissues are raised around it, and with further growth of the stem in a tangential direction the host tissues are torn apart and a canker is formed which may sometimes extend by cracks in the stem, as deep down as the pith. There is thus considerable destruction of the cane tissues due to canker formation, entailing much interference with intake of water and translocation of food. If the next season following heavy cane-infection is dry, the crop matures badly because the vascular tissues of the host have meanwhile been more or less exposed to the drying action of wind, and to injury from frost penetrating the fissures and cankers, so that the berries fail owing to lack of water. Heavy losses are usually incurred under these circumstances, and although a wet season favours infection, the berries, even on diseased canes, mature much better when there is plenty of moisture, than in a season of drought following upon one of heavy infection of the canes [4]. In Britain, as stated, heavy attacks usually occur in late April and May and decrease if delayed until July, the most favourable conditions being the prevalence of warm weather with intermittent rain and sunshine [13].

Much can be done to check cane spot of raspberry by better cultivation [17]. To avoid excessive humidity no weeds, grass, etc. should be allowed to grow around the bushes, and when thinning the stools old canes showing spots and cankers should be destroyed.

Spraying the bushes may be carried out in two stages: (1) when the cane buds are not more than $\frac{1}{2}$ inch long, with Bordeaux mixture 12 : 12 : 100, or lime-

sulphur 1 in 15; and (2) at pre-blossom stage, when the flower buds are just showing white tips, with 6 : 6 : 100 Bordeaux mixture, or 1 in 40 lime sulphur. In Minnesota spraying the young shoots when 6 to 8 inches high with 5 : 5 : 50 Bordeaux mixture plus 1½ lb. of lead arsenate has given good results [19].

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Cane Blight of Raspberry, *Leptosphaeria coniothyrium* (Fuckel) Sacc.

Cane blight of raspberries is well known in Britain [3, 4] and America [1, 2]. One or more canes of a stool may be attacked and the fruiting canes may be destroyed before cropping, from the development of canker at the base. Early symptoms of the disease during the summer appear towards the base in the form of a few irregular greyish patches which later develop lighter-coloured spots bearing the black pycnidial fructifications of the causal fungus. As the fungus develops under the bark, the latter becomes more and more ruptured so that the tissues of the stem are often exposed as deep down as the pith, with the result that a considerable amount of wilting with withering of the leaves occurs. Such exposure of the tissues inevitably brings about desiccation and infected canes are brittle and easily broken.

The fungus has a pycnidial stage (*Coniothyrium fuckelii*) and a perithecial stage (*Leptosphaeria coniothyrium*). The same fungus also causes stem canker of roses (p. 862).

The pycnidia are 180 to 200 μ in diameter, and the pycnospores, 2·4 to 5·0 by 2·0 to 3·5 μ, are exuded through the ostioles in gelatinous masses. The perithecia appear on the cankers usually towards the autumn, and discharge of ascospores takes place in the spring; the asci contain 8 ascospores which are 4-celled, brownish, 10 to 15 by 3 to 4 μ.

Other fungi besides *L. coniothyrium*, as wound parasites, appear to be contributory to cane blight; the midge *Thomasiniana theobaldi* also seems to be implicated [3a, 5].

Infection takes place through wounds on the bark made possibly by insects or through abrasions caused by friction between the shoots, but mainly through pruning wounds. The activity of the fungus at the base of the canes produces cankers which, by injuring the bark and wood, interfere with the supply of water

to the top so that the shoots wilt and die. With the appearance of the fructifications on the canes, the spores formed are disseminated by spattering rain or by contact of one shoot against another, and thus new canes may become infected, but only through injuries. The fungus may live in a mycelial condition in the new canes as well as on old fruiting ones, and may survive in the cankers on old canes for at least four years after the canes are dead. From these sources, by the development of pycnospores and ascospores, abundant infection is secured in spring and summer [1].

The disease does little harm if there is plenty of rainfall during the ripening period; the greatest losses are incurred if a wet spring is followed by drought, for under such conditions the cankers dry and there is insufficient intake of water to the fruit. Infection is reduced by controlling midge infestation [3a].

As *L. coniothyrium* attacks apple, roses, strawberry, and blackberry, raspberry canes should not be planted close to these hosts; the wild red raspberry is also susceptible [1]. All diseased canes should be removed and burned.

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Spur Blight of Raspberry, *Didymella applanata* (Niessl) Sacc.

This disease of raspberry is fairly common in Britain and was first described in 1875 by an Austrian botanist, on material collected at Shrewsbury [17]. The blight is prevalent in Canada and the United States where it was first reported in 1891; it occurs also in Germany, Holland, Denmark, Norway, Switzerland, and Tasmania [1, 2, 5, 6, 7, 8, 9, 10, 11, 15, 16, 18].

Spur blight is caused by an Ascomycete *Didymella applanata* [17]; pycnidia and perithecia are formed. Both the young vegetative canes and the fruiting canes are attacked, and while the disease contributes to a general weakness of the entire plant, the final effect is the destruction of some or all of the fruiting branches or spurs that grow towards the basal parts of the canes, so that only a few of the spurs at the top usually produce fruit [14].

Towards the end of June the lower half of young infected canes may be seen covered with brown or violet-brown, sometimes reddish, discoloured areas, chiefly at the nodes, and more or less encircling the canes at these points (Fig. 378). The bases of the adjacent leaf stalks may also be involved and similarly discoloured, and ultimately the leaves themselves develop a brown shrivelled appearance with a number of brown or black spots in the vicinity of the larger veins. As the result of infection and impaired vitality of the leaves, the young buds in their axils become shrunken and shrivelled, and if they are not killed outright many of them fail to survive the winter so that the provision of fruiting spurs for the following season is seriously curtailed; infected buds which persist until the spring are so weakened that the spurs produced by them often fail to blossom.

During the summer the discoloured areas extend so as to cover practically all

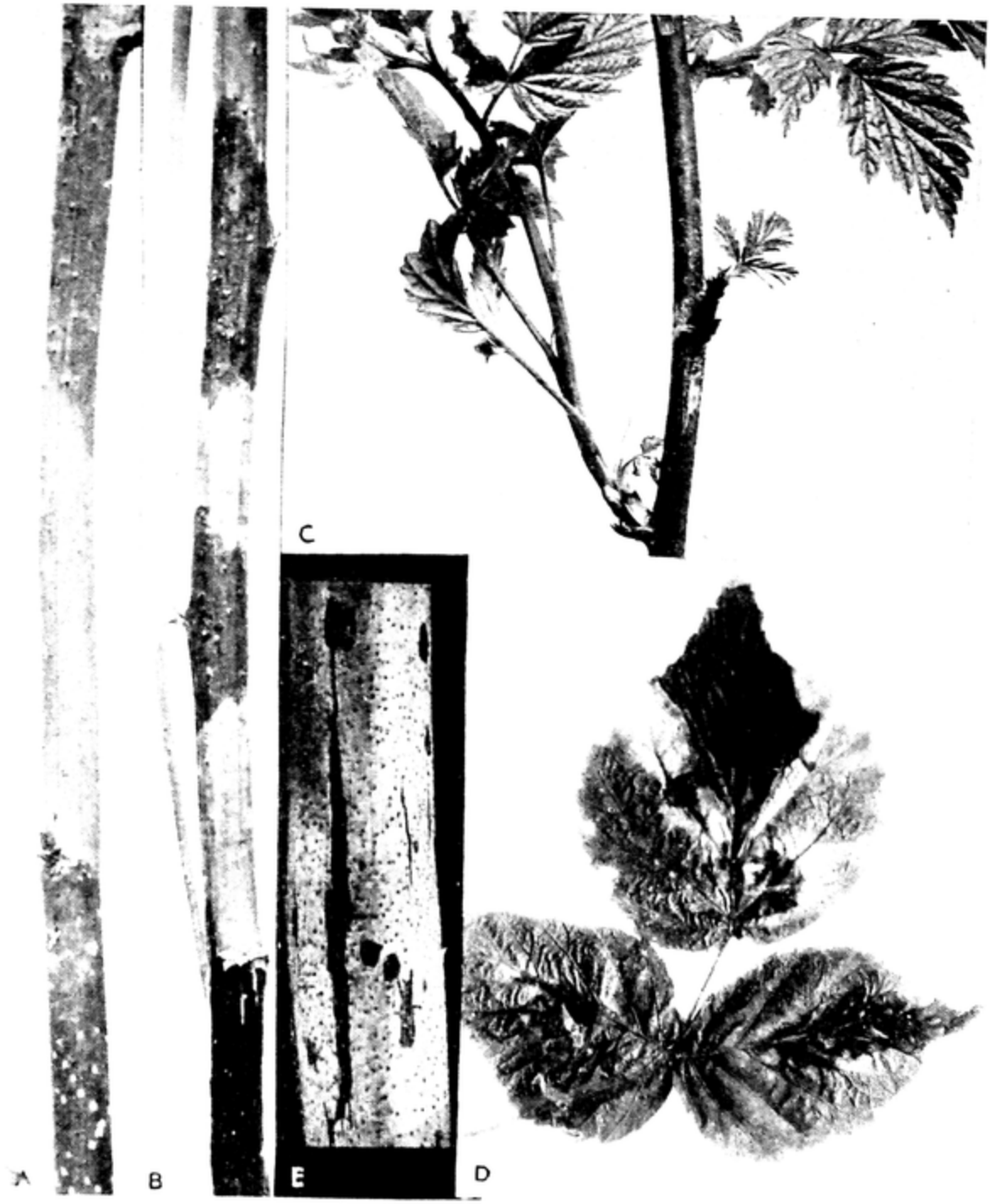


FIG. 378.—Spur blight of raspberry (*Didymella applanata*). *A*, healthy cane of Cuthbert variety, in August. *B*, Cuthbert cane infected, dark lesions around the nodes. *C*, fruiting cane, latter part of May, showing infected node from which is growing a typical dwarfed spur from an infected bud; note split cortex surrounding the node. *D*, natural infection on leaf of Herbert variety. *E*, perithecia on Herbert cane (photos by Koch, *Phytopath.*)

the lower half of the young canes. At this time, if the season is wet, the discoloured parts of canes, leaves, and leaf stalks are covered with tiny brown specks, the pycnidial fructifications of the fungus. These bodies continue to discharge their pycnospores throughout the summer and so many fresh infections arise that the basal parts of the canes become almost completely covered with pycnidia.

With the approach of winter, the plants meanwhile having suffered considerable defoliation through disease, the infected canes turn a grey colour and are then not so easily distinguished from healthy canes which turn a similar colour towards the close of the year. Diseased canes, however, develop a slight silvery effect over the infected areas, and may also show a splitting of the bark, which is often so deep as to expose the inner tissues to frost and decay, and numerous canes may thus be lost during the winter.

The symptoms on the fruiting spurs of the current year repeat much the same features as those on the canes. These short branches, as well as the flower stalks, may, however, be more or less girdled with disease and become lost. Infection does not usually progress from the spur into the flowers and berries. But owing to a general weakness of the plant and lack of nutrition the fruit is small and shrivelled and the shrunken berries remain a dark, purple colour.

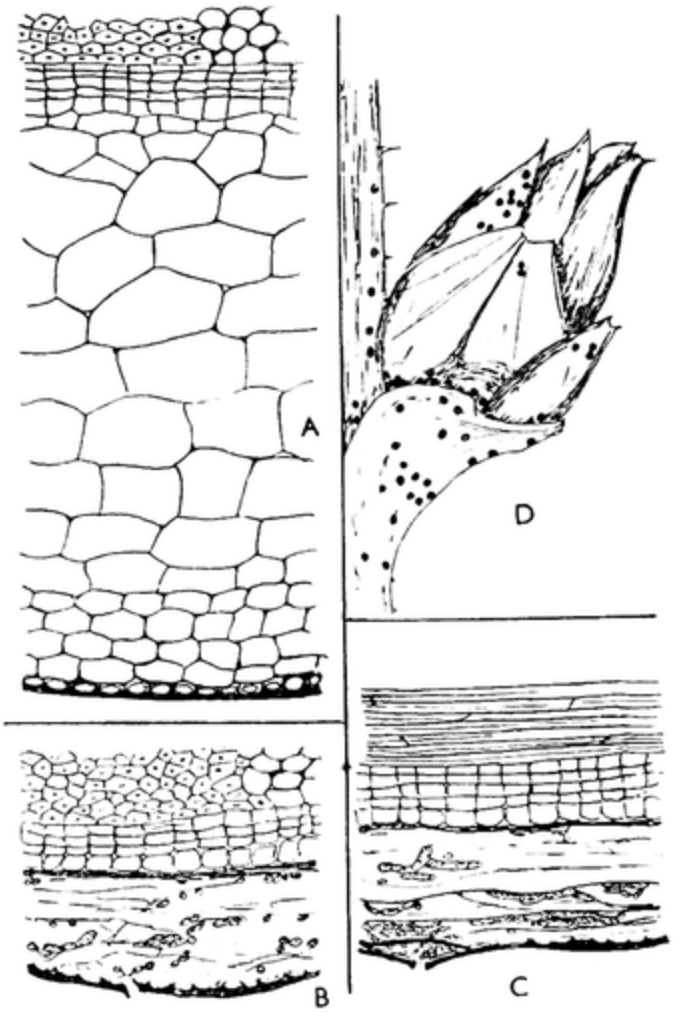


FIG. 379.—*Didymella applanata*. *A*, cross-section normal Herbert cane showing normal amount of cortical tissue. *B*, section of infected cane showing destruction of the cortical tissue (× 400). *C*, longitudinal section of cane showing mycelium and a pycnidium beneath the epidermis. *D*, a naturally infected bud showing the black perithecia of the fungus on the bud-scales ; the bud was apparently killed (after Koch, *Phytopath.*)

The pycnidia (Fig. 379 C) on the host are slightly sunken below the surface, bluntly pear-shaped, ostiolate but not prominently beaked, erumpent at maturity, and measure from 147 to 268 μ by 105 to 231 μ (mean, 208 by 187 μ) ; conidiophores are simple, producing hyaline to light green pycnospores which are mostly bi-guttulate, elliptical to oval in shape, and measuring from 5·0 to 11·2 μ by 1·75 to 3·8 μ (average, 7·1 by 2·9 μ).

The perithecia are developed towards the autumn, on the silvered parts and on the moribund tissues within the fissures of the split canes, and also on the surface of bud scales (Figs. 378, 379 D). These fructifications appear as small black dots, hardly to be distinguished from pycnidia with which, at times, they may be found intermixed, but most of the pycnidia, by now, are defunct, though some may survive the winter, like the perithecia, to become active again in the spring [11]. The latter are not fully developed until about the end of May or early June, at which time the cycle of infection starts again when the perithecia are discharging their ascospores. The spores are ejected in great number and infect the young canes ; infection of new canes from old may also take place



FIG. 380.—*Didymella applanata*. *A*, the perithecium. *B*, germinating ascospores showing anastomosis of two germ-tubes. *C*, ascus and paraphyses (× 330) (after Koch, *Phytopath.*)

by contact, possibly by the wet canes picking up the spores [4].

The perithecia are gregarious, sometimes in extended patches, obscurely papillate, sub-globose, or depressed; sub-epidermal; black, measuring from 165 to 220 μ in diameter; the 8-spored asci are cylindrical and range from 60 to 70 μ by 10 to 12 μ; the ascospores are biseriate, rarely uniseriate, obovate to oblong, uniseptate, constricted at the middle, the upper cell being larger than the lower, hyaline, and measure from 14·0 to 20·2 μ by 5·5 to 9·1 μ (average, 16·5 by 5·6 μ); filiform paraphyses are extended above the asci [14] (Fig. 380).

Infection of sound young canes occur under conditions of high humidity. The fact that both stem and buds without previous injury may become infected following the application of ascospores [13, 14] strongly suggests that a wound is not always necessary for invasion. It is maintained, however, that the fungus can only enter the canes through insect punctures [12] or other slight injuries.

When the fungus, in either of its sporing forms or in the form of mycelium, is applied to the surface of a young cane, a brownish-coloured lesion appears in about two weeks after inoculation (Fig. 379 B). Beneath the lesion practically all the cells of the cortex become filled with fungus; the contents of the invaded cells as well as their walls turn brown and appear to be early killed by the mycelium. The fungus is confined almost entirely to the cortical parenchyma, and only when the stems are severely injured or split deep enough does infection penetrate into the vascular tissues of the stem. From the point of entry at the surface, the fungus travels up and down the cortex by direct penetration of one cell after another, depriving this tissue of its entire contents of starch. After killing a certain amount of cortical cells, the fungus proceeds first to the formation of pycnidia and later to perithecia, both being developed under the epidermis in these necrotic regions on the stem.

In the spring diseased buds frequently show perithecia and sometimes pycnidia on the surface of the outer, protective scales (Fig. 379 D), and in some cases, if the

latter are removed, these bodies may also be found on the inner set of bud scales. These infected buds are always dwarfed and they appear to have become infected, externally, from the top, where the tips of the outer scales emerge slightly. Buds infected in the summer will show next spring the presence of the mycelium in the outer scales in which the tissues have become browned and disintegrated; it appears probable that the infection of the buds under natural conditions takes place mainly from lodgement of spores at the tips of the buds, and not so much from basal infection from the node. The fungus never penetrates the axis of the bud, and only in exceptional cases when the buds are killed outright is the fungus present in their innermost leaves. As on the canes, the fungus is confined to the outer tissues of the buds; nevertheless it appears to make considerable development while they are dormant, for they contain much more mycelium in the spring than in the autumn. While the fungus does not often kill the canes outright, during the winter a high percentage of potential fruiting spurs is lost by the destruction of buds in the dormant season.

First signs of infection in the leaves are usually seen at the tip of a leaflet or at some point close to the midrib, as a small brown-coloured angular area, gradually growing longer, chiefly in a direction along the vein. Brown lesions may also occur independently at more than one place on the petiole and sometimes may girdle it entirely. Further infection of the bud in the leaf axil may take place from the diseased base of a leaf stalk. Within the diseased areas of lamina and petiole the fungus is again observed in the mesophyll, or in the cortical tissues respectively, but the vascular tissues are not usually occupied by mycelium.

Spur blight is greatly encouraged by humid conditions, and especially during wet periods in the spring. It is liable to appear on raspberry canes planted too close together and it is advisable not to have more than three or four young canes to each stock. To check the attacks, the old fruiting canes should be removed as soon as possible after picking is over, and destroyed by burning. As a protective measure the canes should be sprayed with Bordeaux mixture 3 : 5 : 40, with the addition of a soapy adhesive. The first application should be made in May when the canes are about 5 to 9 inches high, a second spraying being given after two weeks. In New York State, 2 per cent. ' fermate ' is also recommended, under similar conditions [19]. Care should be taken to remove all weeds around the canes, since they conserve moisture and thus help infection. When starting new plantations only stock certified free from disease should be planted [3, 14].

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Verticillium Wilt of Raspberry, *Verticillium dahliae* Kleb.

Blue stripe or Verticillium wilt disease of raspberry is caused by a fungus which attacks also a large number of herbaceous and woody plants, some closely related, others widely separated in classification, e.g. apricot, plum, almond, cherry, quince, loganberry, strawberry, black currant, gooseberry, tomato, lupin, mint, chrysanthemum, elm, maple, and others [1, 22, 28, 29, 30].

The disease was first recorded in England in 1923, and it has since been found on all varieties of the cultivated raspberry in this country. It varies in its intensity from year to year, and even in seasons favourable to the disease, is rarely economically serious [16]. Its occurrence in the United States was first recorded in 1904 at Washington, and it is reported to be very damaging to raspberry canes in the eastern parts of the United States, and also in Canada. In America, wilt disease is fairly constantly reported to attack black raspberries to a much greater extent than the red or purple varieties [2, 4, 9, 18].

Early signs of the disease usually appear on the new canes towards the end of June, on leaves situated towards the base of the plants (Fig. 381). Diseased leaves curl up at the margins, exposing the silvery under side, and the upper surface develops a characteristic, alternately yellow and green, or brown and green, striped effect, which extends diagonally from midrib to margin, the two colours being more or less separated by the prominent lateral veins. But the striped pattern is very often confined to one half of a leaflet, that is from midrib to margin, the other half being apparently normal ; in other cases the entire half of the compound leaf (a whole leaf consists of a terminal leaflet and two pairs of lateral leaflets), involving, therefore, half of the terminal leaflet and two entire laterals on the same side, may be affected with the coloured stripes, the corresponding laterals and half-terminal leaflet being green and healthy. Other leaves on the plant, from the base upwards, will show the same striped effect, and in the same order may gradually wither and die. As a result, there is considerable premature loss of foliage and the plant is often left with only a bunch of leaves at the top of the cane, and these may persist for a long time after all the others have perished.

A characteristic feature of blue stripe wilt is the presence of the discoloration, as above stated, in a definite half of the plant organ attacked, that is, it is confined to just half of a leaflet, or a half of a whole leaf, and the same feature may be seen on the cane itself, or even throughout an entire plant. Thus all the leaves along one entire side of the plant may be striped, while the other side will show all the leaves normal. Often the most striking effect is on the cane itself, occurring at the same time or even earlier than the symptoms on the leaves. Starting from ground-level usually, or sometimes several inches above ground, an intensely blue or purple stripe may be seen to extend to varying height, along one side of the

cane, forming a thin line which touches every node along that side where affected leaves are present, and running up also along varying lengths of the petioles. The bluish colour is not due to any pigment formed by the host or the fungus, but to the reflection of light from the waxy surface of the cane, along an area where the natural bloom is interrupted owing to the presence of the fungus in the woody tissues beneath. The blue discoloration may, however, sometimes extend, in parts, all around the stem, and when a good length of cane thus becomes encircled the leaves wither and the entire cane dies.

With the approach of winter and much loss of leaves through disease, the buds on affected parts of the canes become small and shrivelled, and entire canes in a stool may perish during the winter. Infected canes which succeed in producing leaves and fruiting spurs in the spring are much below the average size of healthy canes. Such affected canes produce small, dry, tasteless berries; in other cases the new foliage may turn yellow, and the fruiting branches may perish before the fruit is mature [13, 16].



FIG. 381.—Blue stripe wilt of raspberry (*Verticillium dahliae*). On a new cane of variety Lloyd George; note the interveinal discoloration of the leaves, and their collapse, from the base, upwards (photo by Harris, *J. Pomology*)

Blue stripe disease of raspberry is caused by *Verticillium dahliae* (Hyphomycetes) [17], and although considerable discussion has centred around the relations of this organism with the species *V. albo-atrum* [6, 24], it appears to be accepted that the presence of sclerotial bodies (microsclerotia) in *V. dahliae* and their absence from *V. albo-atrum* justifies their recognition as distinct species (see *Verticillium*-wilt of tomato) [1, 20, 27] (see p. 670).

It is remarkable that the fungus produces neither mycelium nor reproductive bodies on the surface of the host, and is practically confined to a vegetative mycelial existence in the vascular system and pith of the plant. The only means of natural propagation, apart from mycelium, are the minute black microsclerotia, which may be seen during the winter, in large numbers, sunken into the bark of the affected canes. These small resting bodies are thick-walled and

knot-like in structure, and are formed by the repeated budding of a single hypha until a mass of cells is developed; they are much more coherent and compact than the loosely woven groups of dark hyphae seen in *V. albo-atrum*.

The fungus collected from diseased wood, or pith, or from microsclerotia, is easily cultivated on artificial media, and a hyaline, septated mycelium soon becomes covered over with erect conidiophores. The latter are tall, very symmetrically branched, and unlike those of *V. albo-atrum* do not turn brown at the base [26]; each consists of a vertical hypha bearing at intervals several whorls of branching hyphae which terminate, either in a single conidium or in a variable number of short, closely set sterigmata each bearing a conidium. The sterigmata are so close together that their conidial masses look like sporangia or spore clusters at the ends of the branches. These spore aggregates, in preference to single conidia, tend to be developed when the air is dry, but when moist conditions return, the spore masses appear like spherical drops of water, due to the absorption of moisture by the mucilage with which the spores are coated. The verticillate branches of a conidiophore may be as many as 7, commonly 3 or 5 in a whorl, and each of these again may bear secondary whorls or virtels; the conidia measure from 4 to 11 by 1·7 to 4·2 μ [8]. Conidia have not been seen to develop on the canes in the field, and as far as is known have not been detected to play any part in spreading the disease. Cultures obtained from microsclerotia are identical with those obtained from diseased wood or pith, and after the development of conidia, the culture passes over to the formation of chlamydospores, and finally to microsclerotia again [21, 26].

The fungus is capable of over-wintering in the soil on organic matter, and if decayed canes are allowed to remain in the ground, microsclerotia are developed in the bark and liberated into the soil, where under favourable conditions they germinate to produce mycelium and so bring about fresh infections. The fungus is believed to thrive in the soil as a saprophyte on the remains of stems and roots; it is also known to persist from one season to the next in soil from which wilted raspberry plants had been entirely removed, and it is recorded, too, that the mycelium can travel for short distances in the soil [2, 3].

The host plant contracts the disease through the roots, and the fungus in the form of resting mycelium, or from mycelium produced by microsclerotia in the soil, is believed to gain entrance by penetrating the finer rootlets, or root hairs [5] or at the points where lateral roots break through [19]. Opinions differ, however, as to the capability of the fungus to attack sound healthy roots except through wounds [25]. The fungus soon invades the cortical tissues and penetrates into the innermost tissues to occupy the xylem of the roots, crown, and stem. It is not known exactly to what extent the fungus permeates the tissues of the canes, spurs, and leaves, but as already stated, all the symptoms of the wilt are traceable to a clogging of the water-conducting tissues of the xylem, resulting in a wilting of the foliage and in the development of the characteristic striped effects in stems and leaves. In general, the presence of the fungus in the water-conducting elements of the stele, causes a brown discoloration in the xylem, but this feature is not always evident, and it may actually appear before the occupation of the xylem by the fungus. The protoxylem elements are easily entered through their thin cellulose walls, and vessels and tracheids through the pits in their walls, and in this way infection may travel up the stem, and apparently the fungus is sometimes to be found in the vascular strands of leaf petioles, and in the network of veins in the

leaves. The fact that the fungus seems to confine itself to the woody conducting system, growing principally in a longitudinal direction, no doubt explains the characteristic phenomenon of the one-sided development of the symptoms on stems and leaves [21]. As the fungus ascends in the stem, its growing tips are colourless, but the hyphae gradually darken behind as they get older, and the browning effect seen in the wood occupied by mycelium is partly due to the colour of the older mycelium, and partly to a staining effect produced on the lignified walls by the fungal secretions [25]. The histological effects of this fungus on the raspberry have not been investigated to the same extent as for other woody hosts attacked by it [11], and in other hosts the brown discoloration in the wood is attributed largely to its occupation by wound gum impregnated with tannin.

Wilting of the foliage, characteristic of this disease and of many other wilt diseases [10], may either be due directly to a stoppage of the vascular channels by fungus mycelium, or merely to the infiltration into the conducting stream of a toxic secretion from the fungus in advance, acting detrimentally by reducing the efficiency of the cells controlling transpiration. The degree of wilting does not appear to depend on the amount of mycelium in the vessels, for in comparatively long stretches of affected stem there may often be but a few strands of mycelium, with little or no sign of the complete occlusion of vessels by plugs of mycelium. In relation to the appearance of the gummy substance above mentioned, this phenomenon is frequently accompanied by tyloses in some of the vessels, so that the formation of gum and tyloses in the wood must also be taken into consideration as factors retarding the flow of water into the leaves [11, 12].

Warm weather, rather than cool periods, appears to be favourable to the incidence of wilt disease. Unfortunately a great deal of information relative to the influence of temperature in connection with Verticillium wilts is vitiated by confusion over the taxonomy of the species concerned, or to working with different strains of the organisms. *V. dahliae* is recorded to tolerate higher temperatures (up to 30° C. or more) than *V. albo-atrum* [20]. Little appears to be known about the effects of varying degrees of moisture in the soil upon infection in the raspberry; in general, on the various hosts, it is stated that extremes of both high and low soil moistures tend to aggravate the disease [20].

Owing to the survival of the fungus in the soil, it is recommended that a method of rotation should be adopted which allows for at least four or five years to elapse before raspberries are restored to the same ground [5]; but the choice of rotation is not easy as the organism attacks such a wide range of cultivated plants, herbs, shrubs, and trees. Badly affected stools should be lifted and burned, and even young canes, or sucker plants, should not be taken from infected areas for planting in clean soil since the fungus can be carried in soil clinging to the roots. Sometimes the fungus may be present in the stem with no signs of the disease at all, and new plants should always be obtained from certified healthy stock [3].

There is apparently no type of raspberry immune from blue stripe disease, though in America the opinion is fairly general that red raspberries are more resistant than black varieties [25]. In that country the widely grown Revere is highly susceptible under all conditions, Alton Improved, slightly less so, while Columbian Purple, Victory, and Cuthbert are resistant to a fair degree; the variety Syracuse

is highly resistant but not immune. In Britain there are no red varieties wholly resistant, although on some kinds the disease appears to have very little effect [13]. The varieties more seriously affected in this country include Bath's Perfection, Red Antwerp B, and Prior's Prolific [30], but Black Antwerp A is more resistant [13, 16].

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Raspberry Mosaic

Of recent years the raspberry-growing industry has suffered considerably owing to the degeneration of well-known varieties, due to virus infection. Though virus diseases of raspberries in America, and the genetical constitution of the varieties susceptible to them there, *Rubus strigosus* and *R. occidentalis* (red and black raspberry and 'purple' hybrids), are not identical with those attacking varieties of European red raspberry, *R. idaeus*, in Britain, these diseases are a limiting factor in the cultivation of this fruit both in Canada and the United States [2, 3, 4-6, 8, 16, 18, 19, 20, 21], and in certain areas in Britain they continue to be the most troublesome and widespread of the maladies which affect this crop [9, 14].

It is not possible to present a general picture of raspberry mosaic, but there are two more or less well-differentiated types which, in Britain, have been referred to as *Mosaic 1* and *Mosaic 2*. But the symptoms presented by either of these types show considerable variation, according to individual varieties, and to other factors discussed below (Fig. 382). Recent observations in England and Scotland indicate that the virus situation in raspberries is of greater complexity than

previously experienced. No fewer than five distinct symptom types have been identified, some of which appear to originate from multiple viruses [14a].



FIG. 382.—Raspberry mosaic mixture of *1* and *2* on 'Lloyd George'; healthy leaflet, top corner (photo by Foister & Noble)

Mosaic 1 (*Mosaic 1* Harris 1940)

This is a better-defined type than *Mosaic 2*, and may be considered as a single disease of a uniformly mild type of infection. It is somewhat restricted in its varietal distribution, is slow in its deteriorating action, and its symptoms are suspended or temporarily masked by hot, dry weather conditions. It causes a mottling of the leaves on all the canes of an infected stool. The chlorotic spots are somewhat ill-defined, tend to aggregate towards the leaf margins and between the main veins, so that they often give the leaf a slightly striped appearance. The spots are slightly sunken and accompanied by a down-curling of the lamina along the midrib, the leaflets thus becoming boat-shaped, the lower surface being concave; sometimes there is a downward fold in the midrib or keel itself [1]. The pale spots vary in colour from pale green to greenish yellow, and in size from mere flecks to areas about 2 mm. in diameter, which are not sharply differentiated from the normal green of the leaf. A transverse section of an affected leaf passing through the chlorotic area shows the lamina to be thinner than in the normal parts, due to a shortening of the palisade cells. *Mosaic 1* has been observed on the varieties Lloyd George, Baumforth's Seedling B, St. Walfried, and Norfolk Giant.

Mosaic 2 (*Mild Mosaic 2* Harris 1940: *Severe Mosaic 2* Harris 1940)

This form of the disease is a 'complex' and appears to include two or more diseases differing in intensity from the foregoing mild mosaic. Yet this complex type expresses itself in what may again be separated into '*mild*' and '*severe*' phases. A plant affected with the mild phase of *Mosaic 2* does not apparently deteriorate and the symptoms may be so slight as to be hardly detectable. On the varieties Mitchell's Seedling, Bath's Perfection, and others, spots of irregular size and shape are evenly distributed over the entire leaf surface; the individual spots are not appreciably sunken and the leaf is not curled or distorted, and except for the mottling, is almost normal. *Mosaic 2* in its severe phase may perhaps arise as a result of further infection with other viruses, of plants already affected with the mild phase. With the added infection, deterioration is very rapid and the plants soon cease to crop and die out. The leaf spots are bright yellow to greenish

yellow, and are more strongly defined than those of *Mosaic 1*; they are either deeply sunken or raised above the general surface of the leaf, and again the lamina is thinner in the affected areas. The spots, moreover, appear translucent, are generally distributed over the leaf, and the individual spots may be seen often to cut across a vein or midrib. The leaves exhibit a curling of the lamina and finally become twisted and crumpled. Symptoms of *Mosaic 2* are not masked by high temperatures as are those of *Mosaic 1*. These reactions may be exemplified on the varieties Baumforth's Seedling B and Norfolk Giant. But raspberry varieties differ greatly in their reaction towards *Mosaic 2*. Thus, the variety Lloyd George behaves as a symptomless carrier of *Mosaic 2* but, when infected with *Mosaics 1* and *2*, symptoms appear and the vigour of the stools appears to be reduced. The variety Preussen was found to react to infection with *Mosaic 2* in the same way as Lloyd George, though it sometimes showed leaf symptoms [13].

In the American mosaic diseases, spread is said to take place through insect agency, *Amphorophora rubi* being the most important vector in Washington [15]. In Britain, up to 1948, all attempts to transmit the disease by mechanical means have proved negative [7], for the probable reason that any viruses present in expressed sap are immediately precipitated by released tannins [1a]. They are, however, graft-transmissible, and the vector *A. rubi*, as well as *Aphis idaei* [3a], have recently been found to act as vectors of raspberry viruses in Scotland. Another virus disease, 'leaf curl', affecting Baumforth's Seedling B and Norfolk Giant (and 'carried' by the variety Lloyd George) is, so far, confined to Scotland. It shows a yellow blotching of the foliage of new canes, the leaves later becoming tightly curled and brittle; fruiting laterals next season die early and black lesions may show on stems and petioles [1].

It has already been noted that high summer temperatures affect the symptoms of some types of English raspberry mosaic. That susceptibility to mosaic may also be influenced by locality rather than infectibility was shown by observations of mosaic-diseased plants of the raspberry in the Hudson Valley. In this area, in the immediate neighbourhood of Amherst, the cultivation of the variety Cuthbert has largely been abandoned owing to the rapid spread of mosaic; but some 15 miles in a north-easterly direction from this area, up in a range of hills where the native woodland has been cleared and planted up with fruit, this variety is almost exclusively grown. Whilst the plants do not actually escape the disease they show no marked general deterioration in vigour, and so far continue to crop well. Such instances of induced low susceptibility is closely paralleled in Britain, with the relative resistance of the variety Mitchell's Seedling in Scotland, and the very high susceptibility of this same variety in East Malling, Kent. This suggests that the degree of susceptibility of a variety is not absolute but is largely influenced by the conditions under which it is grown [11].

As the means of transmission of raspberry mosaic in Britain have not been fully elucidated, methods for its control must rest largely on the maintenance of healthy stocks. To this end nurseries should be established in comparative isolation from all other raspberry crops, at least 50 to 100 yards from infected sites [1, 16]. Since there are varieties of raspberries endowed with a low symptom-expression, it is clear that control by roguing cannot always prove effective, and some method is

desirable whereby rapid testing of named seedling varieties can be carried out by the use of indicator plants. This procedure is now in vogue at the Research Station, East Malling [13]. Where roguing can be resorted to, the plantation should be examined at least twice a year, and any infected stools, together with those on either side, should be dug up and burnt. All nursery canes should be cut down at planting to within 6 inches of the ground, and at the end of the first growing season the canes should be cut close down again ; if there has been vigorous growth, the canes should be dug out, leaving only sufficient root-stock for the following year. This practice should be observed every year, leaving no fruiting canes in the nursery [1].

Breeding experiments in Holland [19] and America [20] give promise of the production of varieties of raspberry resistant to mosaic in those areas.

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American Gooseberry Mildew, *Sphaerotheca mors-uvae* (Schw.) Berk.

American mildew is considered to be the most destructive of all diseases of the gooseberry ; white, red, and black currants are also susceptible, in lesser degree, to the same disease. It is caused by *Sphaerotheca mors-uvae*, a member of the *Erysiphaceae*, the powdery mildews.

As the name implies, the mildew is of American origin, and is believed to have been carried to Europe first to south-west Russia in 1890, and again to Ireland in 1900 [3, 10, 11] ; in Europe it soon assumed epidemic proportions, and the first outbreak in England in 1906 seems to have travelled from the Continent [11, 13]. The disease has been extensively studied in Britain [12, 18]. It is of interest to note that this disease, in 1907, was included in the Destructive Insects and Pests Act of 1877, which imposes various restrictions on the importation of plants as a means for controlling plant pests in Great Britain.

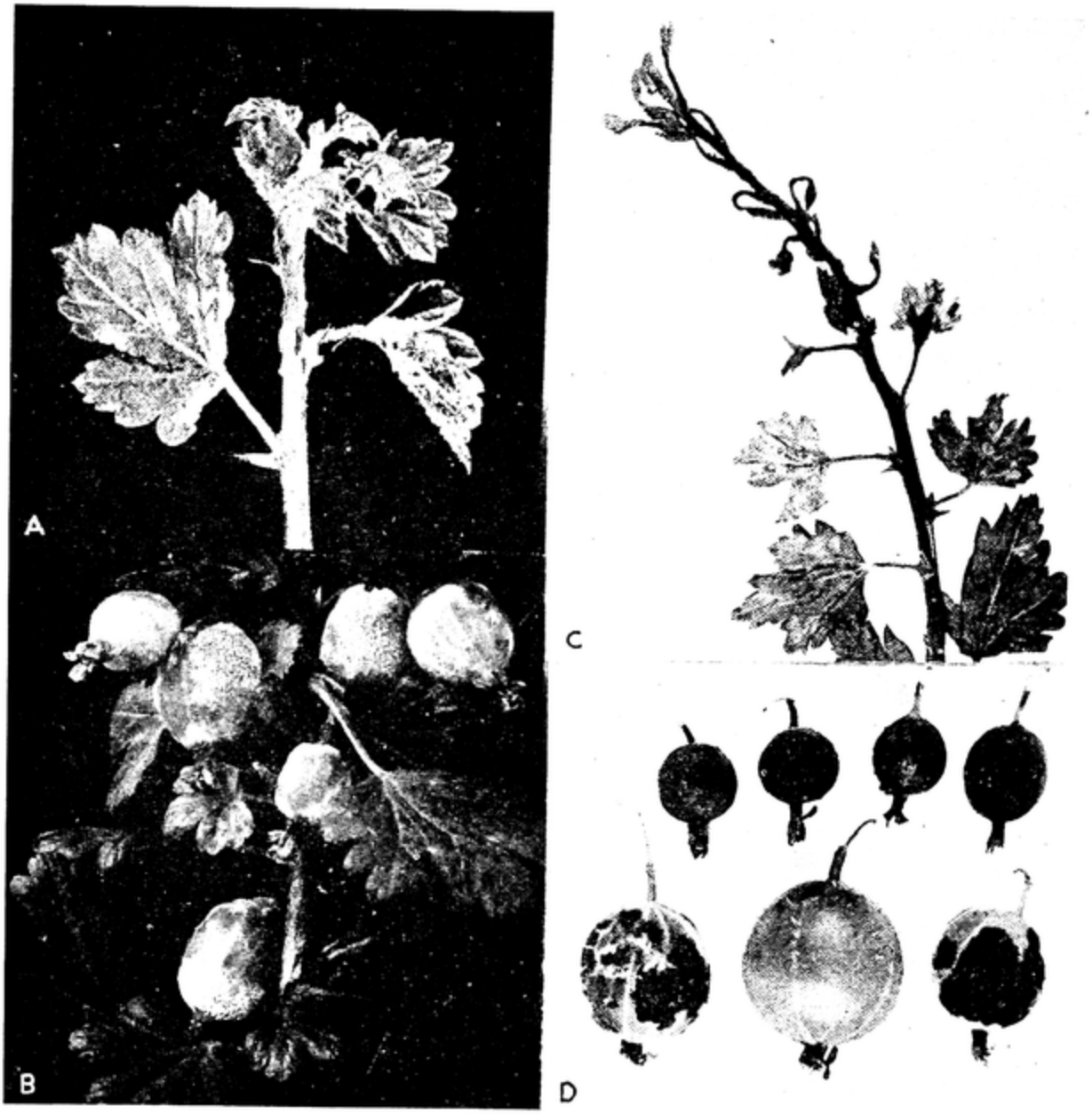


FIG. 383.—American gooseberry mildew (*Sphaerotheca mors-uvae*). *A*, the white, conidial fructifications covering leaves, petioles, and upper part of stem. *B*, the white, conidial stage on the berries. *C*, end of shoot severely attacked and completely covered with brown cleistocarps. D, the berries, all except the large one, covered with masses of cleistocarps (photos *A*, *C*, *D*, by Salmon, Wye Reports; *B*, by Wormald, *Diseases of Fruits and Hops*, Lockwood)

In Britain the mildew is first seen in England in April and towards the end of May or early June in Scotland. The tips of the young shoots and the leaves on both sides become more or less covered with white mycelium which soon spreads over the growing shoots, causing a serious check in growth, and continues to spread over leaves and berries at all stages of their growth. The white effect is due to the prolific formation of conidia which continue to be developed throughout the growing season on twigs, leaves, and fruit (Fig. 383, A, B). The white superficial mycelium sends haustoria into the epidermis, and vertical conidiophores arise in great number, producing long chains of hyaline conidia which

are easily dispersed by wind, and fresh infections often occur on an epidemic scale. Later in the summer the white effect gradually changes to brown, the discoloration being most marked on the twigs and berries, less on the leaves. The change is due to a darkening of the mycelium which now forms a thin, closely interwoven weft of hyphae which remains on the twigs and berries after the leaves are lost (Fig. 383 C, D). The brown weft of mycelium is easily scraped off and embedded in it are very numerous small black cleistocarps which serve to tide the fungus through the winter. The first appearance of the cleistocarps varies considerably according to the time of infection, and sometimes they mature sufficiently early to discharge their spores during the same season, but usually they are not formed until the autumn, remaining dormant on the leafless bushes until the spring. They can also tide the winter on cast-off twigs and on remains of dropped berries.

The cleistocarps are sub-globose, 76 to 100 μ in diameter; the single elliptic-oblong to sub-globose ascus, 70 to 92 by 50 to 62 μ, practically fills the interior; the 8-ellipsoid ascospores measure from 20 to 25 by 12 to 15 μ [12]; the appendages are simple, contorted, pale-brown, but often rudimentary or absent (Fig. 384). The ripe cleistocarp when moistened swells and splits along the top to allow the apex of the ascus to emerge and dehisce and the spores are discharged into the air simultaneously [16, 17].

Both conidia and ascospores are capable of causing infection. The latter, ejected from cleistocarps which have over-wintered mostly on cast-off twigs on the ground, first infect the lowermost leaves and twigs and widespread secondary infections occur from the dispersion of the conidia produced on these parts. Though the mildew is present only on the surface of the host, the presence of the fungus on the leaves seriously affects their functions, retarding photosynthesis considerably, and repeated attacks from season to season have a weakening effect on the entire tree which yields less and less fruit every year.

Different varieties of gooseberries vary in their degree of susceptibility to mildew and a variety which may be resistant in one locality may not always be resistant in other places. Thus the variety Multon which had shown marked resistance in the west of England gave a high percentage of

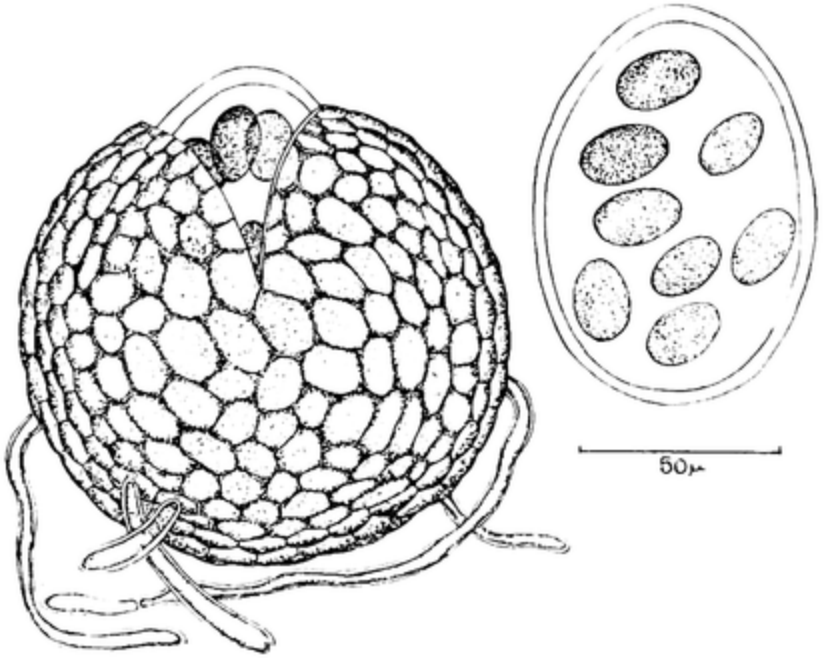


FIG. 384.—*Sphaerotheca mors-uvae*. A cleistocarp slightly crushed to expose its single ascus; note the simple appendages; ascus with eight spores

diseased berries when grown in the north of Ireland, and the complete resistance previously shown by three American varieties also broke down there [6]. The most susceptible kinds are Keepsake, May Duke, Warrington, Lancashire Lad, Rushwick Seedling, Careless, and Whinham's Industry; the more resistant are Lancer, Crown Bob, and Whitesmith.

To check American mildew, bushes should be planted in open ground, well spaced apart and correctly pruned to encourage open growth [2]. Pruning should not be deferred till late in the year for by that time the cleistocarps will have formed and will survive over the winter. All prunings from affected bushes should be collected and burnt. Heavy nitrogenous manures which encourage sappy growth in the twigs should be avoided and a balanced treatment with basic slag, potash, and phosphates should be given. A spraying programme is essential and the choice of fungicide is important. There are several varieties of gooseberries which are more or less sensitive to sulphur applications, and for these, sulphur dust and lime sulphur are not advised. The principal fungicides and methods of application are :

(*a*) Lime sulphur : 1 per cent., applied before the flowers open, again after the fruit has set, and a third treatment given about 3 weeks later. This treatment is not advised for sulphur sensitive or amber-coloured kinds [5], such as Early Sulphur, Golden Drop, Yellow Rough, and Leveller.

(*b*) Ammonium polysulphide : $\frac{1}{2}$ per cent. solution with the addition of soft soap as an adhesive (4 or 5 lb. per 100 gallons) [9]; applied as above, but not for amber bushes.

(*c*) Washing soda, plus soap : Safe for all varieties ; made up with 1 to 2 lb. of soda with $\frac{1}{2}$ lb. of soft soap to 10 gallons of water. This treatment is more effective if the bushes have already been sprayed in February with a 2 per cent. solution of caustic soda [5]. Applied soon after the flowers have set and again 3 weeks later.

(*d*) Sulphur dust : Lightly sprinkled or blown on to the bushes with a sulphurator, covering both sides of the leaves, starting early and repeating fortnightly if mildew is persistent ; applied at the rate of 20 lb. per 100 average-sized bushes [6].

(*e*) Burgundy mixture : 1·5 per cent., applied as a pre-blossom spray ; not generally acceptable as it is inclined to cause scorching, but it stands the action of the weather better than lime sulphur [1, 4, 6, 7, 8, 9].

European Gooseberry Mildew, *Microsphaera grossulariae* (Wallr.) Lév.

This second type of gooseberry mildew which also occurs on the three kinds of currants, is caused by *Microsphaera grossulariae*, another member of the *Erysiphaceae*. It often attacks bushes growing in the shade but is not nearly as serious a trouble as the American mildew. The fungus is thinly developed on the host, and unlike the weft of mycelium growing on both sides of the leaves affected with American mildew, this forms only a thin evanescent veil over the upper surface of the leaves, little, if any, occurring on the under side, and rarely any at all on the fruit. European mildew is, therefore, confined to the foliage and tends to bring about premature defoliation (Fig. 385).

The conidia serve to bring about widespread secondary infections during the season. The cleistocarps afford a ready means of distinguishing this disease from American mildew,

the most striking difference being the way in which the numerous appendages are branched at their tips in characteristic mop-like tufts, unlike those of *S. mors-uvae* which are simple filaments. The cleistocarps, at first yellow, later black, arise on the leaves, embedded in the thin mycelium ; they are globose-depressed, from 65 to 130 μ in diameter ; appendages, 5 to 22 in number, are branched at the tips four to five times in a dichotomous fashion to form short processes ; asci, 4 to 10 in number, broadly ovate or oblong, measure from 46 to 62 by 28 to 38 μ ; ascospores, 4 to 6 in each ascus, measure from 20 to 28 by 12 to 16 μ [12]. The cleistocarps fall to the ground or remain on the fallen leaves and lie dormant until the summer when the ascospores are ejected to start primary infections on the new growth.



FIG. 385.—European gooseberry mildew (*Microsphaera grossulariae*) (photo by Foister & Noble)

This mildew does little harm to gooseberry bushes growing in open situations and properly pruned to admit light and air. If more severe than usual the same measures should be applied as for the American mildew, preference being given to the washing-soda and soap treatment.

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Cluster Cup Rust of Gooseberry, *Puccinia pringsheimiana* Kleb.

Orange-red spots familiar in May and June on the leaves and petioles, and later on the twigs and fruit of gooseberry bushes, are caused by *Puccinia pringsheimiana*, one of the rust fungi (Uredinales). Actually they are only one phase, that of the

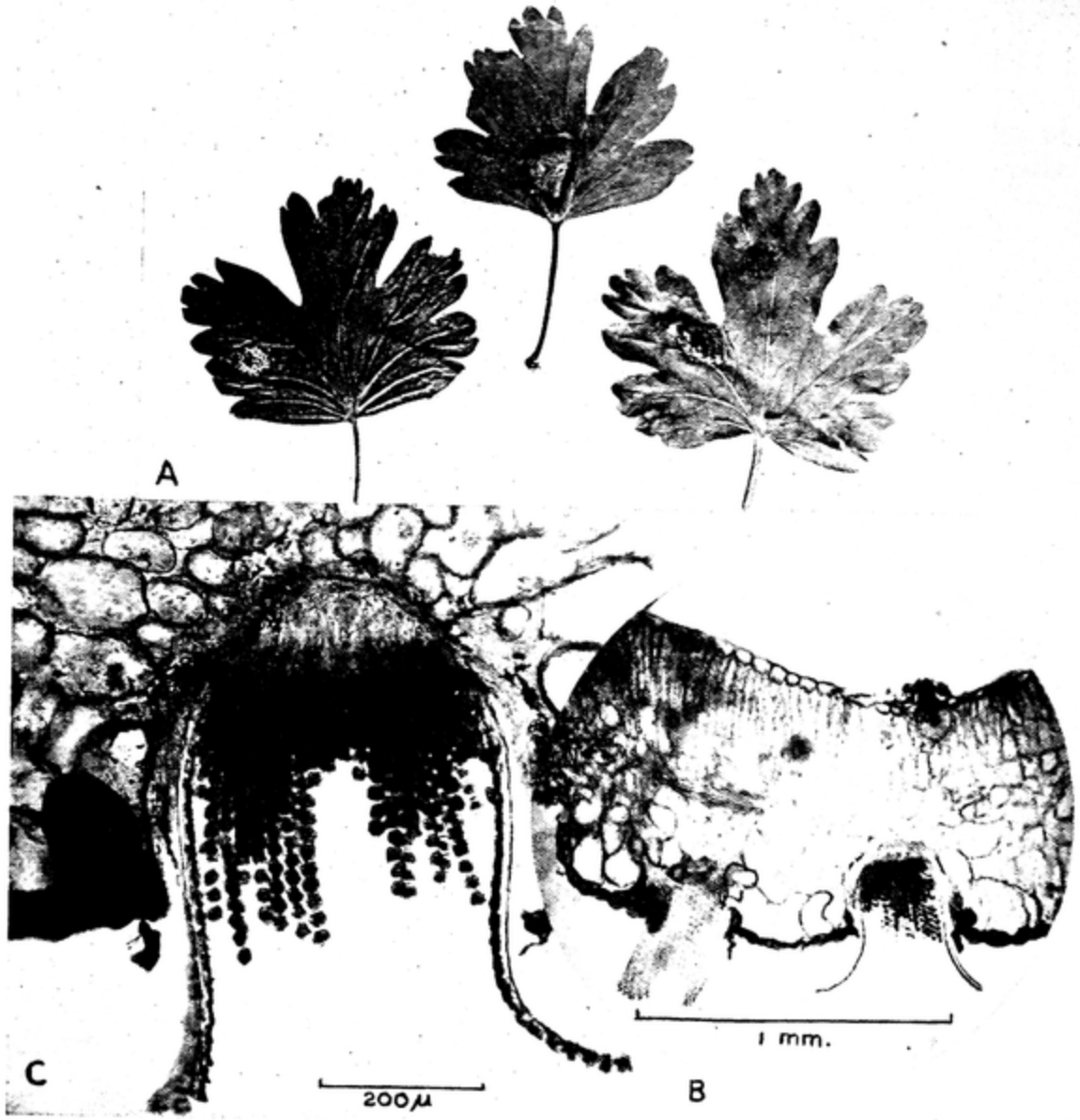


FIG. 386.—Rust of gooseberry (*Puccinia pringsheimiana*). *A*, the accidial cluster cups on gooseberry leaves (photo by Foister & Noble). *B*, transverse section of leaf of gooseberry showing, at upper surface, a spermagonium; at lower surface, on left, a portion of peridium; on right, an aecidium in vertical section, as shown in *C*, enlarged

aecidial stage, in the life-cycle of this heteroecious rust, the other stages producing uredospores and teleutospores on various species of sedges (*Carex*).

The spots are somewhat raised, smooth and blister-like, and often cause distortion of growth on leaves, petioles, and berries, and may sometimes be decurrent from an affected leaf, covering portions of the stem with a red crust often an inch long (Fig. 386 A). The disease is usually of little economic importance, a mild attack consisting merely of a few spotted leaves and berries here and there on the bush, but in severe cases the fruit may be so spotted and disfigured as to render it quite unsaleable.

These spots on various parts of the host become covered in early summer with the so-called cluster cups which consist of a close aggregation of aecidia.

On the leaves the aecidia open towards the lower surface, and after dehiscing, are filled with dry, yellow powdery masses of aecidiospores. On the berries the red spots are somewhat thicker than on the leaves, forming raised crusts or warts studded with aecidial cups. A section of the leaf (Fig. 386 B, C), passing through a spot, shows the presence of numerous spermagonia situated below the upper or lower epidermis, which discharge spermatia and copious 'nectar' to the surface. The numerous aecidia, embedded in the spongy mesophyll, break through the lower epidermis, each producing a long, white cylindrical peridium with a strongly recurved, torn margin. The aecidiospores are spherical, finely echinulate, yellow, 15 to 21 by 14 to 18 μ. They serve only for infection of the alternate, sedge host, in summer and early autumn [3].

Following upon infection of the alternate host by aecidiospores, yellow pustules of uredosori about $\frac{1}{2}$ cm. long arise on the narrow, grass-like leaves of the sedge; the uredospores are spherical, pale brown, echinulate, 18 to 22 by 17 to 21 μ; they serve only to spread infection amongst sedges during the summer, playing no direct part in carrying the rust to the gooseberry host. Later, during the autumn, on stems as well as leaves, infected sedges develop the more important sori of teleutospores because it is from these that infections will start on gooseberry bushes in the spring. It is important to note that while the aecidiospores are instrumental in restoring the rust to the sedge host on which the uredosori are the first to develop, the extent to which uredospores cause widespread infections among sedges indirectly affects the degree of infection on the gooseberry, because from these recurrent uredospore infections more and more teleutosori arise, for it is the same type of mycelium (binucleate) that gives rise to both uredo- and teleutospores. Teleutosori are brown or black, linear or punctiform, up to 1 mm. long, on leaves and stems. The teleutospores are oblong, club-shaped, 40 to 58 by 15 to 22 μ [3]. These spores serve for the survival of the fungus on the sedges and persist over the winter on the remains of these plants. (There is definitely no evidence that the rust remains dormant within or on the gooseberry host during the winter [4, 5].) In the spring the teleutospores germinate in the usual way, producing their sporidia just at the time when gooseberry bushes normally break into leaf. The sporidia reach their highest peak of dispersal several days before the gooseberries commence to flower, and are practically spent some four or five weeks later [5]. As already stated, the sporidia attack the gooseberry in late spring, and, as soon as the red spots are evident, spermagonia begin to be formed and are soon followed by aecidia.

The life-cycle of the fungus is dependent on the existence of the disease on sedges, and if these could be completely eradicated, cluster-cup rust would completely disappear from gooseberry bushes. This, however, is hardly a feasible proposition, as the infective sporidia can travel by air for long distances. Still, an appreciable reduction in the amount of rust on the gooseberry can be obtained by the cutting-down and removal of sedges from adjoining meadows or marshes. This was experimentally shown in a meadow in Northern Ireland, where no fewer than five different species of sedges were known to grow. A number of small gooseberry bushes in pots were placed in the meadow, and during the first year they remained perfectly clean because the meadow had been closely mown the previous year (1933), but in 1934, when left uncut, the sedge plants developed the rust, and in 1935 practically every leaf and berry on the gooseberry bushes were affected with

rust [5]. The importance of sedges as the over-wintering host for the teleutospores was further shown by another experience when an epidemic of the rust occurred suddenly in an upland Highland village where it was previously unknown, but in which in one season all the gooseberry bushes in the cottage gardens were found to be heavily rusted. No sedges could be found growing in the vicinity, but the disease was finally traced to a byre recently thatched with infected sedges which had been collected from an infected area [1]. The rust has also been known to appear on bushes following the use of sedge mowings for mulching.

No variety of gooseberry is known to be immune from this rust [4, 5]; but considerable variation in the degree of susceptibility was found in no less than 40 varieties in Esthonia [4].

Gooseberry bushes should not be grown in low damp situations which are favourable to the growth of sedges. If possible the latter should be grubbed up and destroyed; they should not be used for mulching. Sedge lands adjoining or close to gooseberry plantations should be systematically mown down every summer and the cuttings removed. Ordinarily the rust is not sufficiently troublesome to demand treatment, but if it becomes recurrently severe a spraying programme should be carried out, using 2 per cent. Bordeaux mixture as soon as the gooseberry buds are well opened but before the leaves are fully expanded, and again, 2 to 3 weeks previous to flowering, using a weaker 1 or 0·5 per cent. strength of the mixture [2, 5]. Three applications of 2 per cent. lime sulphur, first, at green tip stage, again 10 days later, and finally just before blooming are also recommended [6].

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Die-back of Gooseberry, *Botrytis cinerea* Fr.

A die-back of twigs and branches is common everywhere on gooseberries and is troublesome on all types of soils, especially where drainage is poor [2, 4]. It usually occurs on individual or on small groups of bushes here and there in the plantation.

The trees may be attacked at any stage of growth, even when in full flower or bearing fruit. The disease may affect the plants in many ways (Fig. 387), the most serious being the death of the tree branch by branch, and this phase may either begin at the periphery of a bush, causing a die-back of a few young shoots, or it may start low down on the main stem, the infection working its way up into the branches until the entire bush is killed [3]. An attack on the leaves and berries is another distinct phase of the same disease. The margins of the leaves turn yellow, then ashen grey or white, and if the discoloration extends throughout the entire lamina the leaves fall off prematurely, otherwise they remain for the normal period. Some twigs may thus be partially or completely denuded of leaves. Affected

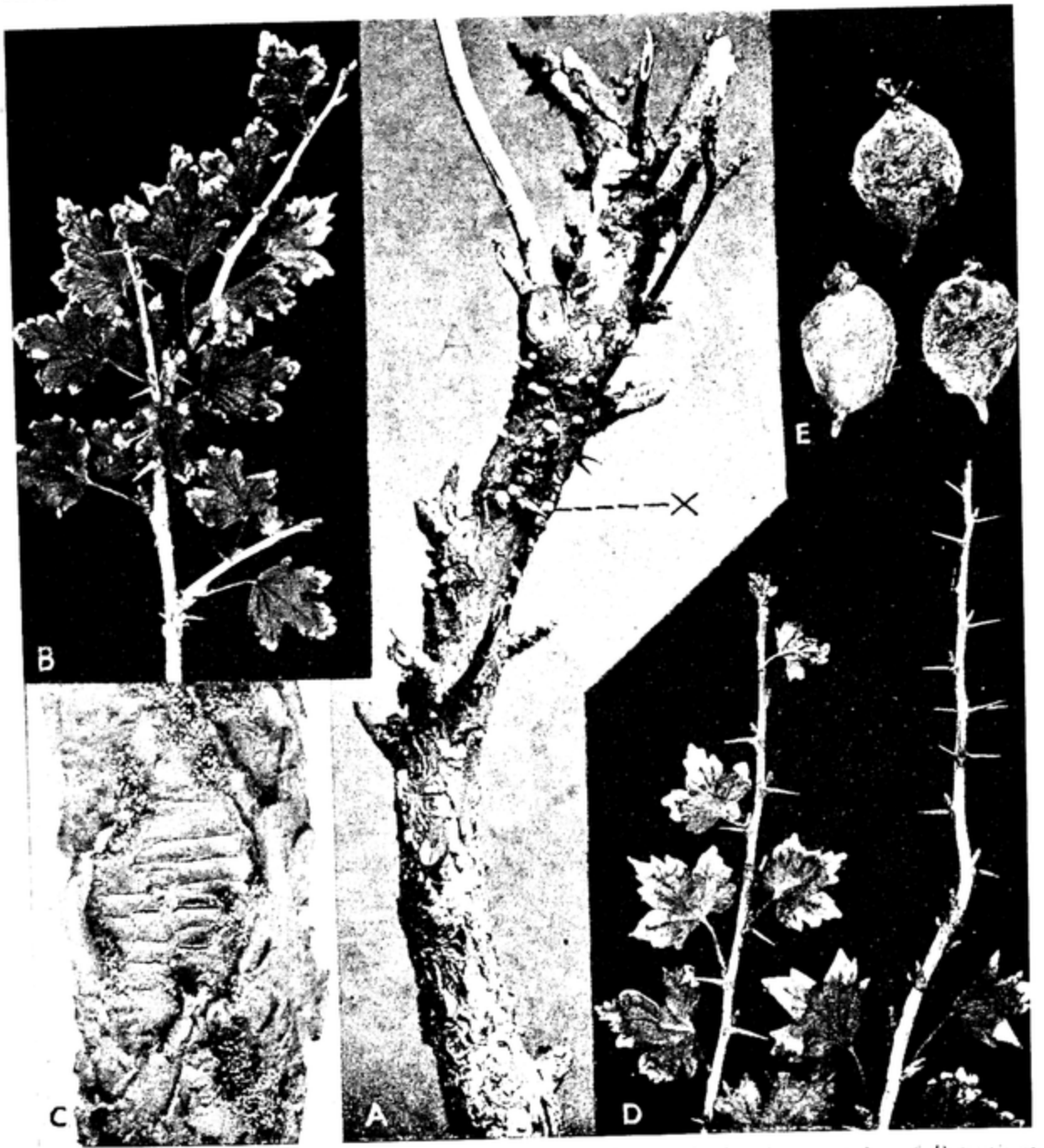


FIG. 387. Die-back of gooseberry (*Botrytis cinerea*). *A*, branch showing pustules of *Botrytis* at *x* and elsewhere. *B*, shoot with attacked leaves white at the edges. *C*, portion of stem just above ground-level showing pustules in cracks in the bark. *D*, the die-back of the stems. *E*, berries covered with the conidial pustules (photos by Salmon, Wye Reports)

berries become spotted brown, at first in small patches which later extend mostly along one side before they finally develop a soft rot whilst still attached to the tree or after dropping to the ground.

On all these parts of affected bushes mycelium and fructifications of the common grey mould fungus *Botrytis cinerea* (see p. 659) causing this disease are developed in great profusion. A characteristic feature of the die-back phase is the splitting and peeling of the bark, and when this is extensive considerable exposure of the underlying tissues to the drying action of wind no doubt hastens the death of the

tree. During periods of warm, moist weather in the spring, cracks and fissures in the bark are seen to be filled with the characteristic smoky-grey mycelium, and upright, branched conidiophores bearing heads of conidia (Fig. 387 C) are developed in great numbers. The conidia are dispersed by wind and probably by insects which, by infesting the berries, no doubt assist in their infection [4]. When conidial production on stem and branches is waning, the mycelial cushions in the fissures proceed to the formation of sclerotia ; these bodies are irregularly shaped, hard and black, and may remain embedded in the bark over winter, either on the tree or on fallen branches on the ground. In the spring, the sclerotia give rise to mycelium which again forms abundant conidiophores and conidia for renewal of wind-borne infections when the trees burst into leaf again.

First signs of die-back at the periphery of the bush may occur almost as soon as the trees are in leaf, when the leaves wilt, turn brown, and wither. In the case of young bushes especially, a considerable proportion of young shoots may be attacked, weakened, or killed, and on many of those killed back, the fungus can be found wintering in the dead buds and producing fresh crops of conidia in the following spring. Such infected dead shoots allowed to remain on the trees or left as prunings on the ground around the trees are a prolific source of infection to the bushes around [4].

When the main stem is attacked, the trouble appears to start at, or a little above, ground-level. The fungus in all probability enters here through open wounds in the bark and, according to the severity of the infection, partial or complete girdling of the stem takes place. The fungus penetrates into the tissues of the cortex and phloem and may travel upwards under the bark to the bases of the branches. Hence the presence of a few dead branches in a bush, or sometimes the death of half a bush is a characteristic feature of this type of die-back. Entire girdling of the main stem may, however, take several years, and meanwhile the bush is being killed branch by branch, but once girdling is complete the whole bush dies. The resting mycelium under the bark enables the fungus to over-winter in this position from one season to the next, and when it has accumulated in sufficient quantities the fungus breaks through the bark in preparation for sporulation. It is recorded that as early as February, according to the mildness of the season, tufts of conidiophores may appear in the fissures, and it is not improbable that, protected more or less within cracks in the bark, conidia may also sometimes survive through a mild winter [4].

As already mentioned, the same fungus attacks the leaves and berries independently of the stem and branches. Whilst it is only in some seasons and localities that the foliage suffers to any great extent, loss of berries through the action of grey mould is usually heavy, even when the leaves are to all appearances sound and uninjured. The characteristic fructifications develop on leaves and berries (Fig. 387 E) usually in June or July, and these, especially the fallen rotted berries, serve for much dissemination of disease, probably through insect agency, during the season [4].

It is probable that gooseberry bushes (and other plants susceptible to grey mould) are attacked by this fungus only through wounds or when they are weakened through some nutritional error or deficiency. It is well known that over-feeding

with nitrogenous fertilisers encourages sappy growth in young twigs, a condition which is probably highly conducive to the die-back of young shoots, mentioned above. It is also known for leaves of gooseberry bushes to develop a yellowness around the margin quite independently of any fungal or other infection, and this is believed to be due to mineral deficiency, probably of potash and lime, in the ground.

Bushes showing die-back of shoots should be pruned well below the affected part, and such bushes may thereafter remain quite free from disease provided infection has not become established elsewhere lower down. Bushes badly wilted or showing numerous bare branches should be sacrificed, these symptoms indicating clearly the presence of deep-seated infection in the stem. All prunings and dead branches should be collected and burned [1].

If *Botrytis* pustules are still evident on the trimmed bushes during the winter, spraying with a solution of copper sulphate (4 lb. in 100 gallons) is advised, just before the buds open. Bordeaux mixture (4 : 4 : 50) may also be applied after fruiting, but certain varieties of gooseberry trees appear to be injured by this treatment [2].

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Leaf Spot of Currant, *Pseudopeziza ribis* Kleb.

Leaf spot, or anthracnose of currant is widely distributed, occurring in the United States and Canada, in Europe from Norway to Italy, Asia, Australia, and New Zealand [2, 3, 4, 10, 11, 12, 13]. The disease does not attack any plant outside the genus Ribes, of which about 25 species, including the wild and the commonly cultivated varieties of currants, red, white, and black, together with gooseberries, are susceptible.

Currant bushes affected with leaf spot suffer considerably from under-nourishment brought about by the premature fall of the leaves. It occurs at a most critical time of the year, about early August, when the leaves are at their greatest capacity in the manufacture of food reserves for the following year. From experiments conducted at Long Ashton in 1928, the weight of buds alone, from bushes which had been specially guarded against infection, was 15 per cent. heavier than that of buds from unprotected infected trees [10]. In the United States a direct loss, as high as 75 per cent. of the fruit crop, followed upon infection, defoliation, and reduced vitality of the bushes, and what little fruit was formed was low in sugar content and of poor quality [1]. Experiments conducted in the Soviet Union showed that from 30 to 32·6 per cent. of carbohydrates and 28·5 to 33·4 per cent. of lipoids were lost from the reserves normally laid down in the storage tissues of healthy plants in the autumn, losses which accounted in a reduction of yield to less than half the weight of a normal crop, due to this disease [16].



FIG. 388.—Leaf spot of currant (*Pseudopeziza ribis*)

Leaf spot is essentially a disease of the mature foliage (Fig. 388), but may occur in lesser degree on any part except the wood [7]. Early symptoms consist of a number of dark-brown spots, irregularly scattered over the whole lamina and visible from either side of the leaf. The spots may either be sharply delimited by the leaf veins, or surrounded by a light-yellow margin. They are about $\frac{1}{16}$ inch across, and may so increase in number, largely as a result of secondary infections, as to cover large patches of the leaves which turn brown and decay. Sometimes, soon after the first appearance of the spots, the entire lamina may turn more or less yellow, but there is still evident a narrow green zone around each brown spot, and in addition, in the outer yellow area, specks of red or purple may appear. Brown spots may also be seen on the leaf stalks and flower stalks, and the lesions may so girdle these parts as to produce canker, with consequent loss of leaves and flowers. Tiny specks of discoloration may also appear on the fruit. When the disease attacks the shoots, the lesions occur usually on the non-woody young parts, on which the spots consist of small superficial areas of a golden-brown colour [1]. In the west of England, infection is first noted usually about the middle of June, and by the time the fruit is ripe spotting of the foliage is fairly general over all but the youngest leaves. By the end of August the bushes may be so denuded as to present a gaunt appearance, and by mid-September may be entirely bare of foliage [12].

Leaf spot of the currant is caused by *Pseudopeziza ribis*, an Ascomycete of the group Discomycetes [8]. The conidial stage (*Gloeosporium ribis*) occurs on the living tissues, and the perfect apothecial stage completes its development on the fallen leaves on the ground or on those caught in the crotches of the branches of the tree or other bushes close to it. The conidia, which are developed in acervuli on the leaf spots, are released in such quantity as to form white, slimy masses often spreading as a film over the entire spot. Conidia are hyaline, curved or luneate, unicellular, uninucleate, 20·5 to 23 by 5·3 to 5·9 μ [1]: smaller, microconidia, rod-shaped, with rounded ends, 8·9 by 2·3 μ, of unknown function, intermixed with functional conidia, or in separate acervuli, are to be found mostly towards the end of the season. The late appearance of the microconidia in the life-cycle, synchronising with the initiation of ascocarp development which takes place before the fall of the leaf, seems to indicate that they may function as fertilising agents, and it is probable that ascocarps remain abortive without their intervention [1]. The apothecia, on lamina and petiole are small, gelatinous, yellow, slightly erumpent; the club-shaped asci are 8-spored, intermixed with paraphyses; the ovate ascospores are 15·5 to 20·2 by 6·7 to 9·3 μ [1, 9].

Apothecia discharge their spores in early March and continue to shoot ascospores until early May. On the over-wintered leaves conidia may frequently be found in addition to the functional ascocarps, and it is not improbable that over-wintered conidia in some localities prove, like the ascospores, to be capable of starting primary infections in the spring, but this observation appears to require confirmation [5].

Different races of the fungus attack red currant, black currant, and gooseberry, but the form to which the gooseberry is susceptible has apparently lost the capacity to produce apothecia, and seems to survive in the form of conidia which presumably must be able to tide through the winter [9]. The form which attacks red currants in England is furnished with spores slightly different in shape from that seen on black currant [17]; other forms of the fungus differ in their pathogenic powers [1].

When the conidia germinate, they early become one-septate and, grown on potato-dextrose agar, at 20° to 24° C., the resulting mycelium produces conidia again in great abundance, but the microconidia do not appear until much later, on old cultures, and only at reduced temperatures ranging from 8 to 16° C. [1]; mature apothecia have not been observed in culture [1].

Primary infections occur when the trees are bursting into leaf and while the earliest infections of all, on the young leaves, may be of ascosporic origin, soon to produce acervuli containing conidia, the bulk of infections are probably secondary, arising from the dispersal of the newly formed conidia which would be ready about the time when the leaves are expanded and mature.

Penetration by ascospores or conidia occurs at either leaf surface, mainly the under side [7], and is direct through the cuticle into the epidermis. An infection, confined largely to the epidermal cell invaded, together with a few adjacent cells of the mesophyll, develops an inter- and intracellular mycelium which soon destroys the occupied mesophyll, often from one epidermis to the other, and in place of the destroyed tissues the fungus collects in more or less dense masses. The conidial acervuli arise just below the epidermis. With the decline of the season and approach of leaf fall, the defunct acervuli collapse, and with the mycelium within the leaf becoming thicker-walled and closely matted together, the fungus is enabled to tide the winter on the fallen leaves. Leaf infections develop conidia readily during moist periods of 12 to 24 hours, over a range of temperature from 10° to 28° C. but not beyond 30° C., the optimum lying between 16° and 24°, and for ascosporic infections the range is from 12° to 20° C., the period of incubation being from 8 to 9 days. There does not appear to be any consistent correlation between *p*H of the leaf sap, derived from young or old leaves, and facility of infection.

The maintenance of currant bushes in good vigour by manurial treatment enables them to withstand infection better than untreated trees. The varieties Boskoop Giant and Seabrook's Black are not as susceptible as the Baldwin, the liability of which is probably due chiefly to its inherent capacity for heavy cropping, which weakens the trees to such an extent as to result in lack of resistance to infection. The varieties Goliath, September Black, Edina, and Victoria are less susceptible. The red currant types, Fay's Prolific and Fertility, are very susceptible, while Earliest of Fourlands and La Constante are among the more resistant kinds [12, 17].

Spraying with Bordeaux mixture (3 : 4 : 50) gives good protection against leaf spot. The first application is given just before the plants bloom, a second just after the fruit is set, a third some three weeks later, a final treatment being given after the fruit is picked. Lime sulphur, 1 in 40, is also recommended, the first application being made when the leaves are unfolding, and others at intervals of 10 to 20 days, until 5 or 6 sprayings have been made, depending on the weather; if the weather is dry over an appreciable period, fewer applications suffice [2, 5, 10-12, 13, 14, 15]. Since the fructifications hibernate on the fallen leaves, the latter should be collected and burnt.

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Reversion Disease of Currant

This disease of the black currant appears to have been first noticed in Holland and Germany about 1904 [5], and in England about 1912, on the variety Boskoop Giant, but is believed to have been existent here before that date [1]. Its true nature was not revealed until some years later and it is now known to be due to virus infection. The trouble is by no means easy to diagnose from other disorders of the currant mentioned below, but a peculiar abnormality in the venation of the leaves on certain branches of the tree is associated with it, and while the effects may also be seen in an irregular or deficient cropping, the final results are a degeneration, reduced fertility, or complete sterility of the bushes [2].

The clues to a ' reverted ' condition are not usually evident in all parts of an affected bush. They must be looked for in the foliage developed from buds formed in the previous season, in the leaves situated towards the *top* of the shoot, or at the *base* of the previous year's wood. No reliance can be placed on an examination of other branches on the host, for perfectly healthy bushes may bear leaves of very irregular character which may quite easily lead to wrong conclusions.

A normal healthy leaf of the black currant is simple and five-lobed, each lobe having a principal or main vein radiating into it from the top of the leaf stalk, and furnished with a toothed margin (Fig. 389). For the correct diagnosis of ' reversion ', attention need only be focused on the character of the largest *terminal lobe* which is delimited from its adjacent lateral lobes by a dent or sinus. In this

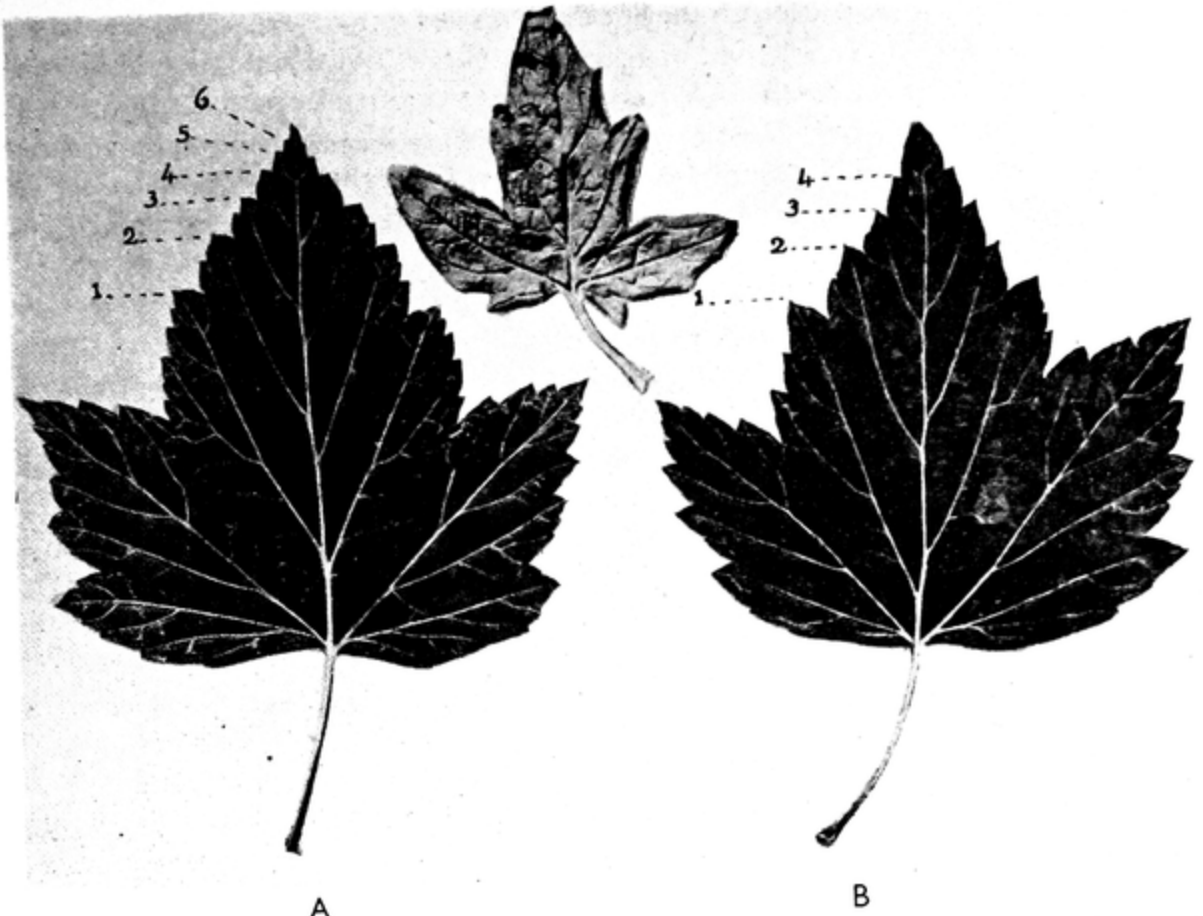


FIG. 389.—' Reversion ' of currant. *A*, normal leaf has five or more main branch-veins passing into the serrations of the terminal lobe. *B*, a ' reverted ' leaf shows in its terminal lobe usually less than five (photo by Amos & Hatton, *J. Pomology*, by permission of East Malling Res. Station). The small leaf shows severe reversion (photo by Foister & Noble)

middle lobe of the normal leaf there are usually five lateral or sub-veins passing out on each side from the main vein or midrib into certain teeth at the margin. Just above the sinus, no sub-veins pass out into the serrated margin. Correct diagnosis of reversion will depend upon an examination of the number of sub-veins, and of the character of the serrated margin *above* the sinus, in the terminal lobe. Other diagnostic features are mentioned below, which relate to abnormalities in leaf texture, inflorescences, and flowers, but they are of subsidiary importance to those concerned with the venation and serration of the terminal leaf lobe.

Referring again to Fig. 389, the sub-veins in the normal leaf on the left are numbered 1 to 5, and while there may be, in different varieties of currant, as many as seven or eight sub-veins (Baldwin, Boskoop Giant, and Goliath rarely have less than six), a reliable clue to a reverted condition is the presence of *less than five sub-veins* on either side of the midrib in the middle lobe of the leaf, *with a coarser serration of the margin above the sinus* than in the normal leaf, as on the right. In a more progressive stage of the disease, as shown by the smaller leaf, the diagnostic symptoms are much easier to detect, but such leaves are quite commonly found, and show clearly a reverted condition. Indeed, such abnormally shaped leaves, developing in appreciable number on reverted bushes, have accounted for such names as ' nettle leaf ', ' tomato leaf ', or ' oak leaf ' being

used to describe the condition, but they have fallen out of use. Neither is the acceptance of ' reversion ' a happy one, for it is entirely misleading, and the condition bears no relation at all to a possible throw-back on the part of the host to a pristine condition in a simpler ancestral type of leaf. These abnormalities are traceable to a definite infection, with ample evidence that it leads to one of the most troublesome of all disorders which attack currant bushes in this and other countries.

Other fairly constant features attendant on reversion may be mentioned. Reverted leaves are generally smaller and relatively longer and narrower than normal leaves, have a flatter base, and a more coarsely rugose surface, below which the network of fine veins is not so elaborately and delicately patterned as in the smooth-surfaced normal leaves. Moreover, reverted foliage during the growing season is deeper green in colour than the healthy leaves.

At flowering time, the character of the trusses and a closer examination of the floral parts will also furnish clues to the presence of the virus in the plants. The inflorescences on affected bushes tend to become longer, and the stalks of the individual flowers are also longer than usual. The flowers themselves become more tubular than the normal urn-shaped flowers ; the sepals are more pointed, hairy, and more highly coloured than normally, while the petals are much narrower ; the style lengthens so as to carry the stigma well out of the flower, so that pollination and consequently fertilisation is interfered with or made impossible ; sometimes the abnormalities extend to the pistil, so that a superior instead of an inferior ovary is developed. Such floral abnormalities may allow for only a partial setting of the fruit, most of which in any case is lost, or may lead to complete sterility.

Currant bushes are liable to suffer from other affections the symptoms of which are not unlike those presented by ' reversion '. Thus a failure to set fruit, a condition frequently called ' running off ' [13], is often observed in healthy bushes in consequence of inefficient, or lack of, pollination, due perhaps to a scarcity of hive bees. Symptoms more closely resembling those due to reversion and known as ' false reversion ' may be seen in the leaves which develop from dormant buds when shoots from the latter are induced to make precocious growth, on account of injury to the tips of the shoots. The leaves from such buds are often wanting in the correct number of sub-veins of healthy leaves. A similar condition may also be frequently detected on bushes which have been cut down to the ground, for the buds which then arise develop shoots on which the leaves show abnormalities ; it will be recalled, however, that reverted leaves are to be looked for at the *top* of affected bushes. Finally, another condition known as ' nettlehead ' is produced when a number of lateral buds, instead of forming inflorescences, give rise to leafy shoots, so that a heavy-topped, unusually bushy appearance is presented, and this again is believed to be due to accidental damage [4].

The virus causing this disease has been named *Currant Reversion Virus* (Lees 1920 ; Amos & Hatton 1926 ; Amos *et al.* 1928) ; *Ribes Virus 1* (Smith) ; *Acrogenous Ribis 4* (Holmes *Hb.*) ; Black Currant Reversion Disease Virus. It is not sap-transmissible, nor can it be conveyed by the pruning knife, but can be transmitted by grafting [9]. The ' big bud ' mite *Eriophyes ribis* is, at least, one agent in its

transmission [2]. It is also possible that aphides may be additional vectors, but there is no transmission by seed.

The virus produces no striking pathological effects in the wood of affected trees. There may, however, be a certain amount of reduction in the increments to the wood, which appears to be reflected in a corresponding increase in the amount of medullary-ray tissue, with a tendency to produce more gum in the reverted plants than in normal [2].

Reversion should not be difficult to eradicate. In addition to its identification from abnormal leaf characters described, if the plants are examined in June and July before the crop is picked, bushes showing decreased fertility should be grubbed out and destroyed, for it is not safe to take cuttings from any parts of them, however healthy they may look, since the virus permeates all parts of the bush. As the virus is not soil-borne no danger would be incurred if a sound bush is planted to replace one pulled up. Cuttings should be taken only from healthy plants, preferably not more than five years old. When new stock is purchased, satisfaction should be obtained that they have come from a source certified to be free from virus [4].

'Big bud' infestation should be controlled by spraying with lime sulphur of specific gravity 1·025, which is roughly a concentration of 1 gallon in 12 gallons of water; or 1 in 25 gallons, for varieties of the Goliath group (Victoria, Edina, Monarch), the operation being performed towards the end of March, as soon as the flowers are seen, but before they open.

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Downy Mildew of the Vine, *Plasmopara viticola* (Berk. & Curt.) Berl. & de Toni

Downy mildew of the grape-vine is much more common and serious on the Continent, in North and South Africa, and America than in Britain, where it is more or less localised [19, 21, 25, 32, 41]. It occurs also in Australia and New Zealand [17, 18, 43, 54].

The disease is caused by *Plasmopora viticola*, a member of the *Peronosporaceae* [6, 7, 23]. There are two stages in the life-history of the organism, namely the spring-to-summer asexual or sporangial phase which is responsible for secondary infections throughout the growing season, and the over-wintering or resting stage



FIG. 390. Downy mildew of grape vine (*Plasmopara viticola*). *A*, the asexual fructification; oospore formation on the left. *B*, haustoria. *C*, sporangium and formation of zoospores (after Millardet, *Die natur. Pflanzen*).

consisting of oospores which germinate in the spring and probably account for the primary infections on the new growth.

Downy mildew attacks all parts of the vine except the roots. The disease may strip entire plants of their leaves and tendrils, flowers may fail to set, fruit may be destroyed in the early stages of growth, or if it succeeds in passing the susceptible stage, the grapes are small and of poor quality owing to the impairment of leaf functions. Early symptoms of the disease may occur in some localities in February or March, but in most places first outbreaks occur in June. They consist of round light-green spots of an oily appearance on the upper side of the leaves, and on the corresponding under side tufts of white downy sporangiophores soon appear, bearing the asexual spores or sporangia in great numbers (Fig. 390 *A*). Later the spots change colour, becoming yellow, or variegated with tints of yellow and reddish brown, and forming patches of irregular shape, especially between the larger veins of the leaf. At this time the leaves are in a moribund condition, and the fungus within enters upon the sexual phase, producing oogonia, antheridia, and finally oospores which survive the winter in the fallen leaves. On the stems the lesions are brown and sunken, and with the death of the affected parts portions of the vine become brittle and break off easily. Similar lesions may also occur on the stalks of the inflorescences, or on the stalks of individual flowers or berries, with the result that entire bunches or a few berries here and there fail to develop, and it is quite common to see healthy berries along with diseased ones in the same bunch. When the berries are infected during early growth they present the characteristic downy appearance similar to that of affected leaves, and soon shrivel up; when, however, they are infected later, after attaining the size of a pea, or more (the fungus having entered through lesions in the stalks [46]), the berries assume a reddish-brown colour and, failing to ripen, develop a soft rot. The berries become more or less resistant to attack once they begin to develop their natural colour, but even then, if they are growing on a vine whose foliage is diseased, the quality of the juice may be affected, owing presumably to the deleterious action of the fungus present in the leaves [54].

The whitish-grey fructifications of the mildew on the leaves or other parts consist of long, branched sporangiophores, 300 to 500 by 7 to 9 μ wide, which terminate in short sterigmata bearing oval-shaped spores (at first uni-, later multi-nucleate) [26], measuring from 15 to 31 by 11 to 18 μ. As many as 20, but usually only some 4 to 6 sporangiophores pass out through a stoma from a mass of mycelium in the leaf. These fructifications arise also on parts of the flower, flower stalks, tips of young stems, as well as on young berries, but only when all these parts are still in possession of their stomata [24].

Later in the season, usually towards September, infected tissues, especially in the leaves, show the development of oogonia, antheridia and oospores ; oospores may also be found in the cortex of the stem, and in the stalks and flesh of rotted berries [1, 24, 25]. Little is known, however, about the development of the sexual organs and of fertilisation ; antheridia are frequently wanting, and oogonia with oospores have been found without antheridia, the resting spores having presumably been formed apogamously [24] ; some report the formation of oospores to be a rare phenomenon [27]. The oospores are spherical and measure from 25 to 35 μ in diameter.

In general, the sporangia are not adapted for long survival but serve to spread the disease throughout the summer. In certain localities, however, where the leaves may remain on the vines all the year round, infections by sporangia may be continuous from season to season [39]. On the other hand, the oospores are capable of survival in the soil for at least a year. Oospores which remain in the fallen leaves, vine debris, or shrivelled berries left to over-winter on the ground are probably responsible for the primary infections which break out in the spring. Moreover the fungus may, in some localities, survive in the form of mycelium perennating in the winter buds, but its presence has not been established in all cases [5, 24, 27, 36, 37].

Primary infections from oospores germinating in the soil are probably relatively few in number, but sufficient, perhaps, to establish foci of infection. The oospores, after stimulation by frost, germinate over a wide range of temperature, from 13° to 33° C., the optimum being about 25° C. Germination of the oospore is direct by a short germ-tube producing a single terminal sporangium which gives rise to a number of zoospores. Primary infections are believed to occur when the zoospores are conveyed by splashing rain-drops from the soil on to the lowermost leaves of the vine, or the sporangia themselves may be carried from the ground to the host. In any case the earliest symptoms of all usually occur on the leaves near the ground. The suckers that develop at the base of the plants are also very prone to take primary infections, and appear to be greatly instrumental in spreading the disease. The spores of *Plasmopara*, whether produced, as above mentioned, from the germinating oospores or from mycelium on any part of the host, germinate almost exclusively in an indirect manner (Fig. 390 C), that is, they behave as sporangia, by giving rise to motile zoospores which are the actual infecting bodies. The zoospores are biciliate and pear-shaped, and measure from 7·5 to 9 by 6 to 7 μ. After coming to rest and secreting a membrane, a zoospore puts forth a germ-tube which enters the host through a stoma [2, 23] or a water-pore [31] in the leaf margin, and by the elaboration of an intercellular mycelium the spongy mesophyll soon becomes invaded [24, 33] and numerous globular haustoria enter the cells. The invasion of the palisade, from the spongy mesophyll soon follows, and the presence of the fungus in the former tissue accounts

Disease

for the appearance of the oily spots on the surface of the leaf, the chloroplasts undergoing fatty degeneration and the lipoids disappear during infection [7a].

From the necessity of finding stomata for entry, the fungus can infect the berries only whilst they are still young, before their stomata change over to lenticels or become occluded through the development of waxy bloom. The older berries, apparently, become infected from within, from mycelium which travels into them from infected stalks, or, farther afield, from the leaves, by way of the axis of the inflorescence. Apart from stomatal entry when young, vine shoots and buds may become infected in the same way by migration of the fungus from the leaves, but little appears to be known about the origin of internal infections in the vine, though it has been established that the fungus can occupy even the tips of the growing shoots [5]; but there is no definite evidence that a mycelium perennating in the buds plays any substantial part in starting the disease in the spring.

Various factors of the environment play an important part in the incidence of downy mildew of the vine. The period of incubation varies with the locality, the weather, the variety of host, the seasons, the water-content of the tissues [8, 9], and the part of the plant affected [24]. Conditions of heavy humidity are conducive to much disease and the trouble in some localities is worse in damp, low-lying than in elevated situations [3], and yet in other places, where vineyards are established on dry, hilly slopes, the disease has been known to break out earlier and the vines to be more severely attacked than in the plains. The explanation appears to be that the slopes, in a position to receive the maximum of direct sunlight and the first to catch the warm rains of spring, offer favourable conditions for early outbreak of the disease [49, 51], and for this reason growers prefer to plant the vineyards with a north-to-south aspect, with due regard to good spacing so as to ensure quick drying of the vines after rain [11]. The disease is considerably worse if, during the preceding winter, the weather has been continuously wet, and this despite the fact that during the primary infections conditions may be comparatively dry. It is a common observation in some of the French vineyards that if the winter has been dry, the vines show but little disease even if the growing season happens to be a wet one [16]. But heavy rainfall with high summer temperatures lead to widespread attacks of vine mildew [10]. In Tunis, in 1915 and 1926, there were no fewer than 11 and 9 waves of infection during these rainy years respectively, whereas in dry seasons, or those with infrequent rains, the amount of disease was negligible, and in this locality the onset of the dry sirocco winds does more to check the disease than any treatment devised for its control [19].

An atmospheric humidity of 70 to 85 per cent. on young, and 80 to 100 per cent. on older leaves provides the necessary conditions for primary infections [38]. While primary attacks thus appear to follow periods of prolonged wetness, it is not essential for the host to be continuously wet for the success of secondary infections, and the wind-borne spores may germinate on the host after showers, or mist, or dew, provided the parts remain moist for a few hours. Infections appear to make better progress during the hours of night than of day, the minimum temperature being 12° to 13°, the optimum 18° to 24°, and the maximum 30° C.; low temperatures check the germination of the spores [1, 24, 26, 36, 37, 38, 52].

There is no evidence that nitrogenous fertilisers are in any way conducive to

downy mildew [12, 35], and an excess of phosphoric acid, potash, and lime tends to increase resistance in the host [48]. The application of calcium is said to reduce the amount of disease. Vines in general are said to be good calcium acceptors, possessing an 'internal resistance' which seems to depend on the amount of this substance present in the aerial organs, this in turn varying according to the availability of the mineral in the soil; some varieties of vine, however, including the wild *Vitis vinifera*, absorb relatively little calcium and are very susceptible to the mildew [3].

The relative concentration of the cell sap is considered to be a factor in respect of resistance to, or immunity from, downy mildew. According to some, resistance in the plant-organ, whether leaf or berry, increases as the water-content falls below a certain percentage, but others state that cryoscopic tests failed to show any significant difference in the concentration of the cell sap of resistant and susceptible vines [29, 42]. Acidity values of certain vines, more or less resistant, were found to vary from 4·3 to 10·3 per cent., whereas the more susceptible varieties had a range from 0·5 to 2·6 per cent. acidity [4]. Moreover, an important factor influencing relative resistance appears to be the value of the calcium-potassium ratio of the sap. Thus old leaves contain more calcium than potassium, while the reverse is the case in young leaves, which are more susceptible; this ratio, again, is much smaller for berries than leaves, in which it becomes greater than unity about flowering time, increasing during the summer, and thereafter declining, the period of increase coinciding with a high degree of resistance, in the leaves, to infection. Finally, there is some evidence to the effect that internal resistance of vines to downy mildew is affected by the type of stock on which the vine is grafted [3, 21, 54].

For the control of this disease emphasis is laid on the value of timely and early spraying, and of the thinning of the foliage during the growing season in order to gain better access for sprays and dusts [10, 12, 55], and of the removal of the shoot tips in order to increase resistance in the remaining parts [47]. It is interesting to note that the well-known fungicide, Bordeaux mixture, was first discovered and improved upon during experiments conducted in France for the control of this disease (p. 233). To avoid scorching, the mixture is employed slightly on the alkaline side [44, 45], the strength being 1·5 per cent., usually, or 2 per cent. in wet seasons [13-15, 46], the proportions being 6 : 4 : 40 or 8 : 6 : 40. The first application is made soon after the buds open, a second being given before the flowers open, and a third after the fall of the petals, a final application being given 14 days later [40, 54]. Towards flowering time some growers discontinue the wet spraying and follow up with the dry powder, directed chiefly towards better covering of the fruit clusters, and when the foliage has attained full development dusting is reported to be more effective than spraying [16, 20, 22, 45].

Certain methods of cultivation are helpful to ward off the disease. Vineyards should be sufficiently open to allow the foliage to dry quickly after rains, and in some localities it is the practice to build up the vines on trellis-work, this method not only ensuring free access of air but facilitating spraying as well, and young growth can be tied up so that the risk of infection from resting oospores in the soil is lessened. All green shoots or suckers that develop at the base of the stocks

should be removed, as they are very susceptible to primary infections (34, 49). Soil cultivation, manuring, and hoeing should be finished before the blossoms open, so as to leave the soil undisturbed during the spring, when the conditions are favourable to the germination of the resting spores (34, 51).

A certain measure of success has been attained on the Continent in the breeding of resistant varieties from European vines, some of them showing somatic mutation in respect of resistance to downy mildew, and the results so far obtained are encouraging that immune varieties may soon be available (50, 50a), but the mode of inheritance is little understood (30). Even with so-called resistant vines, spraying with Bordeaux is advisable (28).

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Powdery Mildew of the Vine, *Uncinula necator* (Schw.) Burr

This disease of grape-vine is much better known in Britain than downy mildew and anthracnose affecting this host. It is reported to have first appeared in this country in 1845 in Kent, and two years later in France, where it assumed such serious proportions that by 1851 there was hardly a vineyard throughout the whole country which was not affected with mildew [1], and in a short time the trouble spread along the Mediterranean coast into Italy, Greece, Syria, Asia Minor, Algeria, and Hungary [3]. The mildew is also prevalent in India and Australia but only in the wetter areas, and is rarely found in the drier inland districts [3, 4, 5]. It is also common throughout America; in California the ordinary vine *Vitis vinifera* is much more susceptible than *V. labrusca*, *V. riparia*, and *V. rupestris* [2].

Hothouse and outdoor vines are liable to be attacked by mildew at any stage of growth. The symptoms of powdery mildew can be distinguished from those of downy mildew (*Plasmopara viticola*) by the presence of white powdery patches of mycelium and floury conidia mainly on the shiny upper surface of the leaves, whereas the whitish areas of fungus and spores of downy mildew occur mostly on the under side, the whole presenting a glossy appearance, but powdery mildew produces a diffuse dusty film which gradually turns grey and finally a dark colour (Fig. 391). Stems, leaves, tendrils, flowers, and, in particular, young fruit bunches, are liable to be attacked (Fig. 392).

If the blossoms are infected, no fruit is set, and early infection of the berries checks their development so that they drop off. When older berries are attacked they may still continue to develop, but growth is distorted and the skin becomes cracked. With further progress of the disease on the leaves, the white patches tend to spread and join together so that large parts of the lamina are covered with mildew and the leaf curls up towards its upper surface. As the shoots lengthen the



FIG. 391.—Mildew of grape vine (*Uncinula necator*). The powdery appearance of the mildew on the leaf (photo, Adv. Lft. 207, by permission of Minis. Agric.)



FIG. 392.—Mildew of grape vine (*Uncinula necator*). The disease on the stems, nodes, and berries of variety Waltham Cross (photos by du Plessis)

young stems are attacked in the same way, and the patches here soon change from white to grey, and if the fungus is rubbed off, brown or black marks are left on the stem. Vines attacked when young fail to mature properly and may turn black all over (Fig. 392).

Mildew or powdery mildew of the vine is caused by *Uncinula necator* (a member of the *Erysiphaceae*) which replaces the old name *Oidium tuckeri* given to it in 1847 [1].

Like other fungi of the powdery mildews, the mycelium of *U. necator* is entirely on the surface of the part attacked, except for haustoria which penetrate the epidermis. The mycelium is very slender, branched, and septated, forming a delicate white web which darkens as soon as the production of conidia is over. The conidia are developed in great abundance on leaves and berries, on which they form a dense fluffy or powdery film. They are developed from erect conidiophores, 3 or 4 in series on each stalk ; they are oval in shape and measure from 25 to 30 by 15 to 17 μ. The conidia are capable of resisting dry conditions, for how long is not clearly known, but in some districts it is thought that they are capable of survival over the winter [3].

Cleistocarps are usually developed in late autumn and winter but in some localities may appear as early as June [2]. They may be found embedded in the epiphytic mycelium on the leaves, or on shoots, chiefly at the nodes, or on buds among the scales and hairs. They are black when ripe, almost spherical, flattened at the top, from 75 to 105 μ in diameter, and furnished with 8 to 25 septated appendages, coiled at the free end. Each cleistocarp contains from 2 to 8 ovoid asci, 48 to 60 by 37 to 45 μ and each holds usually 6 oval ascospores 20 by 13 μ [2, 7]. Other cleistocarpic dimensions given are: 70 to 128 μ in diameter, mean, 98 μ ; appendages, 7 to 32 in number, light or dark amber-brown in the lower half ; asci, 4 to 6, rarely 6 to 9, from 50 to 60 by 30 to 40 μ ; ascospores, 4 to 7 per ascus, measuring from 18 to 25 by 10 to 12 μ [10].

In localities where they have been found, cleistocarps are believed to carry the fungus over from year to year, but they are not common. They have not been found in Britain and other countries, including Australia, but have been reported from America, France, Germany, and at Anapa in North Caucasia [2, 7, 11]. Even in the same localities cleistocarps are formed only in some years, and may be looked for in vain in other years. Apparently environmental factors are partly responsible for their appearance. In some years their development is associated with soil dryness and prevalence of an unusually warm, rainless summer. It is

recorded that in the interior valleys of California cleistocarps are never found, or rare, whereas they occur plentifully on vines subject to the influence of summer ocean fogs. Under these weather conditions if, after an abundant growth of mycelium has been produced, the temperature drops suddenly to the lowest limit for fungal growth (50° F.), the cleistocarps are developed in great numbers [2]. There is evidence that earliness of attack followed by severe infection is also conducive to development of these fructifications, whereas their number is greatly reduced in late and lighter attacks [11].

Cleistocarps remain on the vines and leaves or on host debris on the ground until spring, and the spores are said to be capable of germination 18 months after formation and of infecting leaves and shoots in the same manner as the conidia [2]. In the absence of cleistocarps it is believed that primary infections in the spring are traceable to mycelium which has passed the winter in the buds [9], or to conidia which have survived the winter [3].

A moisture-laden atmosphere is more favourable than a wetted surface to the germination of the conidia on the host. The fungus thrives vigorously on vines in sheltered, shady positions such as walls or trellis and in the humid atmosphere of the greenhouse, but makes little growth in ventilated houses or in the open vineyard. The fungus can grow over a wide range of temperature and humidity. Below 50° F. (10° C.) growth is arrested; above 75° F. (24° C.) growth is rapid and reaches a maximum at about 90° F. (32° C.) or 95° F. (35° C.), but is not killed at 42° to 43° C. [2, 6, 12, 14].

Sultry, warm conditions with dull, cloudy weather are favourable to attacks of vine mildew; bright, crisp sunny days retard it [3, 12]. Different varieties of vines vary considerably in their reaction to mildew, but all of them are liable to injury if weather conditions favour growth of the fungus [2].

After due attention has been given to cultivation of the vines, such as pruning after shedding of the leaves, thinning out and cutting back of laterals, together with removal and destruction of all diseased material, some growers adopt a dormant treatment of washing the vines with an acid solution of iron sulphate, or of coating the stems with a paste of sublimed sulphur and soft soap, the object being to check the formation of mildew when warmer weather comes, the higher temperature causing the finely divided sulphur to fume or vaporise, so that growth of the fungus is checked. The same principle underlies the painting of hot-water pipes in the greenhouse, with a mixture of skimmed milk and sulphur. But the universal treatment against vine mildew is sulphur dusting. Sulphurating, preferably with a bellows or vaporiser, must commence at the earliest possible signs of mildew and should be carried out in the early morning while the leaves are moist, but not in very hot weather, which may cause scalding [4]; and if, from experience, the time of flowering under glass is known, the first sulphur treatment should be given 10 days before this event and continued at weekly intervals until the flowers open, then withheld until the fruit has set, a final dusting being given when the berries are about as big as peas [6, 8, 13, 14, 15].

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Anthracnose of Vine, *Elsinoe ampelina* Shear

This disease, though uncommon in Britain, is one of the oldest of vine diseases in Europe. In South Africa it causes severe losses every year in the wetter districts, especially on sultana varieties [2, 9]. It is serious in practically all vineyards on the Continent as well as in the eastern parts of the United States, and is also one of the most destructive of all fruit diseases in West Australia, especially in the coastal plains and foothills, the newer grape-growing areas away from the coast being almost free from it [4].

Anthracnose of the vine, so called from the black carbonaceous appearance (' charbon ' or ' brenner ') of the lesions on the stem [5, 10], is believed to have originated in Europe [13], and to have spread thence, probably from France or Italy, into America and Australia; the earliest report of its occurrence in the United States in 1885 appears to have been made in Illinois [5, 13].

All parts of the vine may be affected, including the berries, leaves, stems, and tendrils (Fig. 393). Small, more or less circular, greyish-black spots surrounded by a yellow border appear on the leaves and remain small if crowded together; isolated spots enlarge and become light grey at the centre, the outer edge turning dark brown or purple. The central part of the spot becomes sunken, and if it dries may drop out, leaving a shot-hole. On very susceptible varieties, however, during prolonged wet weather, even in winter, the larger spots continue to develop and present a woolly appearance from the presence of spores on the surface. Spots on the leaf veins and on the stem are small and elongated, and, if numerous on the veins, may join up so that the lamina becomes curled and torn, and long portions of the veins turn yellow.

Young shoots up to about two inches long are even more susceptible than the leaves. On the shoots the spots remain small, irregular, and almost black, and from the numerous lesions that may arise an entire shoot may be killed within a short period, meantime becoming black, dry, and hard. On older shoots the spots enlarge and develop a light centre with a brown margin, the central part later becoming cracked and sunken so that a canker is formed. Sometimes a stem canker penetrates into the stem so as to destroy the tissues as far down as the cambium. Within such lesions the mycelium of the causal fungus, though sparsely developed, remains dormant over winter and under favourable conditions in the spring revives to form spores which start the disease afresh. Stem cankers

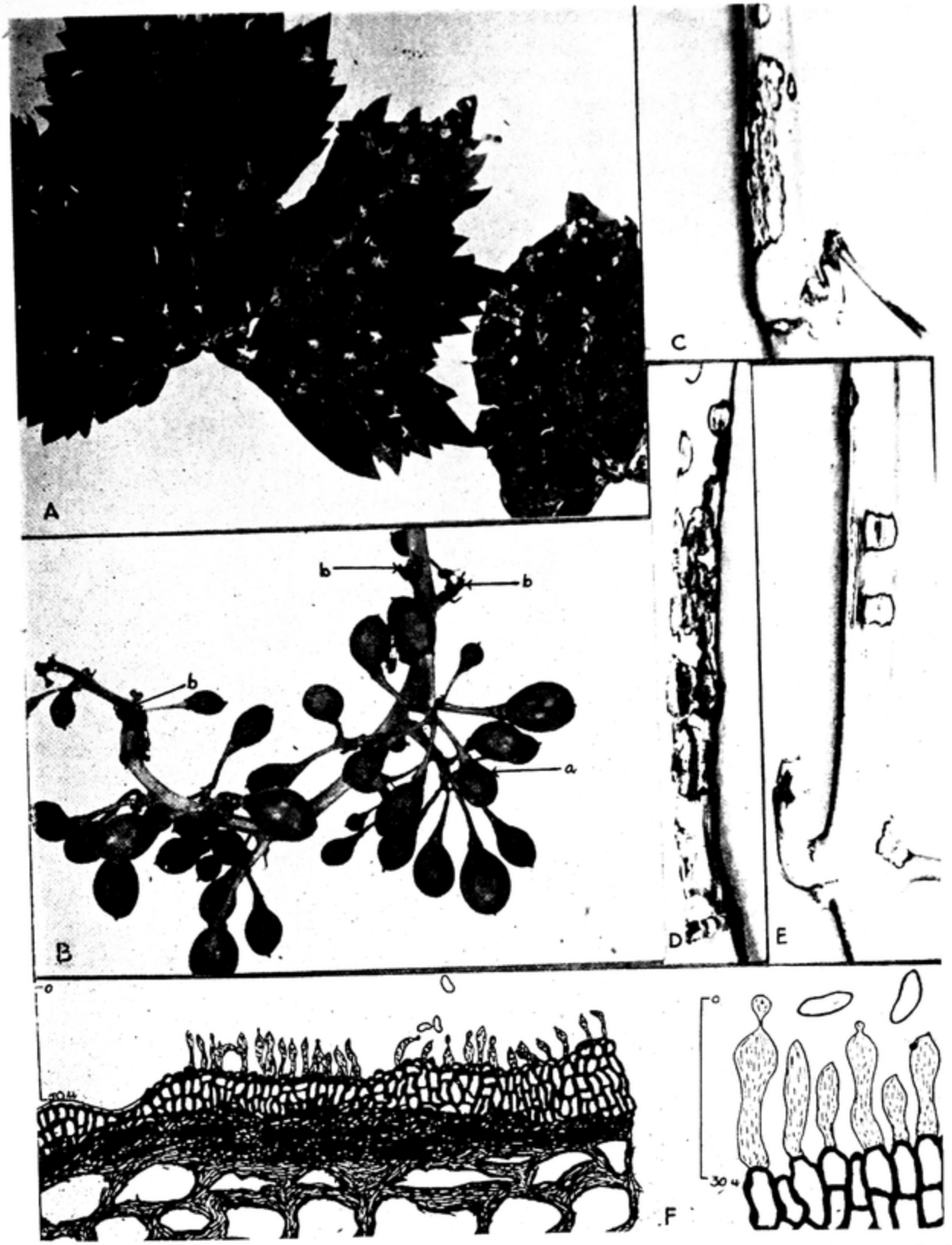


FIG. 393.—Anthracnose of grape vine (*Elsinoe ampelina*). *A*, the spots on leaves of variety Red Hanefoot. *B*, young anthracnose spots on bunches of French grape variety ; *a*, on the berry ; *b*, on the stalk. *C*, *D*, *E*, the canker stage on stems of sultana. *F*, cross-section shoot of sultana showing the conidial fructification ; on right, details of the conidiophores and conidia (photos and drawings by du Plessis)

are sometimes found to cover considerable lengths of the vine, and if the woody tissues are seriously affected both shoots and stocks are rendered worthless.

Lesions also occur on the stalks of the inflorescences, and if girdling takes place entire bunches of fruit may be lost. Similarly, if girdling lesions occur on individual flower stalks, berries may drop at any stage of development. Spots on the berries are round, dull, and sunken, and later develop a light centre surrounded by a dark-brown border [9]. If attacked when small, the berries dry out and fall off, or may remain attached in the bunch for a long time, meanwhile becoming dry and mummified ; later attacks cause the berries to crack and become deformed [4]. When ripe grapes are attacked the spots develop into hard crusts, a condition which renders them, especially those of white varieties, unfit for table or raisin purposes [9].

Anthracnose of vines is caused by an Ascomycete, *Elsinoe ampelina*, better known under the name of the more common conidial form, *Sphaceloma ampelinum* de Bary [1], previously known as *Gloeosporium ampelophagum* (Pass.) Sacc. [6]. The ascigerous stage has been found in the spring on cankered stems that had wintered out of doors [13], but has not yet been seen in Britain. Ascocarps embedded in old cankers are ill-defined, pseudo-parenchymatous, with hyaline asci containing colourless triseptate ascospores which measure from 15 to 16 by 4 to 4·5 μ. The conidial stage occurs on spots on the leaves and berries as well as on cankers. The conidia are formed within sub-epidermal acervuli ; they are oblong-ellipsoid, bi-guttulate, hyaline, slightly constricted at the middle, 5 to 6 by 2·5 to 3·5 μ. Cyst-like bodies of variable size and colour have also been found, but their function is not known [13].

As above stated, the fungus is believed to survive the winter in lesions on the stems, and in which it revives again in the spring. The chief source of infection in new areas is the planting of cuttings harbouring the resting mycelium [4], Nothing appears to be definitely known about the spread of the disease from year to year by possible survival of the fungus in over-wintered leaves or berries, neither have the ascospores been observed to function in the dissemination of the disease.

Anthracnose attacks the vine when the leaves are young, and the foliage increases in resistance with age. Infections commence shortly after the buds come into leaf, and such primary attacks appear to result from spores which developed in cankers formed during the previous season. The spores are exuded from these lesions in gummy masses and during rainy weather are splashed or blown on to the tender green growth. Secondary spread during the season presumably takes place in the same way from the acervuli on leaf spots, young shoots, tendrils, and fruits.

Anthracnose of vine is greatly encouraged during warm wet weather, and there is usually little evidence of the disease in a dry season ; low-lying, badly drained plantations often suffer severely [4, 7, 9].

Varieties of grapes differ considerably in susceptibility to anthracnose. Sultanas, Malagas, Almerias, Muscats, and Grenachi are all susceptible [4], as are also Alicante strains and French and American varieties with thick leaves [10]. The varieties Gros Colman, Raisin Blanc, Barlinka, and Cabarnet are more or less resistant in Cape Province, South Africa [9].

For control of the disease the vines should be carefully looked over at the end

of the season and all infected shoots cut out and destroyed. All mummified fruit clusters and decayed leaves should be collected and burned, and the ground dug over or ploughed in the spring [12]. Since the trouble is carried to new plantations chiefly by cuttings, the latter should be immersed in a 25 to 30 per cent. solution of sulphate of iron before planting [11]. In South Africa, spraying the vines towards the end of winter with 4 per cent. sulphuric acid, or 1 in 8 lime sulphur, or with a solution of copper sulphate of 1 lb. in 2 gallons of water, is recommended; if winter spraying is omitted, early spring treatment, though not so efficacious, may be given by applying the same copper sulphate spray to the leaves, and dusting them during the summer with sulphur [9]. Many vine growers adopt a method of 'swabbing' just before the buds swell, using a mixture of iron sulphate and sulphuric acid in the proportion of 5 lb. of sulphate and $\frac{1}{2}$ pint commercial sulphuric acid in every gallon of water [2, 3, 8]. At the time when buds are bursting it is further recommended to spray with Bordeaux mixture, 6 : 6 : 40, or Burgundy mixture, 6 : 9 : 40, followed by a second application just before blossoming, a final treatment being given after the fruit has set [4].

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Chapter XVI

DISEASES OF ORNAMENTAL AND MISCELLANEOUS PLANTS

Rust of Antirrhinum, *Puccinia antirrhini* Diet. & Holw.

This serious rust disease of the snapdragon (*Antirrhinum*) was first discovered in 1895, in California and Oregon [3, 4, 9] and has been prevalent in the United States, Canada, and Bermuda, for many years [7, 8a, 10, 19, 26]. Outside these areas it was practically unknown until 1933 but soon spread at an amazing speed. In England, where the disease has been extensively studied [8, 13-18], it appeared in the Thames estuary on 2nd July of that year, and before the end of the season had travelled into thirteen counties and was, at the same time, reported in Jersey and north-west France. By 1934 it was present in twenty-eight counties in England and in 1935 was reported in Scotland and Ireland, and meanwhile fresh records came from Holland, Denmark, Germany, Czecho-Slovakia, Italy, and Austria [1, 2, 6, 23, 25]. In 1936 it was reported in Egypt, and in 1939 in the eastern Cape Province of South Africa [5, 12]. Since the host, *Antirrhinum majus*, is a native of the Mediterranean region where the rust was hitherto unknown, it seems fairly clear that the rust must have been indigenous on some Californian species of the plant and, having been introduced in some unknown way into the new areas, found in the cultivated species an unusually congenial host [22]. There is no evidence that the disease was carried by infected or contaminated seed [14, 24] and the probability is that dissemination took place by the widespread distribution of infected propagants in the form of cuttings, and that these, in turn, acted as foci for further spread of the rust through spore dispersal.

The rust (*Puccinia antirrhini*) attacks the host at all stages of growth, seedlings, cuttings, and mature plants about to flower being subject to it (Fig. 304). Seedlings may be attacked in the boxes and swept off with disease, and even slight attacks on grown plants so mar their appearance and reduce their vitality that shoots and flower spikes are much below normal size, and in severe attacks entire plants are killed. The parasite attacks the plants under glass and in the open and is most severe on cuttings and plants just before the flowers are due to open. Leaves, branches, stems, sepals, and occasionally seed capsules may be so badly affected that they are literally peppered all over with the brown spores of the fungus.

Early symptoms of the rust occur on the under side of the leaves, as small light-coloured spots which show right through to the upper surface. The spots soon produce brown pustules in great profusion, first on the lower side, and then on the corresponding upper side of the leaves. On the stems, the pustules are somewhat elongated and may sometimes so girdle the stem that entire branches



FIG. 394.—Antirrhinum rust (*Puccinia antirrhini*). *A*, on the leaves and flowering shoot (photo by Smith). *B*, on the seedlings (photo by Green, *J. Roy. Hort. Soc.*)

are lost. These pustules contain uredospores and, later on, dark teleutospores follow in the same places, on all parts, even on sepals and seed capsules.

The uredospores may be found on affected glasshouse plants at all times, and on plants out in the open frequently to the end of October and are more plentiful, in some localities, than the teleutospores, even after periods of frost. But though the uredospores may serve to carry the fungus from one season to the next under glass, survival over winter in the form of uredospores in the open, at least in Britain, probably occurs only in the warmer, southern counties. The uredospores arise in somewhat round sori, and are spherical or slightly oval, measuring from 19 to 30 by 15 to 22 μ (average, 22 by 19 μ) [14]; they are yellowish-brown, with an echinulate wall; are not viable for more than about 6 weeks, and are not adapted for carrying the disease over long periods.

The teleutospores are dark brown to almost black, two-celled, and measure from 33 to 52 by 17 to 24 μ; they are found either mixed with uredospores or in separate sori. They have been observed to germinate [18, 19, 21] but will not infect antirrhinums or any other plant and, so far as known, this rust has no alternate host on which an aecidial stage could develop. The fungus is known to possess at least two physiologic races and various strains [27].

In the greenhouse and in the open the disease spreads, therefore, under favourable conditions by means of uredospores [21]. In Britain, in the colder areas, it seems certain that the rust is carried over the winter on old infected plants on which uredospores are produced in the spring [18]. Though it has been

suggested that the disease may have been introduced into South Africa with infected seed [5], careful experiments conducted in England with seeds infected with the spores failed to give any indication of disease on seedlings grown in sterilised soil [18].

Uredospore infection from leaf to leaf externally takes place on an extensive scale and sporulation is so prolific that mere contact with the plants when cutting the flowers is sufficient to disperse the spores on a wide scale [23]. These spores germinate best at about 10° C., showing but slight growth at extremes of 0° and 30° C. [10, 22]. The germ-tubes produce appressoria before entering the stomata, and the developing mycelium, colourless and septated, passes in between the host cells, into which it sends small knob-like haustoria [14]. The period of incubation varies from 8 to 14 days [24].

In some parts of the United States plants which were deemed to be resistant to this rust were found later to have become highly susceptible owing, it is believed, to the appearance of another strain of the parasite, but this second race of the fungus has so far not been found in Britain [18]. In this country, the varieties Wisley No. 3 (magenta-coloured), an American stock named Orange Pink, and another, Terra-cotta Pink, together with the varieties Yellow Sport and Brightness, have proved resistant [18]. In Denmark, the majority of yellow and white varieties are found to be fairly resistant, while pink, red, and the variegated classes prove to be more susceptible [6]. In Germany, however, yellow varieties are very heavily attacked [20].

The experience of early autumn planting in California showed that the plants were able to escape infection so that they reached maturity before the rust became active in the spring [11]. Protecting the plants by spraying with Bordeaux or Burgundy mixture, or by applying sulphur dust, appears to be of doubtful value, some reporting better results than others [2, 7]. In general, copper-containing sprays have proved to be more effective than sulphur, and Bordeaux mixture with the addition of a spreader has, in some areas, given satisfactory results [12, 15]. In other places, Burgundy mixture (4 : 5 : 50) has given better control, and has the added advantage that it does not discolour the foliage as Bordeaux mixture is apt to do; in Britain, applications are advised on the following approximate dates: 1st to 2nd July; a second treatment on 15th to 16th July; a third, 29th to 30th July; a fourth, 12th to 13th August; and others at fortnightly intervals, a final spraying being given on 7th to 8th October [15].

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15. — 1936. *Ibid.* lxi, 64.
16. — 1937. *Ibid.* lxii, 214.
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Shot-hole of Antirrhinum, *Heteropatella antirrhini* Budd. & Wakef.

This common and destructive disease of cultivated antirrhinums was first recorded in Britain in 1917 [7] and was again observed in 1920 [4]. It was not known elsewhere in Europe until 1936, when report of its occurrence in Switzerland was probably the first record for the Continent [6].

During periods of cool moist weather in the summer, from July to September usually, the disease may be seen to break out on the lower leaves in the form of small pale-yellowish spots furnished with a well-defined margin. Owing to collapse of the affected tissues, the central portion of each spot gradually pales and dries out; sometimes a definite purplish margin may be developed around a spot, but this is not always present. The dried central area of the spot often drops out, giving a 'shot-hole' effect (Fig. 395), but actually this is not an invariable feature although it gives the disease its name [1]. Frequently the spots are quite large, irregular in shape, and sometimes the entire lamina may be involved. On young leaves and young stems, curling and deformity of growth may also be seen; later the leaves become dry and turn brown all over, the final effect giving the entire plant a scorched appearance. Though the above symptoms on the leaf



FIG. 395.—Shot-hole of *Antirrhinum* (*Heteropatella antirrhini*). Leaves of *A. majus* showing spots caused by the *Cercosporella* stage; upper row, early stages; lower row, subsequent shot-hole effect. On right, the pycnidial, *Heteropatella* stage on over-wintered dead stems (photos by Buddin & Wakefield, *Trans. Brit. Myc. Soc.*)

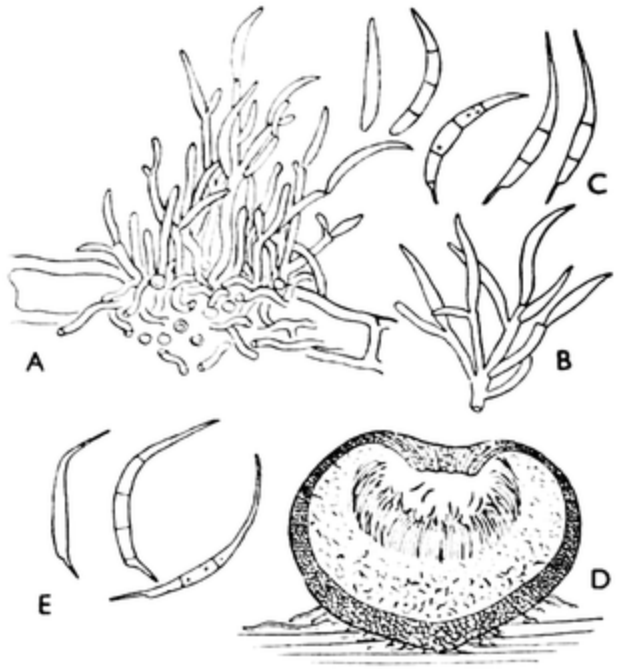


FIG. 396.—*Heteropatella antirrhini*. *A*, vertical section of an acervulus. *B*, branched conidiophore. *C*, conidia of various stages. *D*, vertical section of pycnidium. *E*, pycnospores (× 570) (after Buddin & Wakefield, *Trans. Brit. Myc. Soc.*)

are the most conspicuous, in severe infections the stems also develop lesions of a cankerous nature, in which case the development of the inflorescence is poor and distorted, the plant presenting a sickly appearance, and in very bad attacks entire plants may be lost outright [2].

There are two more or less distinct phases in the life-history of this disease, namely that of the actively parasitic leaf-spot stage caused by the Hyphomycete, *Pseudodiscosia* (*Cercosporella*) *antirrhini*, which is actually only the summer, conidial stage of *Heteropatella antirrhini* causing the second stage which develops pycnidia on the dead or decorticated stems (Fig. 395). These forms occur in the *Excipulaceae* group of the Sphaeropsidales (Fungi Imperfecti) [2, 3, 5]. The acervuli (Fig. 396 A) containing the summer spores are closely crowded on spots occurring on the leaves or the green parts of the stem and present a moist, waxy appearance, of a pale-pink colour. Erect, branched conidiophores (Fig. 396 B) break through the epidermis to produce the conidia which are of characteristic form (Fig. 396 C). They are narrowly obclavate and curved, from 1- to 3-septate; the apex is drawn out into an awl-like portion, and the basal part is furnished with a short tail or appendage. The conidia measure, in the cellular, septated part, from 20 to 35 by 3·5 to 4 μ, and the apical extension is from 20 to 25 μ long, while the 'tail' (which is formed largely after the spores have dropped off the conidiophores) measures about 10 μ long [2]. The pycnidia of the *Heteropatella*-stage (Fig. 396 D) occur on the wood after the cortex has fallen off; they are sparsely developed, brownish-black, globose-depressed, 500 to 600 μ in diameter; they are closed at first, later opening irregularly by a laciniated margin, exposing a pink spore bed in which the conidiophores are branched and very densely packed together. The pycnospores are falcate and, like the conidia, furnished with an apical extension and a basal appendage; they are hyaline, 2- to 3-septate, and measure from 25 to 30 or even 35 by 3 to 4 μ, the apical portion being from 20 to 25 μ long, the basal portion forming a short stipe or pedicel; the pycnospores are extruded from their pycnidia in pallid rosy globules [2, 5] (see p. 53).

The organism is of slow growth in culture; the optimum temperature is 18° C., no growth occurring at 25° C.; minute pinkish pustules of conidia are developed on a scanty white mycelium; later, solid, bun-shaped sclerotia-like bodies appear, externally black, and hyaline within, producing when sub-cultured the summer conidia in great profusion [2].

The pycnidia on the dead stems arise between cortex and wood, the fungus in this region bringing about a decortication of the stems. Pycnospores collected from the dead stems, inoculated into the living leaves produced the characteristic

leaf spots, thus repeating the parasitic phase of the disease [2]. The spores from the summer acervuli and those from the pycnidia germinate in the same way.

The fungus appears to survive from one season to the next in the form of black sclerotial bodies similar to those obtained in culture, and these in turn give rise to pycnidia, and presumably primary infections are brought about by pycnospores. The disease spreads on the plant from below upwards and attacks the leaves and young green shoots. The spores are probably dispersed from the fructifications in drops of rain or dew, and conveyed to the host by splashing of rain or by watering. Infection of the leaf is direct into the epidermis, not stomatal, and the mycelium occupies the intercellular spaces and cells of the mesophyll. In the stem lesions, the phloem and cambium of the vascular bundles are disorganised, and the cortical tissues are permeated with mycelium; the fungus may also be found in the phloem, cambium, and medullary rays. In preparation for sporulation, hyphal aggregations form in the epidermis, or between cuticle and epidermis, on both stem and leaves [4].

Some varieties of antirrhinum having red pigment in the leaves and stems appear to possess greater resistance than others; green-leaved, bedding varieties have proved to be very susceptible [1].

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Leaf Spot of Dahlia, *Entyloma dahliae* Syd.

Leaf spot or smut disease of dahlia was discovered on *Dahlia variabilis* in 1911 in Natal, South Africa [5, 6]. Since 1918 it has occurred rather frequently on the Continent [1, 2, 4], and appeared in Britain probably about 1927 [5]. By damaging the foliage, the disease greatly interferes with the normal development of the tubers [4].

The disease attacks the plants when they are more or less established, usually about flowering time, and is greatly encouraged in damp, shady positions, under which conditions it may assume epidemic proportions [5]. The lower leaves suffer most, while the upper may escape altogether [1]. Early symptoms consist of small flecks of a yellow-green colour (Fig. 397) traversing the whole thickness of the leaf, and as they increase in area (up to a quarter-, sometimes even three-quarter inch, in diameter) may remain circular or become angular in shape if they abut on the larger veins. The spots turn brown at the centre so that a ' halo ' effect is produced around each area. Sometimes the browned necrotic centre drops out, leaving a ' shot-hole ', but the halo still remains. New spots continue to be formed and may join together to develop into quite large areas of browned leaf surface; if infection is severe, entire leaves shrivel and die, and sometimes there is considerable loss of foliage [3]. The petioles may also be affected, the spots here

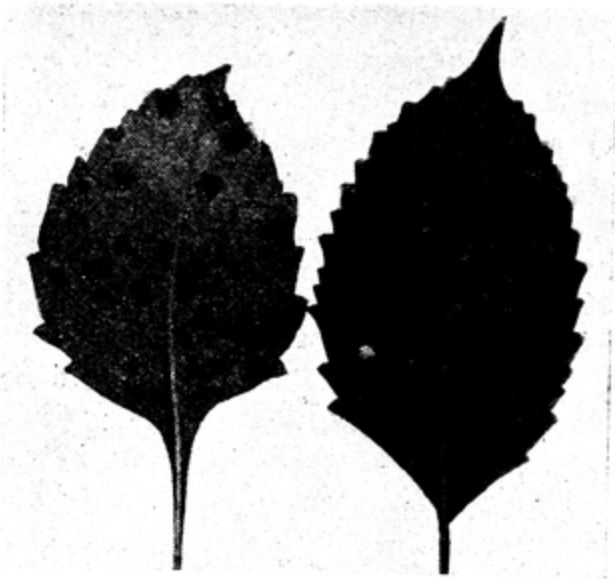


FIG. 397.—Smut of dahlia (*Entyloma dahliae*) (photo by Green, *J. Roy. Hort. Soc.*, by permission of the Editor)

being longer, of an oval shape, and not infrequently forming long stripes along the leaf stalks [4].

Leaf spot is caused by *Entyloma dahliae* [6], one of the smut fungi (*Tilletiaceae*). The fungus is confined to the leaves, occupying only the leaf spots and forming in the mesophyll of the affected parts a sparsely developed, slender mycelium. The life-history is very brief, for the hyphal cells early pass over to sporing, with the formation of chlamydospores. These are developed in the leaf tissue, in chains, and may be polygonal in shape from mutual pressure, or if they occur more or less isolated, somewhat spherical [2]. They are yellow to light brown, later becoming darker in colour and thick-walled, and measure, according to various authors, 11 to 17 μ (average, 13·5 μ) [3]; 12·5 by 16 μ [2]; or, 10 to 16 μ [4], in diameter. The spores germinate (at 22° C.) to produce a germ-tube (promycelium) which gives rise to a whorl of sub-branches, 5 or 6 in number; most of these branches grow out into thinner, thread-like hyphae which eventually produce sporidia. The latter are narrow, needle-like, straight or slightly curved, non-septate, and measure from 45 to 75 μ (average, 61 μ) by 2 μ [3].

Under natural conditions the spores are not released from the host but germinate in the leaf tissue, sending up the promycelia to the surface of the leaf to produce each about three or four sporidia. The chlamydospores probably remain dormant in leaf debris, or in the soil, until the next season, when under favourable conditions they are able to germinate and produce sporidia to start fresh infections. The disease is favoured by high humidities, late planting, and a deficiency of lime in the soil [4].

'Cactus' dahlias are observed to be more susceptible to leaf-spot disease than the 'pompon' types; and while no varieties of *Dahlia variabilis* are known to be immune, those derived from *Dahlia merckii* are resistant [5]. Spraying with Bordeaux mixture, 4 : 4 : 50, the first application being given early in August followed by a second, three days later, is recommended as a protection [3].

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Leaf Rot of Carnation, *Heteropatella valtellinensis* (Trav.) Wollenw.

Leaf rot of carnation, known in Germany since 1921 [2], was found in England in 1927 on plants growing in the open near Brighton, Sussex [3].

The disease is characterised by large, soft, greyish or brownish patches (Fig. 398), occurring near the base of the lamina in more or less transverse bands up to 1 cm. wide, which show on both surfaces of the leaf [4]. Sometimes the leaves may be affected from the tips down, for about a third of their length, these parts being almost white in dry weather, the colour changing to a grey brown under wet conditions. On leaves recently attacked, a zone of purple frequently occurs between the affected white and the healthy green part of the blade [3]. The diseased portions of the leaves soon become withered and broken up. Similar symptoms may occur on the stems, flower stalks, bracts, and sepals. If stock plants are badly diseased, difficulty may be found in getting suitable shoots for use as cuttings, since the leaves of the terminal rosette (' spike ') are also frequently attacked. The central leaves of the rosette may apparently become infected by contact and friction with the surrounding diseased leaves [3].



FIG. 398.—Leaf rot of carnation (*Heteropatella valtellinensis*). *A*, lesion at base of leaf, under side. *B*, lesion half-way up. *C*, basal lesion, upper side of leaf (photo by Ware, *Grdnrs'. Chron.*)

Leaf spot is caused by *Heteropatella valtellinensis*, a member of the Fungi Imperfecti [5]; this name now replaces *Pseudodiscosia dianthi* [1]. The spores are developed in two kinds of fructifications, acervuli and pycnidia (Fig. 399). The acervuli occur on the leaves and consist of small, raised pimples, originating beneath the cuticle ; they are disc-shaped, from 0·17 to 0·38 mm. in diameter, and break through the cuticle which then surrounds each pustule like a raised collar ; the conidia are abstricted from simple or branched conidiophores 3 to 4 μ wide. The conidia are oblong, mostly spindle-shaped, slightly curved like a sickle, thin-walled, colourless, mostly 2- to 3-septate, somewhat constricted at the septa, tapering at the upper end into a long awl-shaped process, while the opposite lower and broader end is provided with a shorter thread-like tail or appendage. The spores measure from 12 to 42 by 3·7 μ ; but without the apical process and tail, from 12 to 24 μ long ; both these prolongations to the spores may sometimes be absent. The pycnidia are later in appearing than the acervuli, on the lower, old or very rotten leaves. They are globose-depressed, from 0·25 to 0·5 mm. in diameter, and open irregularly ; the pycnospores are extruded in dirty-white globules, and are very much the same in shape as the acervular spores ; without the prolongations they measure from 20 to 25 by 4 to 6 μ ; with, from 20 to 30 μ in length [1]. In culture, conidia and pycnospores produce similar growths, consisting of somewhat pinkish, slimy masses of small, 1- to 2-septate

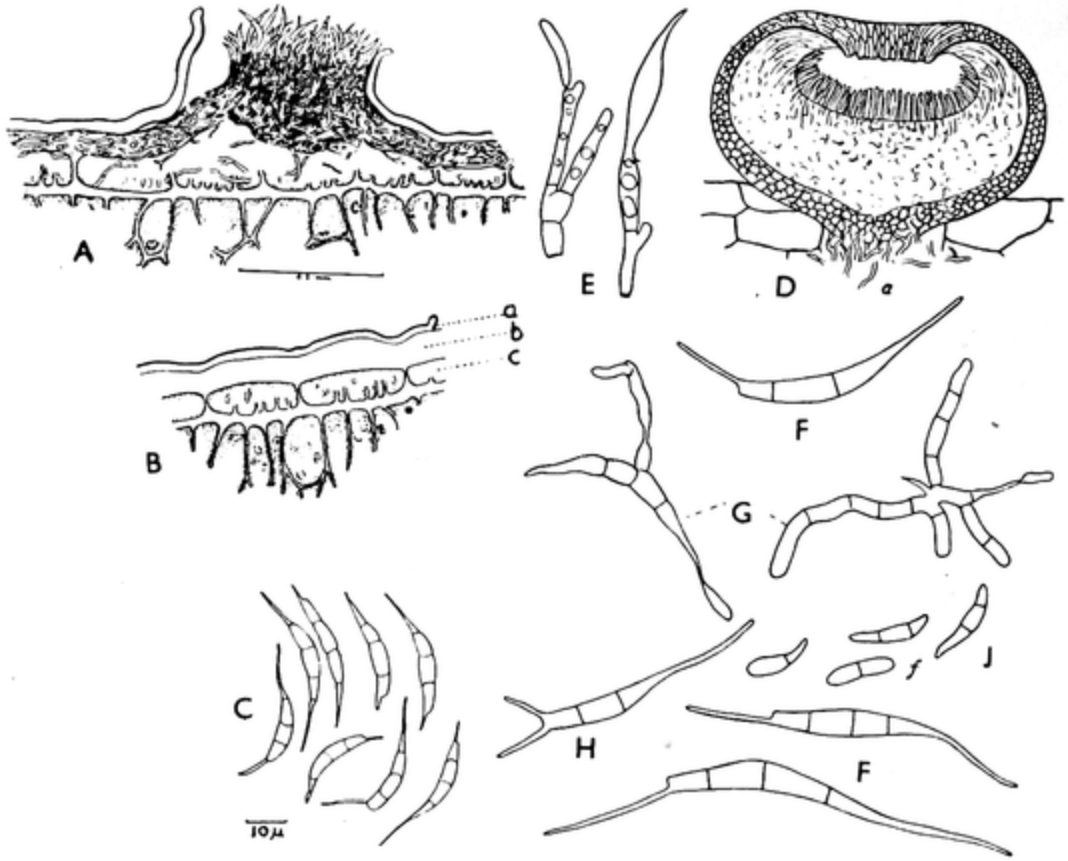


FIG. 399.—*Heteropatella valtellinensis*. *A*, section of acervulus showing the conidia breaking through the cuticle and thick-walled epidermis. *B*, section of healthy leaf showing cuticle '*a*' and thick-walled epidermis '*bc*' (×150). *C*, conidia showing the characteristic appendages (×424) (after Salmon & Ware, *Grdnrs'. Chron.*). *D*, vertical section of pycnidium. *E*, sporophores and young pycnospores. *F*, mature pycnospores. *G*, germination of the spores, with probable appressoria. *H*, an abnormal spore with two appendages. *J*, primary spores found in cultures. (*F–J*, ×640; *D–J*, after Buddin & Wakefield, *Trans. Brit. Myc. Soc.*)

spores, without appendages; no pycnidia were observed to develop in culture. The organism grows well about 20° C. and up to 25° C., but not at 30° C. (1).

The fungus probably survives from season to season in the decaying leaves; to what extent, if any, it may occupy the stock is not known, but infection does not usually proceed further from the leaves than the points at which they are attached to the stem (3).

The spores germinate easily from any of the constituent cells, to form a colourless, septated mycelium. The disease is favoured by dampness and low temperatures, and produces its worst effects towards the end of the summer, continuing during the autumn and practically throughout the winter, after which the new growth is liable to become infected. The fungus occupies the leaf from one side to the other, but chiefly the epidermal cells. Thereafter the hyphae collect at various places between the epidermis and cuticle to form a plectenchyma which, in turn, gives rise to the fructifications above mentioned.

Leaf rot may be kept in check by correct cultivation and avoidance of stagnant conditions. Where the disease has occurred, all affected parts of plants should be removed and destroyed. In some cases protection has been obtained by spraying

with Bordeaux mixture, but the results are not always encouraging, though rooted cuttings may be dipped in the mixture, or sprayed with it as a precaution before planting. Careful inspection of the cuttings should be made during the season and those showing signs of disease should be destroyed [3].

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Rose Mildew, *Sphaerotheca pannosa* (Wallr.) Lév.

Rose mildew is rarely absent from greenhouse or garden during the growing season. This very common disease of all kinds of roses was first discovered in 1819 in Germany [21], and is prevalent throughout Europe, America, and Australia [5, 7, 11, 19].

The disease first appears on young leaves and shoots as grey or white spots which soon spread until entire leaves, long stretches of stems, buds, and even the basal parts of prickles become more or less covered with the white powdery spores of the mildew (Fig. 400). In severe attacks growth may be checked, the plants remain stunted, the leaves curl, wither, and drop off, and buds smothered in mildew fail to open. Sepals and receptacles of flower buds are often covered with the fungus and sometimes the petals remain small and dry [10]. Mildew is rarely sufficiently serious to kill the plants, but its recurrence from season to season hinders normal development.

Rose mildew is caused by *Sphaerotheca pannosa* (*Erysiphaceae*) [9, 21], which is usually further designated as the variety *rosae*, to distinguish it from another form, *persicae*, which attacks almonds, apricots, and peaches [22]. *S. pannosa* is the species which causes the disease in Europe ; in the United States, *S. humuli* is also said to attack roses, but others have found only *S. pannosa* [10, 14]. From the superficial mycelium, which forms haustoria



FIG. 400.—Rose mildew (*Sphaerotheca pannosa*). The whitish conidial pustules on stem, leaves, and fruits

in the epidermis, erect conidiophores cut off from 6 to 8 elliptical or barrel-shaped conidia, 22·92 to 28·68 by 13·63 to 15·8 μ (average, 25·5 by 14·3 μ) [3]. The perithecia or cleistocarps are found on the leaves, petioles, stems, and around the bases of thorns, embedded in the ' pannose ' or felted patches of persistent mycelium, and are usually to be found mostly on the petiole and at the back of the midrib. They are globose to pyriform, 85 to 120 μ in diameter; appendages, few or absent, are short, tortuous, pale brown and septate; the single ascus is broadly oblong to globose, 88 to 115 μ, averaging 100 by 60 to 75 μ, 8-spored; ascospores measure from 20 to 27 by 12 to 15 μ [17].

After their separation from the host the conidia are very sensitive to environmental conditions [8, 10]; they are not adapted for long survival and lose their germinative capacity in less than 24 hours [6]. They are wind-borne and are, no doubt, dispersed for short distances, but appear not to be adapted to withstand extreme temperatures and humidities [10]. During mild winters conidia are probably enabled to survive in sheltered places in the open, or on protected plants in the greenhouse, and together with the ascospores from over-wintered cleistocarps, bring about fresh infections in the spring. There is no substantial evidence that mildew of roses is able to tide the winter in the form of a resting mycelium on the host, but is believed to survive sometimes during the winter out of doors, protected in the buds [14].

The conidia may germinate on either side of the young leaf, producing several germ-tubes, the whole giving the growing spore a cruciform appearance. The superficial mycelium forms dense white satiny patches of interwoven hyphae which are thick-walled (6 μ), refractive, and somewhat rigid. Later the colour becomes grey, dingy-buff, or rarely pale brown [4].

Mildew appears to break out under most diverse conditions of the environment. If warm muggy weather occurs in late spring or early summer, the disease may develop rapidly [20], but it varies considerably in intensity from season to season, and is often severe during a dry summer [7]. The conditions under which the conidia and ascospores germinate and attack the host are not fully known. These conditions are probably of the same order as have been elucidated in connection with the germination of the spores causing potato blight (p. 520) or those of white rust of crucifers (p. 637). (Conidia (sporangia) of potato blight, collected from leaves of very high water content, in a saturated atmosphere, failed to germinate immediately after removal, but did so when gathered after the water content of the host tissues had been reduced by exposure to dry air [13].) At 21° C., a temperature favourable to the germination of the conidia of rose mildew, the effect on germination was found to be determined largely by the moisture content of the air; immersed in or floating on water the conidia germinated poorly, but at 80 and 90 per cent. relative humidity, at the same temperature, germination reached a high proportion. A high degree of germination appears, therefore, to be a matter of relative humidity not only of the atmosphere [10] but also of proper humidity relations between the infective spores and the host cells attacked by them.

Good control over rose mildew may be obtained by spraying with copper oxychloride in oil (' Bouisol ' plus white-oil emulsion), used at a dilution of 1 oz. in 5 pints of water, applied towards the end of June; if severe, applications may

be repeated at 3 weeks' intervals [1, 2, 12, 15, 18]. Liver of sulphur and lime sulphur may also be used on roses in the open [2, 18], but is inclined to leave a deposit on the foliage and has a tendency to harden young growth under glass, especially if soap is used as an adhesive [2]. Under greenhouse conditions in New York wettable sulphurs afforded good control and proved superior to copper fungicides [23].

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Black Spot of Rose, *Diplocarpon rosae* Wolf

Black blotches familiar on the leaves of rose trees impair their vigour and in severe attacks bring about extensive defoliation of the bushes. While the spots occur but very rarely on the blooms themselves (Fig. 401, insets), yet their recurrence on the green leaves from season to season renders the provision of food reserves in the perennial tissues inadequate, in consequence of which affected trees make poor growth and the blossoms dwindle every year in number and quality. The disease attacks all varieties of roses, under glass and in the open, and while some varieties may, in one locality or another, or in different years, show some degree of resistance, no variety is, so far, known to be immune from it under all conditions [3, 7, 10]. The disease is very common in Europe and America; it occurred first in Italy in 1824, and appeared in the United States in 1887 [12]; it is prevalent also in Australia [9].

In Britain the disease breaks out usually about the middle of June, as small brownish-black spots on the upper, sometimes on the lower surface of the leaves, and the spots may either be distinctly round or more frequently furnished with a radiating fringe-like margin showing through under the cuticle (Fig 401 A). Individual spots often reach a diameter of 1 cm., and in heavy infections extensive areas of the leaves may be blackened and killed from the accumulation of fresh,

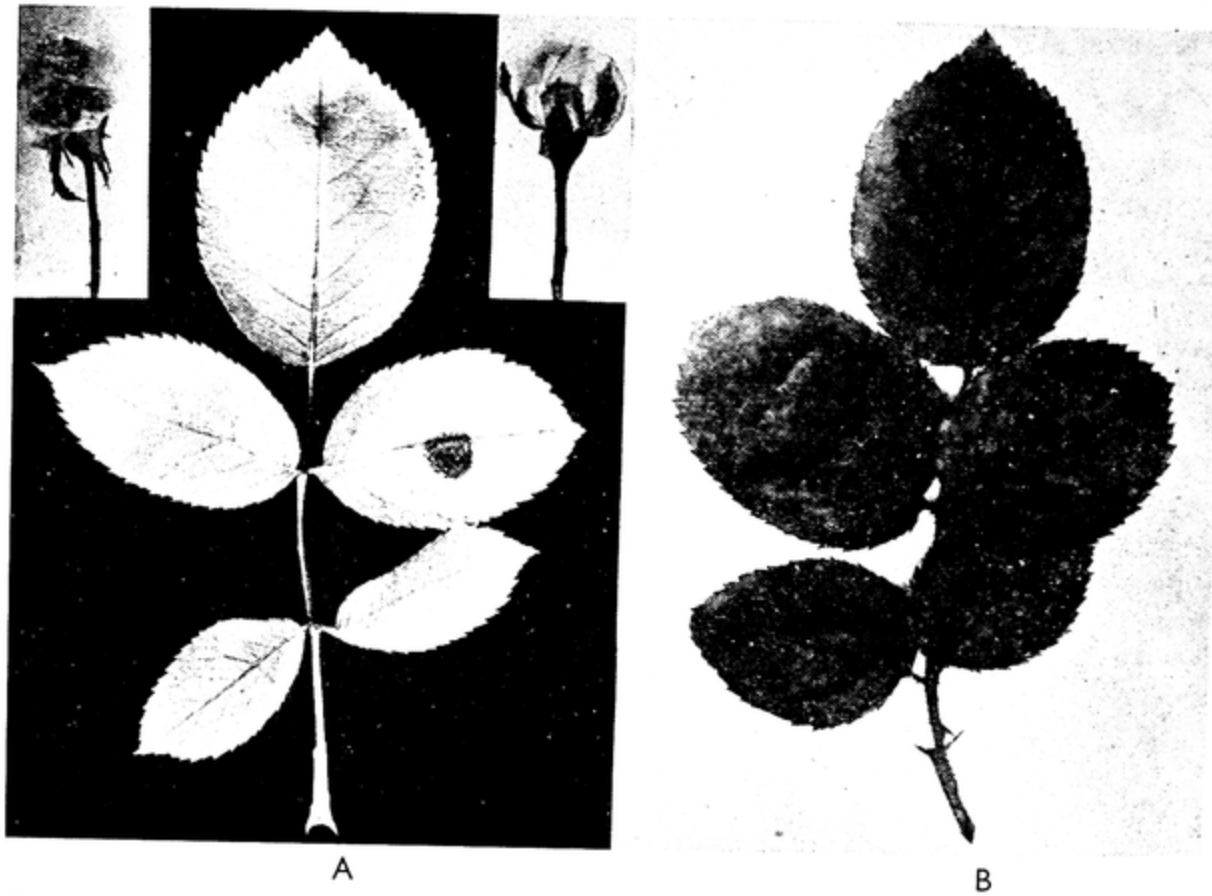


FIG. 401.—Black spot of rose (*Diplocarpon rosae*). *A*, the disease on a leaflet, and (insets) on the flowers. *B*, a severe, diffuse type of infection (photos by Green, *J. Roy. Hort. Soc.*)

secondary infections (Fig. 401 B), so that by about the middle of August affected bushes often look very bare from loss of leaves. In consequence of premature defoliation, buds which normally remain dormant until the following season are stimulated to grow during the current season to make good the loss and, by opening late, cause a considerable drain on the already depleted reserves of the plant. Such buds are often killed by frost, so that the weakened trees fail entirely to flower in the following season [9, 15]. Tiny lesions, easily overlooked, also occur on the stems and, when sporulating, they serve to propagate the disease [1, 11].

Black-spot disease of roses is caused by the Ascomycete *Diplocarpon rosae* (Perisporiales); the ascigerous stage was discovered by Wolf [15] in 1910 in the United States, on over-wintered leaves, but it is not common (it has not been found in Britain) and the fungus is more familiar under the old name *Actinonema rosae* given to the parasitic stage, which is mainly responsible for spreading the disease [13]. It grows slowly and loses pathogenicity in pure culture [5].

The fungus has three types of reproductive structures: the summer acervuli containing conidia of the *Actinonema* stage, on leaves and young shoots; spermagonia, on older leaves, in association with the ascigerous stage; and the cleistocarpic *Diplocarpon* stage, on over-wintered leaves. All three fructifications are developed between cuticle and epidermis and appear as tiny raised specks within the blackened areas, especially towards the fibrillar margin of the spots. The conidia are hyaline, oval to elliptical, bicellular, and constricted at the septum, the cells

sometimes breaking apart at the septum to function as separate spores; typical conidia measure from 18 to 25 by 5 to 6 μ [15]. The tiny spermagonia contain myriads of bacilliform spermatia 2 to 3 μ long, and sometimes typical bicellular conidia in addition to spermatia [4]. Cleistocarps, on dead leaves, sunk deep into the tissues, are spherical to disciform, 100 to 250 μ in diameter; the asci are oblong or sub-clavate, 70 to 80 by 15 μ, and are interspersed with capitate, septated paraphyses; the ascospores closely approximate the conidia in dimensions, from 20 to 25 by 5 to 6 μ, and vary considerably in shape, but are hyaline and bicellular like the conidia; the cleistocarps open at the top in a stellate manner and the ascospores are exuded in viscid masses [15]. Fructifications resembling the cleistocarps, and arising, like them, deep within the leaf tissues, but containing conidia in place of, and identical in dimensions with, the ascospores and discharged to the surface in the same way, have also been found [4]. These differences in the contents of the deep-seated fructifications, observed by different authors [4, 5, 15], are explained on the hypothesis that the true cleistocarps are developed following an act of fertilisation in which the spermatia take part, and that in the absence of spermatia, or failure to effect fertilisation, cleistocarpic initials pass over to the production of pycnidia [4].

The mode of survival of the fungus and provision for future infections depend largely on locality and environment, and while cleistocarps containing ascospores may be the means of carrying the fungus through the winter in some areas, in Britain and other places where cleistocarps have not been found, infections in all probability are provided by conidia which develop on infected leaves that persist on the trees or on the ground [5] throughout the winter. But another and important source of infection, discovered in England, is the development late in the season of conidial acervuli on the young wood [1]. Such acervuli, with unbroken cuticle and conidia below, have been found during January and February; they remain thus protected throughout the winter until the spring, and the spores are dispersed at the time when the buds are breaking into leaf [6]; but some varieties of roses appear to be more susceptible than others to the formation of these over-wintering lesions on the young wood. It has been suggested that the disease may not always be carried over the winter on any part of the rose tree itself, and that possibly an intermediary plant may exist, but so far no such alternative host has been discovered [3]. In this connection it is significant to note, however, that in certain areas where the disease is not common it does not appear to spread, and even when introduced into the neighbourhood soon apparently dies out, possibly from the absence of a carrier host [1].

Both conidia and ascospores are probably disseminated when the trees are splashed with rain, and perhaps by insects, for the viscid nature of the spore exudate, in both cases, precludes dispersal by wind. First signs of infection following inoculation of the leaves with either conidia or ascospores appear in about 10 days, and acervuli containing conidia are developed 8 days later, but growth is much faster on young leaves than old. Penetration, direct through the cuticle, is favoured by temperatures from 20° to 26·5° C., and a humid atmosphere of 92 to 97 per cent. saturation. The germ-tubes form appressoria, and after passing between the epidermal cells the infection hyphae establish haustoria in

the epidermis before proceeding to ramify between the epidermis and the cuticle. Soon an appreciable patch of the leaf epidermis turns brown from the death of its cells and later turns black to form the familiar blotch (2, 15).

When acervuli or spermagonia are about to develop on the blackened areas, the spots become dotted over with small blisters due to the formation of conidia or spermatia from thin stromata of mycelium developed between cuticle and epidermis. In pustules, destined for over-wintering, which develop on the young wood late in the season, the mycelium is more or less confined to the cortex, and the stromata, which are somewhat thicker in this region than in the leaf spots, develop in the same way, between epidermis and cuticle, to form short conidiophores from which are developed conidia of the same type as those formed by the leaf acervuli, and they are liberated in the spring to collect in viscid masses on the surface of the twigs.

Resistance to black spot appears to be a purely relative character, depending largely on locality and environment. In general, varieties of roses with thin leaves, e.g. Hybrid Juliet, a most susceptible variety, are very subject to black spot, while thicker and tougher-leaved kinds are frequently found to be more resistant. Wichuraiana and Polyanthus roses withstand the disease well in England, but exhibit considerable seasonal variation in the severity of symptoms. In general, Tea, Hybrid Tea, and Hybrid Perpetual roses differ greatly in resistant powers ; all bush roses are more or less liable, but in the United States, Wichuraiana types, Rugosa Hybrids, Pink Princess, and Moss Roses are rarely attacked (11 b, 14).

The manner of dissemination of the spores by splashing of water indicates that great care should be exercised when the trees are watered or syringed. To prevent carrying the fungus over from old leaves and wood prunings, it is advisable to collect all such material for destruction by burning. As an additional precaution it is often the custom to remove a layer of the soil from infected beds in early winter, to ensure not only the complete removal of infected plant debris, but of any other form of the fungus that, so far as is known, may be capable of survival in the soil (13). The disease can be kept well under control by fungicides. Bordeaux mixture, 4 : 4 : 50, with saponin, 1 oz. per 50 gallons, as an adhesive, applied towards the end of June and repeated frequently at fortnightly intervals, gives good protection. An alternative method, which does not discolour the foliage, is to dust the leaves with a sulphur-copper dust, or a specially prepared sulphur, finely divided and dyed green (' pomogreen ') in imitation of the natural foliage ; used with the addition of 10 per cent. lead arsenate, good results are obtained, the plants blossom freely and defoliation is checked (7, 8, 11 a). A combined treatment, controlling ' red spider ' as well as leaf blotch, consists of spraying with ' selocide ' mixed with pyrethrum oil as a spreader, two or three applications being given at intervals of 7 or 10 days (10). Potassic manures appeared to reduce the amount of leaf infection, but lime seemed to have no effect (14 a).

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Brown Canker of Rose, *Cryptosporella umbrina* (Jenk.) Jenk. & Wehmeyer

Brown canker is prevalent on Tea, Hybrid Tea, and Hybrid Perpetual roses, in various parts of the United States [1, 2], and was recorded for the first time in Britain in 1931 to cause a wilting and die-back of roses [5].

The disease attacks the stems, leaves, and flowers. From June to August small lesions may be seen on the stems, at first purple, then turning white at the centre, and ultimately forming conspicuous buff-coloured cankers with a purple margin. The central, lighter part of the lesion becomes speckled with the ostioles of numerous pycnidia, and later, usually in circles surrounding the pycnidia, with the protruding beaks of perithecia. On the leaves, purplish spots arise which may remain so without further change or, like the cankers on the stem, the centre may turn white or a cinnamon-buff colour, and develop pycnidia; leaf stalks and stipules may also become similarly spotted. On the petals, the spots are of a cinnamon-buff colour and pycnidia may form on them as on the leaves, in well-defined concentric circles, and these fructifications may be seen, too, in abundance on dead blossom and fruits [2].

The causal fungus is *Cryptosporella umbrina*, a member of the Sphaeriales [3, 4]. The pycnidia are sub-epidermal, sub-globose, thick-walled, irregularly ostiolate, unilocular or chambered, 200 to 300 μ in diameter; pycnospores are straight or slightly curved hyaline, 4·8 to 11·2 by 2 to 3·2 μ [1]. The perithecia, 100 to 290 μ in diameter, concentrically arranged around the pycnidia, are immersed, globose, the beak 150 to 195 μ long, scarcely projecting above the epidermis; asci clavate, 30 to 50 by 6·4 to 8 μ; no paraphyses; ascospores ellipsoid, hyaline or light-olivaceous, unicellular, measure from 8 to 11·2 by 3·2 to 4 μ [1, 3].

The mode of over-wintering of the fungus is not clearly known, but since perithecia may be found practically all the year round, the mycelium is probably perennial in the cankered wood. Primary infections in the early spring are due to ascospores which are dispersed by wind, splashing rain, insects, or conveyed on garden implements [1]. Infection probably occurs only through wounds in the bark, and buds are also liable to infection, but only if injured. Attacks from both pycnospores and ascospores require a high degree of humidity for successful infection.

During the dormant period and again early in the spring, a spraying with

Bordeaux mixture may be given as a protection. All cankered parts should be destroyed. Careful watch should be made in the spring for stems showing infection, and these should be removed as they are the sources of secondary infections [1].

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Stem and Graft Canker of Rose, *Leptosphaeria coniothyrium* (Fuckel) Sacc.

Leptosphaeria coniothyrium, besides causing cankers on stems and stocks of roses [17, 18, 19], attacks apple [13], blackberry [3], and willow [12], and has already been described in this book in association with a blight of raspberry canes (p. 804). Another affection of roses described in Europe [9, 10] and the United States [6, 7, 14, 22] as brand canker, caused by a related fungus known only in its pycnidial form, *Coniothyrium wernsdorffiae* [9, 10], shows symptoms very similar to those of stem and graft canker, but is apparently confined to roses and does not occur in Britain [20, 22]. Some authors [5, 8], however, do not recognise these diseases as being separate nor the organisms causing them as distinct species, but recent work [21, 22] (despite the admittedly wide polymorphism of *Coniothyrium fuckelii*, the pycnidial form of *L. coniothyrium*) maintains a separate identity for brand canker caused by *C. wernsdorffiae*.



FIG. 402.—Rose canker. *A*, graft canker (*Leptosphaeria rosarum*. *B*, *C*, stem canker (*Leptosphaeria coniothyrium*) (photos by Green, *J. Roy. Hort. Soc.*)

Young lesions of stem canker (Fig. 402) appear on the one-year-old green wood as more or less confluent red-brown to purplish spots on the bark, in any parts, irrespective of the position of branches, thorns, or buds. The pycnidial fructifications in these regions ultimately break through to liberate sooty masses of spores, and with the progressive rupture of the bark deep fissures or

cankers develop on the shoots [5]. In the case of graft cankers (Fig. 402 A), the colour is darker brown than that of stem cankers, varying from cinnamon brown at the centre to hazel or auburn at the margin, and here, again, the sooty spore masses break through from the erumpent pustules, and with repeated attempts at healing of the cankers by callus formation, the affected parts gradually become girdled with large warts and finally die [16]. Graft canker of roses (long associated with another species of *Coniothyrium*, *C. rosarum* [2], now believed to be synonymous with *C. fuckelii*) results from various causes, such as an infected condition of the stock before grafting or budding, or the presence of spores at points where grafting has been imperfectly performed, or through the separation of the bud by drying, or by infection of the top of the stock left as a support for the growing scion [1].

The pycnidial stage, *C. fuckelii*, is much more prevalent than the perfect ascigerous stage, *L. coniothyrium*. The pycnidia are 180 to 200 μ in diameter, ostiolate ; conidiophores are barely perceptible and the pycnospores appear to arise by budding of the cells lining the loculus ; the globose or slightly elliptical, olivaceous spores are exuded in gelatinous masses, and measure from 2·4 to 5 by 2 to 3·5 μ. The organism in culture is polymorphic, giving first a yeast-like form of short chains or glomerules composed of simple or septate cells, and these, in transfers, yield a mycelium which gives rise to the characteristic pycnidia [15]. The perithecia of *L. coniothyrium* have already been described (p. 804). The minimum temperature for growth is 1°, the optimum between 25° and 26°, and the maximum between 32° and 35° C.[22]. The production of fructifications appears to depend on the strains used and the age of the culture, and there are apparently some strains of *C. fuckelii* which sporulate abundantly while others yield only sterile mycelium [22].

Infections by spores from cankers, dispersed in splashing rain-drops, take place through wounds caused possibly through broken prickles, or by friction between the shoots, or by hailstones [16], or insects. The mycelium is apparently capable of wintering on stubs left on the plants after pruning (pycnidia have been found to develop close to the pruned cuts), or in pieces of diseased stem on the ground [21].

Spraying of the bushes is of little avail against canker. All diseased parts should be cut away at the base and burned. Graft-work should be done carefully, the scion being cut so as to avoid leaving a stub above the node and the joint covered over with wax [1, 11].

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Rose Rust, *Phragmidium mucronatum* (Pers.) Schlecht

Rust of rose trees, caused by *Phragmidium mucronatum*, is common everywhere on wild and cultivated roses. In Britain it appears to be somewhat localised, but in some areas has proved to be rather troublesome [1], especially on stocks grown for grafting, and when severe there is a decided check to growth as a result of premature defoliation [4, 8, 10].

Rose rust pursues its complete life-history on the one host, and is therefore autoecious. Uredosori and teleutosori develop on the leaves, and aecidiospores, in caeomata, may be found on buds, stems, leaves, petioles, and fruits, but occur most commonly on the stems. Infected branches may become hypertrophied and deformed, and on the stocks histoid galls (p. 194) may develop [11].

The uredosori may first be seen in June on the under side of the leaves as small yellow pustules, somewhat localised, and surrounded by a circle of clavate hairs; the uredospores are ovate or ellipsoid, yellow, echinulate, 21 to 28 by 14 to 20 μ, with numerous germ-pores.

The teleutospores arise usually in the same sori as uredospores in late summer or autumn; they are black, bulbous, and furnished with long stalks, 70 to 80 by 15 μ, septated into 6 to 8, rarely 5 or 9 cells, the terminal cell being furnished with a pointed papilla; the spores measure from 65 to 120 by 30 to 45 μ. The teleutospores will only germinate after a period of rest and freezing, and most attempts to produce infection with them have failed [2, 3, 10].

The caeomata may occur singly or joined together, and break through the covering epidermis, exposing a sorus of bright-orange aecidospores in short chains; the spores are verrucose, orange-yellow, 24 to 28 by 18 to 21 μ; spermagonia are produced on the upper side of the leaf.

Though experiments to bring about infection with teleutospores have failed in England, yet these spores have been seen adhering to the stems of rose bushes and may persist in cracks in the bark or between bud-scales, and it is not improbable that under certain undetermined conditions they may cause infection [10], as they have recently been observed to do in California, the sporidia producing caeomatal lesions on both leaves and young stems, while uredospores caused only leaf infections [2a]. But aecidiospores and uredospores infect readily if the leaves are thoroughly wetted; the latter do not survive long in localities of cold winters or high summer temperatures, but survive to cause fresh infections in places where the temperature is uniformly favourable, with adequate rainfall for the spread of infection [2b]. Strong light apparently depresses the rate of uredospore germination [2c]. Caeomata have been found to develop in successive years on the same branch lesion, showing that the mycelium had survived within the host for at least two years. Aecidiospores continue to be formed

for some weeks and caeomata have been found as early as March, a further proof of their development from a perennating mycelium. There is some evidence, too, of bud infection, no doubt by transmission from the stem, but infection is believed not to be systemic and there is no proof of direct migration of the fungus from an infected stock to the graft [10].

Temperatures between 60° and 70° F., with sustained moist conditions, such as heavy dew formation, are necessary for infection with uredospores.

There are apparently no morphological differences between uredospores isolated from different sources, but some constant variations have been observed in different collections of teleutospores, some strains showing 6-celled spores in abundance while others showed 7- or 8-celled types, and some strains of the fungus are apparently less susceptible to fungicidal action than others [1, 2, 9].

Since the fungus is carried over mainly through a perennating mycelium producing caeomata, all stems showing these fructifications should be cut out as soon as observed [10] and all leaves and cuttings should be collected and destroyed. Good control may be obtained by spraying the bushes in spring and autumn with lime sulphur, or 2 per cent. Bordeaux mixture, or 2 to 8 per cent. carbolineum, and periodical summer treatments may be given with 1 per cent. Bordeaux or Burgundy mixture [7]. Colloidal sulphur, with soft soap, also gives good control (1 fluid oz. ' sulsol ' and $\frac{1}{2}$ oz. soap per gallon) [6]. Liming and applications of potassium sulphate and magnesia to the soil are reported to afford increased resistance to the rust [5].

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White Mould of Narcissus, *Ramularia vallisumbrosae* Cav.

This disease is common on various kinds of narcissus cultivated in the open in districts of high spring rainfall and humidity, such as the Isles of Scilly, Cornwall, and Devon. It has been closely investigated by Gregory, in south-west England, where it has been reported to assume epidemic proportions [1, 6-8]. The disease, first described in 1899 in Italy [3], was observed in England in 1906 [4], and recognised in 1912 as being the same as the Italian disease caused by *Ramularia vallisumbrosae* [3]. It also occurs in France [2] and a severe outbreak of the trouble in the United States in 1930 was reported in Oregon [9].

Considerable losses ensue when the blooms are marred by blemishes of spore infections, and flower stalks of the variety *Narcissus poeticus* are sometimes rotted

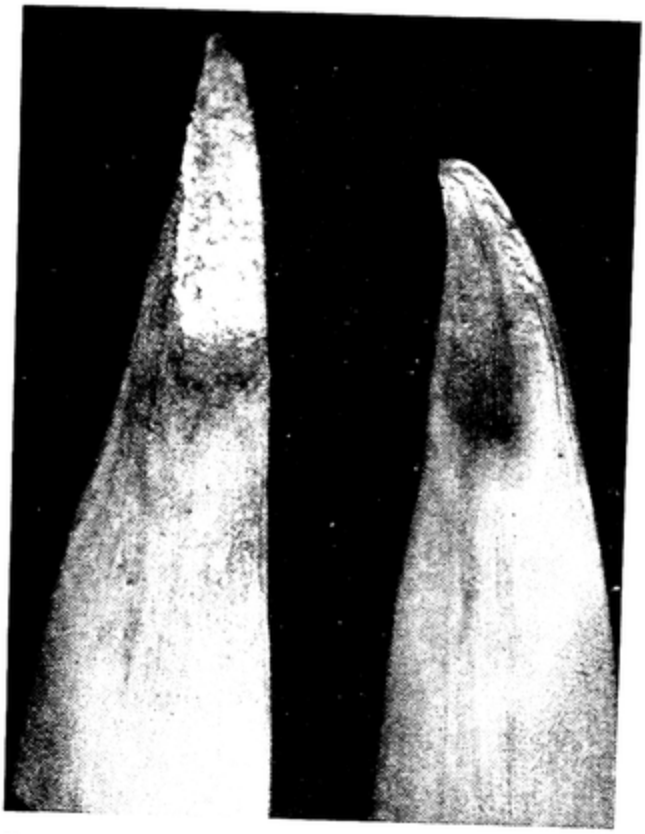


FIG. 403.—White tip of narcissus (*Ramularia vallisumbrosae*) (photo by Gregory, *Trans. Brit. Myc. Soc.*)

by this disease. By attacking the foliage leaves and causing premature destruction of leaf tissues, the fungus prevents the developing bulbs from obtaining adequate provision of food reserves, so that only weakly bulbs are produced.

White mould, like other diseases affecting narcissus, is not usually severe in the first year of planting, but becomes aggressive in the second and third flowering seasons. First signs of the mould are manifest when the young leaves are about three or four inches above the ground, the two protecting sheathing leaves being unaffected. At this time small, sunken grey streaks may be seen near the tips of the leaves, facing the outside. As the streaks enlarge, yellowish-brown necrotic spots appear on them and these become covered with a dense layer of buff to chalky-white conidia arising in tufts or sporodochia (Fig. 403). In some varieties of the host the affected area is surrounded by a narrow dark-green band, and outside this again a straw-yellow border, which fades imperceptibly into the natural green of the leaf, may be detected. When secondary infections develop, the parts of the lamina lower down, as well as other leaves and flower stalks, also become affected under damp conditions. With the advance of the season, when the leaves are beginning to wither and spore formation is about over, small black sclerotia develop in the older parts of the defunct sporing lesions. The sclerotia are especially numerous just beyond the limits of the old sporing areas and somewhat more sparsely developed over the remainder of the withered leaf.

White mould disease, confined to narcissus, is caused by a member of the Fungi Imperfecti and has been variously named *Ramularia vallisumbrosae*, *R. narcissi*, and *Cercosporella narcissi*, the last name being probably a synonym. The nature and mode of development of the sclerotial bodies in the life-history of this organism would seem to suggest similarity with perithecial formation, but so far no ascigerous fructification has been found. The fungus is highly polymorphic (see Chapter I, p. 53), and during its parasitic career produces no fewer than three kinds of conidia, namely scolecospores, phragmospores, and amerospores, corresponding to the '*form genera*', *Ramularia*, *Cercosporella*, and *Ovularia* (Fig. 404). No real demarcation, however, can be recognised between one spore type and another [7].

Penetration of the leaf by any of the conidial forms is stomatal. The infection hypha, which swells slightly in the sub-stomatal space, develops a branching mycelium which extends between the cells of the mesophyll from one side of the

leaf to the other, without, however, penetrating the vascular system. The yellow zone of discoloration seen on the leaf surface following infection, occurs somewhat beyond the limits of the internal mycelium, but it is not long before the palisade cells contract and collapse, and near the edge of the affected area the radial walls of the epidermal cells also give way, collapsing on the disintegrated mesophyll below. Only in the collapsed epidermal cells does the fungus become intracellular and within these cells the sclerotia develop later. The mycelium in other parts of the leaf aggregates chiefly in the form of plates between the epidermis and the mesophyll, and it is from these plates that individual hyphae arise, which by insinuating themselves between epidermal cells come into contact with the cuticle, under which they finally develop sporodochia.

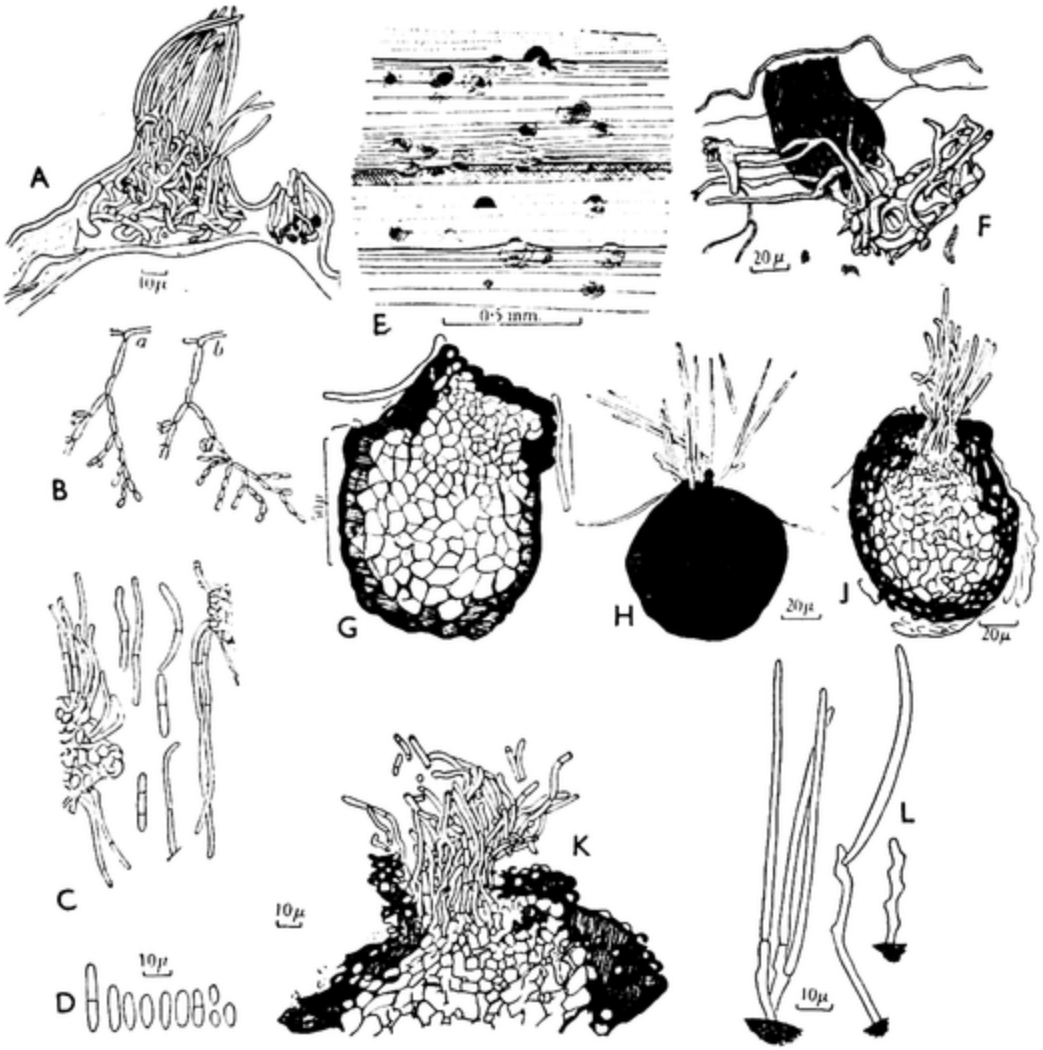


FIG. 404.—*Ramularia vallisumbrosae*. *A*, section of sporodochium developing beneath cuticle covering the guard cells of a stoma on lower surface of leaf. *B*, development of amerospores in a hanging-drop, *b*, 24 hours after *a*; *C*, phragmospores, and possibly, scolecospores. *D*, conidia from leaf spots. *E*, immersed sclerotia beneath leaf cuticle. *F*, section of leaf showing dark, thickened hyphae connecting sclerotium with rest of mycelium in leaf. *G*, section of sclerotium in dormant condition of aestivation. *H*, scolecospores produced on conidiophores in neck of a sprouting sclerotium. *J*, sclerotium after 36 hours' incubation. *K*, after 48 hours' incubation, showing mass of conidiophores and conidia. *L*, geniculate conidiophores and conidia from sprouting sclerotia (after Gregory, *Trans. Brit. Myc. Soc.*)

A sporodochium (Fig. 404 A) begins as a single hypha under the cuticle. After spreading laterally between the cuticle and the outer cellulose wall of the epidermis, the hypha produces a number of swollen vertical branches which lift the cuticle from the epidermis, the process of separation being entirely a mechanical one. Rupture of the cuticle is finally effected by the elongation of the vertical hyphae, and by further growth of the hyphae at the base of the sporodochium a plectenchyma is formed. Neighbouring sporodochia may link up by extension of marginal growth and in this way large areas of the leaf may become covered with spores.

As above stated, the spores that may develop on a sporodochium are very variable, even on different parts of the same leaf, and no precise dimensions can be given for any of them. The factors which appear to favour the production of one type of spore in preference to another are not known, but the observation that scolecospores are almost the only kind to develop in the early months of the year may possibly be accounted for by the effect of lower temperature or differences in nutrition ; relative humidity may also be a controlling factor.

The sporodochia arise mostly on the primary lesions. The closely packed, vertically arranged hyphae of the young sporodochium constitute the simple conidiophores whose distal parts are transformed into conidia. When a sporogenous hypha has reached a length of 40 to 100 μ, the distal part is cut off from the basal portion and the terminal portion is detached as a scolecospore. The scolecospores measure from 40 to 95 μ in length, being from 2·5 to 3·5 μ wide at the base, but often tapering to 2 μ across at the apex ; they are from 3- to 7-septate (Fig. 404 H). The scolecospores appear to be the normal and most highly developed of the conidial types found on infected leaves in the field. The phragmospores (Fig. 404 C) similarly cut off from the tips of the hyphae projecting from the sporodochium are shorter than the scolecospores, so that after these spores have been dispersed the conidiophores project somewhat beyond the leaf surface and often radiate at various angles. Phragmospores may occasionally be produced in chains, a second conidium being formed from the apex of the first, a feature which has not been observed with scolecospore formation. According probably to the available food the same conidiophore which has at one stage produced scolecospores may at another time develop phragmospores. Amerospores (Fig. 404 B) are the type least often observed and appear to be developed only under exceptionally moist conditions ; they are non-septate conidia, measuring from 3 to 10 by 3 to 4 μ, and may develop in acropetal succession so that chains of conidia of an *Ovularia*-type are formed.

The sclerotia (Fig. 404 F–L), first discovered in 1926 [1], follow the sporodochia in the same lesions, during May and June, as the leaves wither. A sclerotium begins as a small knot of hyphae within an epidermal cell, and soon develops into a mass of pseudoparenchyma rich in fat reserves ; the peripheral layers eventually turn black, forming a carbonaceous rind. By the development of a neck these sclerotial bodies bear some resemblance to pycnidia or perithecia, and within the neck region a brush of hairs which develops in the spring gives rise to conidiophores ; this is the stage reached by the sclerotia in June and is the condition whereby the fungus passes through the summer months, the dormant period. The sclerotia become rejuvenated, to produce conidia, during the middle of February, the period coinciding with the appearance of the first leaves above ground, and at this time primary infections take place. The conidia which develop within

the neck of the germinating sclerotia are of the scolecospore kind, but in the laboratory phragmospores and amerospores may be produced by them, and may even predominate. The organism is carried over from one season to the next by means of sclerotia on old leaves lying on the soil, the conidia produced by them early in the year serving to infect the new leaves; the bulbs do not appear to become affected.

Inoculations performed with scolecospores on uninjured leaf tips in a moist atmosphere produced the characteristic white mould lesions in three to four weeks, these lesions being similar to those which resulted from infection with phragmospores; the conidia on all the lesions were of the *Ramularia* (phragmospore and amerospore) type. Repeated experiments showed that the various forms of conidia from sporodochia and sclerotia all come within the life cycle of this one fungus, *R. vallisumbrosae*.

White mould of narcissus may be kept under almost complete control by the application of 4 : 4 : 40, or 4 : 3 : 40 Bordeaux mixture, first treatment being given when the shoots are 4 to 6 inches high, with further applications at monthly intervals. Since the decayed leaves harbour the resting sclerotia, these should be raked off and burned [7, 8].

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Shanking of Tulip, *Phytophthora cryptogea* Pethybr. & Laff. & *Phytophthora erythroseptica* Pethybr.

Shanking disease of tulips, so called because it attacks the basal part of the flower stalk, was first discovered in Britain in 1928, and has not been recorded, so far, outside this country [24].

It occurs on forced, glasshouse tulips and does not ordinarily attack the bulbs in the open, probably because of the lower temperatures. Enormous losses are often incurred by commercial growers and entire houses of several thousand bulbs may be ruined by this disease.

Suspicion of the trouble is first aroused when the growing plants, to all appearances a healthy crop, are slow in producing blossom. In other cases, when flower buds have appeared, they either fail to open or soon perish after partially opening, the stalks drooping over because of contraction or wrinkling of the tissues at the base (Fig. 405). But ' shanking ' is not the only symptom of the trouble and closer examination of the beds may reveal gaps or misses indicating complete failure of the bulbs to grow, or other bulbs may produce only short young shoots which can be twisted out readily from the bulbs because of the rot at the base. Diseased plants also frequently show a tinge of red colour at the tips of some of the leaves,



A B

FIG. 405.—Shanking of tulip (*Phytophthora cryptogea* and *Phytophthora erythroseptica*.) *A*, tulip with shrivelled flower and poorly developed, fading foliage; the bulb is cut to show the rotting of the basal plate. *B*, bulb planted in contaminated soil, showing progress of the rot from the roots into the flowering shoot, but the old bulb scales are not attacked (photos by Buddin, *Ann. App. Biol.*)

followed by the usual symptoms of shanking. When affected bulbs are lifted most of the roots are yellow and dead from the tips up, and with the access of secondary organisms the dead root stumps turn a dark-brown colour. Cutting the bulb in half will show much discoloration in the flattened disc plate as well as in the base of the flower stalk, and sometimes, but not commonly, the discoloration may extend into the bases of the scale leaves. But quite often, though the base of the flowering shoot may be completely rotted, the scales escape the rot entirely, and such bulbs may actually yield a number of bulbils which may or may not become infected from the parent bulb [1].

Shanking is caused by two species of *Phytophthora*, namely *P. cryptogea* (already described in connection with a foot rot of tomato, p. 664) and *P. erythroseptica* (also described as causing pink rot of potato, p. 509) [5]. These parasites may occur singly or together and it is difficult to state which is the commoner agent causing this disease. Sometimes a species of *Pythium* is also implicated [3], but it does not appear to play so important a part as either of the Phytophthoras.

The resting oospores of both these parasites are produced in the dead tissues of the host and upon disintegration of the rotted bulbs the spores are liberated into the soil. Presumably, new infections take place only from this quarter. When cultures of the organisms are mixed with soil the disease is produced on planted bulbs, and there is abundant evidence that both fungi can exist in a virulent condition in the soil from one season to the next [2]. In the case of tomatoes attacked by *P. cryptogea*, oospores left in the soil will attack tulips grown in the same soil. There is no evidence that the planting of infected bulbs is responsible for widespread infection in ordinary glasshouse practice; it was found that from the planting of one or more inoculated bulbs in the midst of healthy ones the rate of

spread was comparatively slow, but the trouble may be carried over in any way that ensures retention of contaminated soil, such as using dirty boxes in which clods of soil had collected from diseased bulbs.

Infections by the organisms, acting separately or together, follow much on the same lines, and presumably from the germination of the resting oospores in the soil the first parts to be attacked are the roots. Starting at the root tips, infection spreads into the stem disc and up the flowering stem, but extension into the neck is only for about a distance of an inch or so, and only at an advanced stage of the disease, if at all, do the scale leaves become infected. Entry into the bulb may also be made through wounds but there is no evidence that direct infection can occur through the shoot above the neck of the bulb. A temperature of 25° to 27·5° C. is most favourable to the progress of the disease.

Most of the popular varieties of tulips are susceptible to the disease, William Pitt and William Copeland being particularly so, followed closely by Rose Copeland and Bartigon ; Pride of Haarlem and Madam Krelage are less susceptible, while Clara Butt is relatively resistant. The varieties Vermilion Brilliant, White Swan, White Hawk, Double Early Tea Rose, are all susceptible single varieties. The out-of-door May-flowering cottage tulip Inglescombe Yellow is very resistant.

To check the trouble, diseased bulbs should be removed and destroyed. About a fortnight before planting, the soil should be treated with a 2 per cent. solution of formalin. The temperature of the glasshouse should be kept as low as possible without injury to the plants and too great humidity should be avoided [2].

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Tulip Fire, *Botrytis tulipae* Lind

This disease is very common on cultivated tulips and it also attacks the wild species *Tulipa sylvestris*, but no other plant. ' Tulip fire ' has been known for over a century but it was not until 1888 that it was first described, in Italy [5]. Its occurrence in England was noted in the same year, [16], and again in 1900 in Herefordshire [4]. It was found in America in 1902, on material believed to have been imported from Holland [8]. The disease now occurs extensively in the United States, Canada, and throughout Europe.

In some years the trouble is accountable for heavy losses in the tulip industry ; in 1927 and 1928 fire blight was so severe in West Cornwall that the growers were unable to market the bloom [3]. The disease affects the leaves and bulbs as well as the flowers ; the bulbs fail to fill out owing to impoverishment of the leaves, and in severe attacks gaps in the beds indicate complete destruction of bulbs below ground.

A cold, wet spring is generally regarded as being favourable to the disease and epidemics are encouraged during periods of high rainfall.



FIG. 406.—Tulip fire (*Botrytis tulipae*). Severely diseased leaves and bloom. Inset, the conidial fructifications on leaf lesions

The fungus causing tulip fire was first named *Sclerotium tulipae* owing to the prominence of black sclerotia on the bulbs, but on the discovery of a *Botrytis*-stage in the life-history the fungus was renamed *Botrytis tulipae* [6, 10, 15]. The mycelium of *B. tulipae* often shows considerable anastomoses and in the bulb scales the vascular bundles are markedly disrupted following infection. The conidiophores arising directly from the mycelium are erect, brown, twisted at the base, and proliferate to form repeated crops of conidia which are liberated along with their sterigmata (Fig. 408). The conidia are formed on the leaves, flowers, stalks, and capsules, but never on the bulbs [3]. They are obovate, reddish brown in the mass, grey to hyaline singly, and fairly large, measuring, according to various authors, 12 to 24 by 10 to 20 μ [9]; or, 10 to 15 by 6 to 9 μ [3]; or, 2 to 22 by 8 to 13 μ [17]. Smaller microconidia, globose, 3 μ in diameter, occurring on special penicillate conidiophores arising in white tufts from the substratum, are also found [9]. Sclerotia appear on moribund tissues in the bulb scales and base of the flower stalk, but not if atmospheric humidity is low; on potato-dextrose agar they develop after the mycelium has become adpressed to the surface; at first white, sclerotia are finally black, rather small, 1 to 2 mm. in diameter, circular or elliptical in outline, flattened, often convex [9]. The fungus thrives at comparatively low temperatures and sporulation does not occur above 25° C.; at higher temperatures the low humidities prevailing are probably a limiting factor in infection [3].

Tulip fire appears on the green leaves as grey, sunken spots of variable size and shape, each spot being surrounded by a darker, water-soaked area, and towards the centre abundant conidiophores with conidia are early produced (Figs. 406, 408). These primary lesions enlarge quickly in wet weather, often uniting so as to cover the whole leaf, which wilts and finally appears as if scorched, hence the name 'fire blight' commonly applied to the disease. In dry weather affected leaves are liable to split and tear at the tips, becoming much lacerated by wind action, and as any of the broken parts may be covered with conidia when damp conditions return,

much infection is conveyed by wind to neighbouring plants and beds. If the lesions occur towards the base of the leaf stalk the lamina usually falls over and withers on the ground.

Conidia carried by wind or in rain-drops splashed from the primary fire lesions are accountable for extensive secondary infections of near-by plants. The leaves of plants secondarily infected do not, however, develop the large grey spots but merely become flecked with small, dry, slightly depressed spots of a yellow-white or greyish-white colour furnished with a border of darker green than the surrounding tissues. Apparently there is so little mycelium in the leaf tissue below these small secondary spots that they do not appear to spread and they do not usually give rise to conidia, possibly because the mycelium below them has, through the action of some undetermined factor, been killed or checked in its growth. On very rare occasions, however, when these small spots have actually yielded conidia, sclerotia have been found to follow them, but only after the death of the leaves. On the blooms the symptoms resemble the fire lesions on the foliage, and the spots may occur on the unopened flower buds or on the expanded petals (Fig. 407 B). The greyish lesions spread quickly and entire buds or flowers may be ruined in a short time. These lesions on the blooms sporulate so freely that affected flowers,



FIG. 407.—Tulip fire (*Botrytis tulipae*). *A*, primary infection in variety William Pitt (photo by Dillon Weston). *B*, blister spots on the bloom. *C*, bulb showing sclerotia (photos by Beaumont)

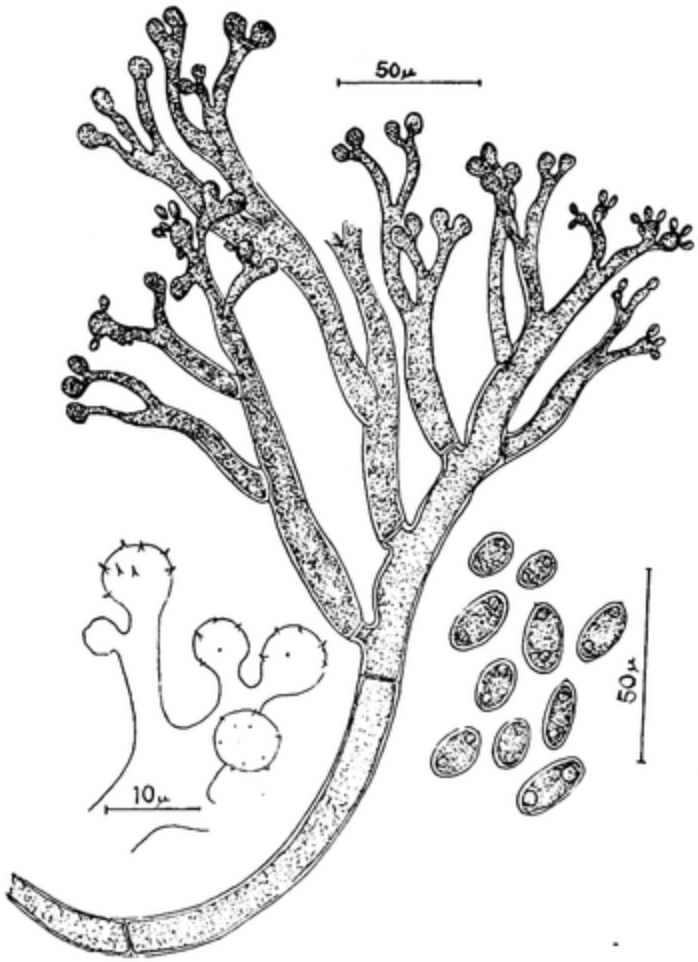


FIG. 408.—*Botrytis tulipae*. The sympodially branched, curved conidiophore; portion on left showing the minute sterigmata on the swollen terminations of the sporiferous branches; on right, mature conidia

elevated on their stalks, are a prolific source of infection in the beds. Two other kinds of spots may also occur on the flowers. One, a small white spot type (producing a striking effect on red tulips), resembles somewhat the small blemishes of limited growth seen on the foliage leaves and, like them, remains small and non-sporulating: the second kind consists of larger, white, but spreading type of blister-like spots each surrounded by a raised, wrinkled margin (Fig. 407). In a damp atmosphere the blisters sporulate freely and this serious phase of the disease accounts for very heavy losses in flowers packed in boxes for transit. Under such enclosed humid conditions, ideally maintained within the unexpanded blooms, the fungus spreads to the stamens and pistil, and in this way the young capsular fruits and even the seeds may become infected. On the flower stalks the lesions resemble those on the foliage leaves but are smaller and elongated, and towards the base of the stalk, that is, close to the bulb, the lesions may contain black sclerotia. It appears that sclerotia are very rarely found on decayed leaves in the field [3].

On the bulbs themselves, the lesions are confined mostly to the outermost scales. The bulb lesions are yellow or brown sunken areas containing a few or numerous sclerotia, which sometimes may be so abundant as to form a continuous crust covering the bulb (Fig. 407 C). Sclerotia may appear on both surfaces of the discoloured and decaying scales. The roots are not affected. The presence of the disease on the outer scales may or may not involve the inner scales. Unless the season has been continuously wet the disease is usually confined to the outer scales, and if affected bulbs are lifted with a good deal of adherent soil, which, with the outer scales, is carefully removed, the inner scales are usually found to be entirely free from disease, but in a wet season whole bulbs may be destroyed [2].

There is yet another and distinct form of this disease which may or may not be accompanied by the various types of spotting of the foliage and flowers described above. In this case the tulip plants are dwarfed, there is a general wilting and softening of the leaves, and no flowers, or only a few small ones, are produced.

When the bulbs of these sickly plants are lifted, their scales are found to be covered with black sclerotia and in severe infections of this kind no new bulbils are formed. This severe type of bulb rot, however, is not common, and is believed to follow only when the bulbs are injured, or as a result of unusually heavy infection [3].

B. tulipae is capable of hibernating both as mycelium and sclerotia in the scales of dormant bulbs, or as sclerotia liberated into the soil when the bulbs are left to decay. It is probable that true primary infections are traceable to germinating sclerotia in the soil and experiments have shown that tulip fire may start afresh from this source [3]. When the bulbs begin to grow, the first infections usually occur on the emergent neck, and in the case of bulbs already infected the fungus may spread through the entire outer scales, but it is not usual for the reviving fungus to penetrate from the outer infected to the inner healthy scales. These initial infections arise on the young shoot, during emergence, from contact of the shoot with, and penetration by, the revived mycelium sent out from the outer scales, and as only the outer parts are penetrated the rest of the shoot grows out, as yet unaffected. With the development of the fungus in the outer tissues of the neck it is not long, however, before conidiophores carrying their first load of conidia emerge through the stomata in this region, above soil-level, and when the spores are carried by wind or in splashing rain-drops to the expanding green shoot leaf infections quickly follow. Thus, the fire blight spots on the leaves arise from external infections. When sound bulbs are planted in soil contaminated with sclerotia, penetrations take place when hyphae from germinating sclerotia attack the young shoot as it emerges from the bulb (not through an outer scale, unless wounded) and thereafter the progress of infection is the same as described above. In severe infections, when the entire emerging apex is penetrated at various points, the whole young shoot is usually destroyed before it appears above ground. This serious phase of the disease accounts for bulb rot and gaps in the beds.

At the close of the season the proportion of new bulbs found to be affected is generally small and, as already stated, even if the outer scales are diseased, the new bulbs formed around the base of the flower stalk usually remain clean. In a wet season, however, conidia may be carried, perhaps washed down by rain from badly diseased tops to the bulbs, and by infecting the scales produce mycelium and sclerotia [9].

Much can be done to avoid outbreaks of tulip fire by attending to cultivation [1]. No bulbs showing any blemishes on the scales should be planted and planting should be deeper than customary, setting the bulbs more apart than usual, care being taken not to damage the neck tissues [3]. If possible the beds should be given over to rotation and contaminated soil should not be used for tulip bulbs for at least two years, after which it may be assumed that all sclerotia have been starved out [11]. Growers who spray the foliage usually perform the treatment in early spring [13]. Good results, with little danger from spray injury, are recorded by the use of potassium sulphide with resin, prepared in two lots : (*a*) by dissolving 4 lb. flowers of sulphur in a hot solution of 5 lb. caustic potash in 10 lb. water, and (*b*) heating 4 lb. pine resin with 2 lb. caustic soda in 10 lb. water, mixing equal parts of these, as required, using for the spray a dilution of 0·8 per cent. of the mixture [12]. Another method is to steep the bulbs in 2 per cent. formaldehyde [7],

or 0·03 per cent. solution of mercuric chloride, or 0·5 per cent. uspulun solution, before planting [12, 14]. Careful watch should be made of the beds in early growth, and any plants suspected of disease should be removed, together with some of the soil around them, for it is from such individuals here and there in the beds that infection is spread. Should a large number of the bulbs show early infection it may be found advisable to lift the entire planting, selecting only the clean bulbs for resetting in a fresh bed. All tulips are not attacked by fire blight to the same degree; in Devon and Cornwall, the variety Baronne de la Tocnaye appears to suffer less than the widely grown Darwin varieties, William Pitt, William Copeland, and Bartigon, and these observations appear to be true for all districts [3].

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Tulip Breaking

'Breaking' of tulips is believed to be the oldest of all known virus diseases of plants. It is so named because of the effect it produces in disturbing the natural, uniform colouring of the flowers. All varieties of tulips are affected. Narcissi and hyacinths are also susceptible and both become definitely more diseased than tulips [4]. The disease is widely distributed in Holland, France (believed imported from Holland), Belgium, the British Isles, and the United States, and probably occurs wherever tulips are grown [1, 2, 5, 7, 8, 15].

Whilst the virus is not lethal to the plants, infection has a weakening effect, and in an unfavourable environment, or when growth conditions are poor, infected plants are always the first to suffer from a deterioration and loss of the bulbs [11]. Nevertheless some of the earlier growers, ignorant of the true nature of the disease, who noticed that an attractive variegation sometimes developed in a number of self-coloured tulips, discovered that the 'symptoms' could be induced by grafting healthy tulips on to the bulbs of the 'new' flowers, often with the desired results. But sometimes the infective principle appears to be localised, so that a 'broken' bulb may have 'unbroken' parts, and the latter may give rise to healthy offsets, and vice versa [3].

The effect of the virus in the plant is either to remove, or augment, or sometimes to rearrange the anthocyanin pigments in various parts of the epidermis of the

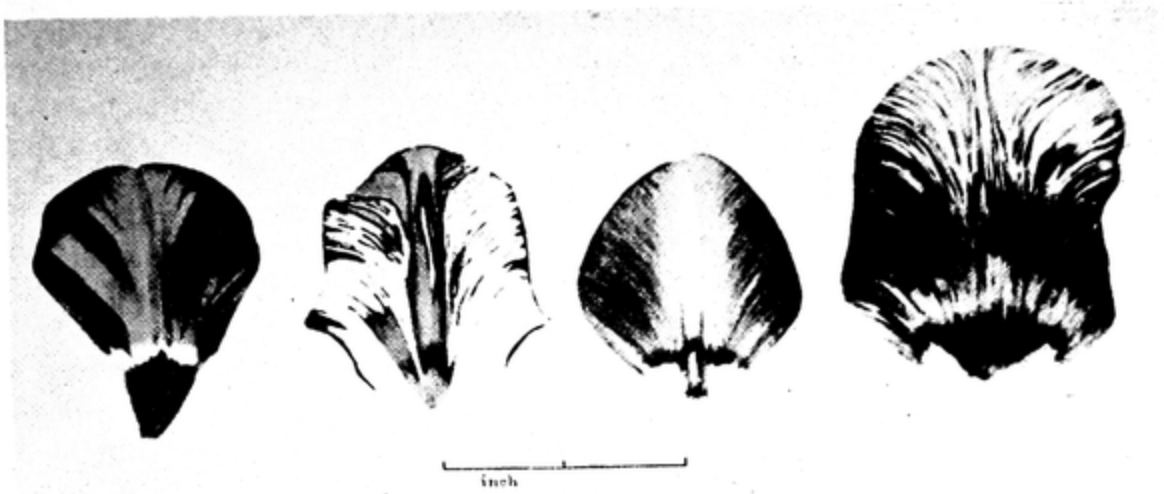


FIG. 409.—' Breaking ' of tulip. Left to right, ' clotted break ' (Isis) ; ' full break ' (Bartigon) ; ' self break ' (Bartigon), light ; ' self break ' (Bartigon), heavy (after McKenny Hughes, *Ann. App. Biol.*)

flower, so that a variegated pattern is produced [4, 15]. The effects could not, of course, be expected to be always identical, and three or four more or less distinctive types of ' breaks ' (Fig. 409) are now recognised [11, 14] :

(*a*) *Full Break* : when the natural colouring of the ' breeder ' is removed, so as to give place to the yellow or white colour of the mesophyll, which becomes the dominant colour of the flower ;

(*b*) *Self Break* : when the colour of the ' breeder ' becomes darker, or intensified into darker streaks or stripes ;

(*c*) *Clotted or Clotting* : when, in dark-purple or dark-red shiny varieties, which never show the ' full break ', the self colour is intensified in streaks and patches ; and

(*d*) *Typical Break, or Average Break* : a condition which is characteristic of commercial Rembrandts where the balance between epidermal pigments and exposed ground colour (white or yellow) produces a ' flashy ' or ' flag ' break. The usual colour of the flower is intensified in some areas, unchanged in other areas, and is removed in still others [14]. Clearly (*c*) and (*d*) deal with closely parallel effects.

In some varieties of tulips, not in all, the virus also induces a mottled or striped effect in the green leaves. Sometimes this may be very slight or indistinct and consist of spots or streaks of a silver-grey to a light-green colour ; in other cases the effect becomes more conspicuous as the affected parts lose their chlorophyll and the leaf becomes more or less transparent, loses turgor, and develops a tough texture [2]. Affected bulbs do not proliferate so freely and the plant comes into full bloom a week or so later than the ' breeder ' [4].

The virus of tulip ' breaking ' is named *Tulipa virus 1* Smith em. Brierley & F. F. Smith 1944 ; *Tulip ' break ' virus* and *Tulip mosaic virus* appear to be synonyms [4, 6, 15]. It is not clear, however, whether ' breaking ' should be regarded as a ' composite ' disease due to two viruses, one tending to *add* colour

to the flowers, and the other to *remove* it, but recent investigations support the so-called ' antithetic ' virus theory, applied to two naturally associated viruses to connote differences in the symptom-picture which relate to some inherent antagonism between the two viruses. Accordingly the antithetic viruses of tulip ' breaking ' are *Tulip virus 1* and *Tulip virus 2*, with the following properties :

Virus 1 : colour removing; inhibits chlorophyll formation; retards growth; more virulent than *Virus 2*, being directly responsible for actual disease in the plant; it reduces plant growth to one-third of the normal.

Virus 2 : colour adding; has no effect on the ground tissue of the flower, or on the ground colour; has no visible effects on the leaves, and little on the growth. That *Virus 1* has proved to be *dominant* indicates that the differences between the two is of a higher order than that of ' strains ' (12, 13, 14).

While it is understood that ' full break ' results from the presence within the plant of the two viruses, there is clear evidence that the symptoms of ' self break ' can be transmitted separately, for the insect vector implicated is able to ' select ' from ' full break ' tulips the infective principle which, when transmitted to healthy tulips, produces ' self break ' alone. Such ' self break ' tulips can then only transmit ' self break '. There is, however, no evidence of the transmission of a second virus alone, and ' full break ' is comparatively constant; but ' self break ' is apparently not so stable, as in some instances it has reverted to normal. There is evidence that some varieties of tulips are more susceptible to attack, and that some carry the virus in a more virulent form (11). The present position with regard to tulip viruses is, however, not clear. The plant is susceptible to attack by several viruses which apparently form a group of closely related strains (16).

' Breaking ' is transmitted by insect agency, the vectors being *Myzus persicae* and *Macrosiphum gei* in the glasshouse and out of doors, while *Anuraphia tulipae* is a vector in the bulb store, but not on the growing plant (7, 15). It can also be transmitted by grafting and plugging bulbs with tissue from ' broken ' bulbs, but is not conveyed in the true seed (Fig. 173). Artificial inoculation with a hypodermic needle can be performed with juice expressed from tulip stems and leaves, filtered through cloth, and diluted to 1 part in 10 or 20 with sterile distilled water; inoculations can be made into stems while the plants are in bloom or shortly after (14).

The disease may be kept under control by roguing of infected bulbs, and it is of first importance to destroy the insect vectors by fumigation of the glasshouses. Lifting and storage of the bulbs should be carried out under aphis-free conditions in the autumn (4).

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Downy Mildew of Hop, *Pseudoperonospora humuli* (Miy. & Tak.) Wilson

Downy mildew of hops was first recorded in 1905, in Japan [17], on native wild and cultivated hops; in North America in 1909 [7]; in England on a small scale in 1920, but not in commercial hop gardens until 1924 [6, 27, 33]. It was fairly general in Europe by 1920 and is now widely distributed in all hop-growing areas, causing severe damage to the crop. The disease has been extensively studied in this country by Salmon and Ware [19-32, 34, 35], and in Czechoslovakia [3, 3a]. Commercial losses are gauged on damage to the 'burrs' or 'cones', and extent of infection may vary from mere discoloration of the hop cones, which renders them unsightly and unmarketable, to the total destruction of the bines through disease. Some growers pick the cones before they are mature, in order to avoid discoloration, but as this practice entails an appreciable reduction in the weight of the crop, the immature hops being also less rich in brewing properties, it is quite a common experience to suffer a 25 per cent. loss through early picking [26].

In March or April, when the normal young green bines are emerging from the crown or root-stock, the disease is known to be present if amongst these healthy slender shoots there arise a number of dwarfed thicker shoots, pale green or silvery grey in colour, and varying from a few inches to a foot or more in length (Fig. 410). These thickened shoots are invariably infected, and are known as 'basal spikes'. Later in the season some of the thin and apparently normal healthy stems, which may have grown as high as 5 to 14 feet, become checked by the formation of thickened extremities, or 'terminal spikes' as they are called. Furthermore, in some cases similar hypertrophied shoots, a few inches long, may also be seen growing as lateral branches on the otherwise normal and healthy main stem, and are called 'lateral spikes' [22]. Thus there are three positions on the plant where the thickened abnormal shoots occur, basal, terminal, and lateral; all these spikes are infected. Such attacked bines lose their twining habit and are difficult to train [16].

The early formed basal spikes bear reduced, brittle leaves growing close together. These leaves are grey on the upper surface, but on the under side, as well as on the surface of the stem, there are dense masses of dark-coloured spores and sporangiophores of the mildew causing this disease. These spores, dispersed by wind or splashed by rain, bring about secondary infections of the more or less expanded leaves of healthy shoots. On the leaves, the resulting spots are dark brown above but paler on the lower surface, and characteristically angular in shape. The spots may join together to form large patches, and it is then, when the sporangiophores are massed together, that the under side of the affected leaves becomes blackish grey, or sometimes tinged with violet, especially along the



FIG. 410.—Downy mildew of hop (*Pseudoperonospora humuli*). *A*, plant showing the normal, thin shoots, and infected, thick, short ' basal spikes ' at the base. *B*, a normal bine. *C*, a basal spike. *D*, *E*, two infected ' cones ' showing dark infected scales (photos by Ware, Wye Reports)

flanks of the midrib, and along the margins of the leaves, which later turn brown and shrivel. The hop ' cones ' may also be attacked. The cones, the female inflorescences of the plant, consist of a number of stipular bracts and bracteoles, both of which become large and membranous, and are covered with yellow glands which secrete lupulin, to which the hop owes its brewing properties. The bracteoles are usually attacked first, turn brown, and give a striped effect with the alternating stipules which remain green ; but they, too, turn brown later and the

entire cone becomes worthless. The cones are attacked mostly as they approach maturity and suffer little damage to their brewing properties in late attacks, but earlier attacks are naturally destructive to the lupulin glands and heavy losses are incurred [26].

Downy mildew is caused by *Pseudoperonospora humuli*, a member of the Peronosporales (Fig. 411). The sporangiophores, from 100 to 450 μ long, develop in subdued light (they emerge at night) and are violet-black in the mass; they are aseptate and branch three or four times in dichotomous fashion before forming the ultimate branches, thin and pointed, bearing the oval, thin-walled, papillate sporangia which range from 22 to 26 by 15 to 18 μ [17]. With a high degree of humidity (65 to 95 per cent.), sporangia are viable for some 30 days and are not killed by freezing water. They germinate by the production of zoospores, the activity of which depends largely on temperature, lasting for 24 hours at low temperatures of 4° to 7° C. and from 2 to 6 hours at 20° to 22° C., but only some 25 minutes at 30° C. [1]; germination was improved when calcium citrate or a hop leaf was added to the water [16 a]. In early autumn, oospores may be found in the diseased tissues of practically all parts, in the leaves, stems, spikes, and the cones; they are round, smooth, light brown and thick-walled, from 36 to 40 μ in diameter; the presence of antheridia has not been determined but the oospores are enclosed within oogonia which vary from 40 to 50 μ wide. The oospores germinate at 20° to 22° C., producing a hypha which bears a much larger sporangium than the ordinary kind and which gives rise to more and larger zoospores, 40 to 60 as against 5 to 12 for the ordinary sporangia [1].

With the decay of infected parts, the oospores are released into the soil and are believed to be responsible for carrying over the disease from one season to the next. There are, however, conflicting reports as to their capacity for resisting desiccation. It is stated that while they may persist for years in the soil under ordinary conditions, they did not survive after being kept dry for 4 months in the laboratory [1], but oospores which were picked from pressed herbarium material, 2 years old, germinated after being ground with a pestle and mortar [5], an operation which, by causing

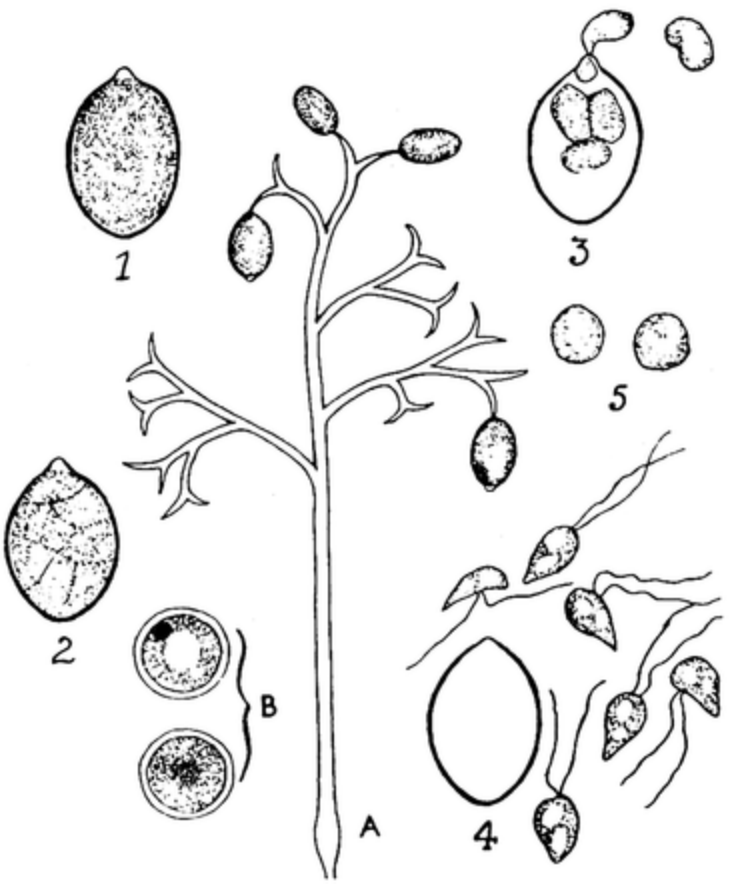


FIG. 411.—*Pseudoperonospora humuli*. *A*, sporangiophore. *B*, two oospores (× 250), from infected leaf; 1-5, stages in formation of zoospores within a sporangium; 5, zoospore just prior to infection of hop leaf or cone (× 562) (after Salmon & Ware, Wye Reports)

the tough wall of the oospores to crack, possibly induced the spores to germinate; and the oospores are also reported to have survived dry storage for 2½ years at laboratory temperatures, and even as low as −18° C. (16a).

Primary infections, by zoospores produced by the germination of resting oospores in the soil, in the spring take place only under wet conditions and are directed against the young buds emerging from the crown. First infections of healthy plants are external, at or near soil-level, through the stomata, which are entered by the zoospores (1). Shoots thus infected are retarded in growth and become the basal spikes above mentioned; those not infected, despite possibly close contact with them, grow forward as healthy bines. It remains, therefore, difficult to explain how some of the latter, several feet long, sometimes come to possess diseased spikes at their tips, well clear of the ground. Though there is abundant evidence that the fungus, besides entering the basal spikes in the manner described, later invades and persists during the winter in the underground parts of the hop, in one-year parts of the root-stock (crown), in nursery 'sets', and even in the roots, it appears that such resting mycelium is not continuous from below, through the thin climbing bine to the thicker apical and lateral spikes. Though infection of the root-stock is by no means general (and setting aside for the moment the probability of infection by oospores), it is not unreasonable to suppose that the first spikes of the season, that is, the basal ones, may become infected by the migration of hibernating mycelium from the root-stock into the buds which produced them. It was once held that the apical and lateral spikes might also be infected in the same way, but it is characteristic of the disease that infected buds make little headway and are effectively 'spiked'. How, then, are the long, more or less normal internodes of these affected bines to be accounted for if eventually they come to develop apical or lateral spikes, since it has been established that there is no continuity of mycelium from crown to spike? There are several suggestions: (*a*) that the resting mycelium had already invaded a bud which had started growth and, carried up by the rapidly elongating bine, the mycelium got left behind in small portions at certain nodes or, making a complete break at the base, got carried up with the bud in its entirety; this view is now considered unlikely, despite the fact that a shoot can often make considerable growth after it has become internally infected and before it becomes definitely spiked, but there is no direct evidence that bines over two or three feet high, ending in terminal spikes, can carry internal mycelium from the start; (*b*) that, following upon a number of independent penetrations by zoospores, the mycelium developing at each of these separate points remained separate in the stem and capable of setting up distinct infections of, say, the lateral branches, resulting in the formation of lateral spikes; (*c*) a modification of the foregoing, that whole internodes might have been internally infected when young, but that, by dint of very rapid intercalary growth, the mycelium got torn apart in places and came to reside in separate units of the stem; and (*d*) the most feasible explanation, that all three types of spikes, the basal, the terminal, and the lateral, arise following upon infection when in the bud stage. This receives its strongest support from the fact that young buds yield readily to external infection by the application to them of a suspension of zoospores. Spraying a water suspension of zoospores

on to the elongating buds sets up infection in the protective stipules and these rudiments soon become covered with conidia before the latter appear on contiguous leaves. Infection may or may not reach the petioles of the leaves, but if it does it is conceivable that, starting from an apical bud at the end of a main stem or lateral branch, the fungus developing from the applied zoospores may spread downwards through several internodes, but not, however, to reach as far down as the crown [8, 35]; this downward method of infection would, therefore, explain the lack of continuity between mycelium in the bines and mycelium in the crown.

The mycelium within diseased shoots of all kinds occurs mostly in the pith, and from the tissues of the crown (root-stock) it may extend into recent one-year-old parts of the root-stock and deeply down into the underground parts of the plant, even into the roots (Fig. 98). In a longitudinal section of a hypertrophied shoot the fungus in the cortex and pith of the stem may be seen to invade the petiolar tissues to occupy the lamina from base to apex; the axillary buds are likewise invaded, even to the tips of every scale leaf. In the stem it traverses the phloem, travelling radially by means of the medullary rays, browning all the tissues in its path, those of the woody cells included. The mycelium is furnished with haustoria, especially in the phloem region [18]. It is recorded that when a sixteen-node bine with a terminal spike was examined, up to the tenth node was healthy, but from the eleventh node up, which constituted the 'spike', the tissues were infected, and the internodes of this region were considerably shorter than those below. Occasionally, at a node, one leaf of a pair or a lateral may be infected and bear sporangia, while the opposite leaf or lateral may be free [34]. Oospores may be formed in any of the affected parts, even in the bracts of the cone, and are especially abundant in the dead leaves and in the pith of the stems. When cotyledons of young hop seedlings were inoculated with fragments of infected leaves, oospores were found in these organs as well [14]. In British Columbia experiments on seedling infection showed that it occurred at or below soil-level, the cotyledons being penetrated first, the fungus thereafter causing systemic infection [13].

Periods of high rainfall and high atmospheric humidity during the growing season are highly conducive to downy mildew of hops, and are most closely correlated with incidence of infection [2]. With humidities of 90 to 100 per cent. the incubation period is about 6 days at temperatures of 20° to 22° C., but is retarded in dry weather and at low temperatures of 6° to 7° C. [1, 11, 36]. Experiments in Czechoslovakia showed that infection by zoospores occurred at 16° C. and upwards, and it is believed that spring infections result from oospores [3].

Infected plants have a higher percentage of nitrogen and phosphoric acid, but less potash and lime contents, than healthy plants. The application of potash and lime is said to decrease the susceptibility to mildew, the reverse being the case with phosphoric acid and nitrogenous substances [4].

No variety of cultivated hop is immune from downy mildew. Tolhurst, Bromling, and Prolific are susceptible and the once resistant Fuggle is now in most districts liable to severe attack, more so on leaves and stems than on the cones. The cones of Fillpocket and Early Promise show marked resistance [32].

Removal of the thick basal spikes as they appear during the season will reduce the spread of infection, since they have been shown to be the principal source of

the mildew. When they are very prolific their constant removal often causes a serious shortage of bines for training, and entire plants have frequently to be cleared from the hills. If terminal and lateral spikes appear they should be cut off and healthy laterals should be encouraged for training. To reduce the risk of secondary infections from below, the leaves should be removed from the bines, in stages, up to the ' breast wire '.

Good control is obtained by spraying the bines with 10 : 10 : 100 Bordeaux mixture [2, 10, 26]: (*a*) when the bines are three-quarters way up the poles; (*b*) when they have reached the top; (*c*) just before the plants come into ' burr '; and (*d*) immediately after the ' burr ' has gone, when the hops have set. No spraying should be done when the hops are in ' burr ' [26]. Some growers employ the mixture in dust form (copper sulphate 16, hydrated lime 100 parts, at rate of 1 oz. per hill); the soil around each hill is turned back so that the young shoots can be cut close to the crowns and the powder is then applied evenly on and around the crowns, which are partially covered again with soil. The treatment is said to reduce the number of basal spikes [12].

All prunings and cones should be removed and burned. Sets from infected gardens should not be used to start a new crop and all wild hops in the vicinity should be grubbed out and destroyed.

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Powdery Mildew of Hop, *Sphaerotheca humuli* (DC.) Burr.

Mould or powdery mildew is a serious disease of hops. It is caused by a specialised race of *Sphaerotheca humuli* (*Erysiphaceae*). Other races of this fungus attack raspberry and strawberry, in each case affecting the leaves and rendering the fruit small and worthless [1, 2, 4, 3-10].

The mildew first appears in May or June, on young leaves, petioles, and tender stems, as white patches which early become covered over with powdery masses of conidia. The flowers are especially susceptible, and infected ' cones ' are useless for brewing purposes; the latter are frequently covered with the red cleistocarps of the fungus and the term ' red mould ' is often given to this stage of the disease.

First symptoms of infection on the leaves consist of small raised blisters, hardly discernible because of their green colour, but after a day or two each blister develops on its surface a white mycelium which branches and radiates in all directions but remains entirely superficial except for small haustoria established in the epidermal cells.

The fungus increases on the surface to form a web of mycelium from which dense formations of conidiophores bearing conidia develop. The latter are oval and unicellular and serve for rapid propagation of the fungus during periods of warm, moist weather. They are not adapted for survival through the winter. The cleistocarps are developed later, on leaves and ' cones ', and may pass the winter whilst still on the bines, or more commonly on the fallen leaves and shattered cones on the ground. The reddish cleistocarps are scattered or crowded, and measure from 58 to 120 μ in diameter; the long appendages, straight or twisted, are dark brown in colour; the single ascus is broadly elliptical to sub-globose, 45 to 90 by 50 to 72 μ; the 8 ascospores measure from 20 to 25, rarely 30 by 12 to 18 μ (mean, 22 by 15 μ) [3]. A winter resting period of the ascospores within the cleistocarps appears to be essential for their successful germination [2, 4].

The ascospores are responsible for primary infections, the conidia for secondary infections, and both produce germ-tubes with appressoria at infection. Depending on the wetness of the season, leaf spots may appear in 5 days to 3 weeks from infection by ascospores, and after the first appearance of conidia on the leaves widespread secondary infections follow in about 10 days. In wet periods entire bines with flowers and ' cones ' become rapidly infected. The cones are attacked in the inflorescence stage, at a time when the tiny female flowers are exposing their stigmas for the reception of pollen, in which condition the young cone is known as a ' burr '. Carried like pollen by wind, the conidia are caught up and germinate on the stigmas. The fungus may travel so rapidly in the flowers as to inhibit further development, and in place of the normal fruits or cones there are formed only hard knobs, and after an attack of this kind the cones are worthless.

The burrs can also be attacked, with less serious consequences, through their stipular bracts and bracteoles, in much the same way as leaves.

Different varieties of hops vary in susceptibility to mildew [2], and 'immune' kinds may sometimes show slight susceptibility, when submitted to a change of environment, without, however, the general immunity being lost [9]. Experiments at East Malling in 1945 on a number of new seedling hops revealed the interesting feature that there appeared to be a tendency for heavy and poor cropping to be associated with a high and low incidence of the disease, respectively. But amongst the seedlings which kept free from mould some proved to be heavy croppers and of distinct commercial promise [11].

Good control over the disease may be obtained by dusting the bines with sulphur. The first application is usually made in May (when the ascospores are about to appear), preferably before any spots have begun to appear. The leaves, especially those at the base of the bine, should be well dusted over, and the dusting should be repeated every fortnight until the hops are approaching maturity. All diseased bines and cones should be collected and destroyed and any runner shoots and unwanted laterals removed. A balanced manurial treatment (see downy mildew, p. 883) should be planned and heavy nitrogenous fertilising avoided.

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Chapter XVII

DISEASES OF TREES

Watermark Disease of Willow, *Bacterium salicis* Day

THIS bacterial disease of the cricket-bat willow (*Salix alba* var. *caerulea*) causes a brown, watery stain in the wood and renders the timber quite unsuitable for processed work [9]. It was first observed in England in 1921, in the Chelmer Valley, and has been extensively studied in this area by numerous investigators [2, 3, 4, 9, 11]. The trouble has since spread to Essex and Hertfordshire and is present also in Cambridgeshire. The white willow (*S. alba*) is also susceptible and the crack willow (*S. fragilis*) is occasionally subject to the watermark disease; in Holland, *S. amygdalina* and *S. purpurea* suffer from what appears, with slight differences, to be the same disease [1, 6, 7, 8].

Watermark disease is rarely seen on trees less than three years old. In general the symptoms are those of a partial die-back of the crown, starting, in Britain, during the late spring and apparently ceasing during the summer, continuing again the next year; but in Holland these symptoms are prolonged until September, due probably to the continuance of conditions favourable to the disease. In Britain the trees are affected much more severely in riverside plantations and in damp, riverside meadows, where the subsoil is permanently wet, than in drained, moderately moist areas [3].

The trees usually show the first symptoms of the trouble soon after commencement of growth in June, or later, when the leaves and tips of the new shoots on one or a few branches wilt and die. These early affected branches often occur on a part of the tree which is shaded over, but the disease is not confined to shaded branches. Following the withering of the leaves, affected branches die back slowly from the tips, but after the loss of the leaves the branches show no further disease during the same year. The browned and withered leaves, however, often remain attached to the branches for a considerable time, a phenomenon which probably accounts for the name 'red leaf' being given to the disease in some localities [5]. In succeeding years, the trouble continues to spread within the tree and death may ensue in a year to two, or the infection may remain dormant or die out, but it is obvious that trees once affected are of little value, and are better removed and burned to avoid risk of spreading infection.

A striking symptom of watermark disease is the exudation of bacterial slime on the dying twigs and young branches, leaving dark, sticky tracks, but this feature is not usually seen on the larger branches. The slime is created internally, in the wood, and is exuded to the surface through wounds, or bore-holes made by insects. In the Dutch form of the disease the exudate has also been found in the

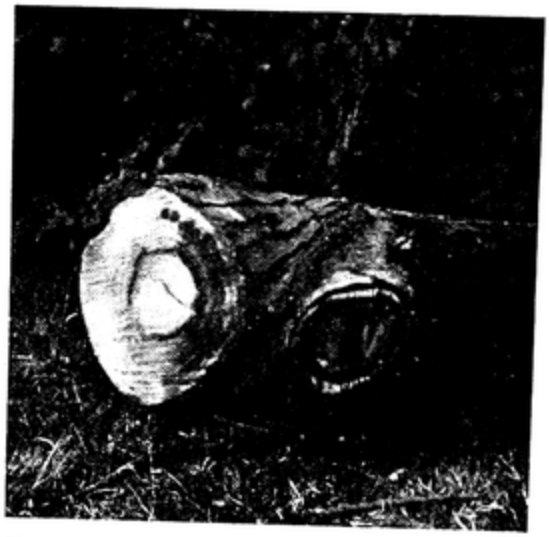


FIG. 412.—'Watermark' of willow (*Bacterium salicis*). A trunk watermarked on one side only and with a very badly affected branch on the same side (August 1922) (photo by Day, *Oxford Forestry Memoir*, 3)

leaf axils, at the base of small lateral twigs, in leaf scars, and in fissures reaching as deep as the cortex [8]. On the branches the exudate forms a substrate for various yeasts and fungi, and unless washed off by rain, dries hard like a yellow or brown varnish [3]. One of the first fungi to appear on the dying parts is *Cytospora chrysosperma*, and so frequently is this fungus associated with watermark disease that it was suspected to be the causal organism; but it is clearly an intruder which, however, along with other fungi assists in the ultimate death of the tree; on the dead bark, the pycnidial fructifications of *Cytospora*, with their orange-coloured spore tendrils, are usually conspicuous during the autumn and winter. Various other fungi may also thrive between the bark and wood of affected trees, and entire branches and trees may be debarked and bared down to the wood, through their agency.

When affected branches are cut across, the disease is readily identified by the brown 'watermark' in the rings of the woody xylem (Fig. 412). This discoloration may appear in one part only, or occur more or less general over the entire section, or form discrete patches of stained wood. It is due to the presence of bacteria in the vessels of the wood (Figs. 413, 414). Watermark disease is essentially a vascular trouble which kills the tree if the bacteria find their way into the current year's wood of the main stem.

It is not clear whether this disease is due to one bacterium or a number of different bacteria, but the organism named *Bacterium salicis* appears to be the true parasite [3]. Others, almost constantly present, have been designated Bacterium 'A', 'B', and 'C' [9, 10]. *B. salicis* is a short, rod-shaped organism, 0·8 to 1·0 by 0·3 to 0·4 μ; non-sporing; capsulate; furnished with peritrichic flagella [4]; not acid fast; non-liquefying; aerobic and facultative anaerobic; nitrates not reduced; the temperatures for growth are:—optimum 29° to 30°, minimum 5° to 10°, maximum 33° to 37° C.; a soluble toxin is formed [4]. The Dutch organism, regarded as a distinct species, *Pseudomonas saliciperda* [8], has an average length of 1·7 μ; its optimum temperature for growth lies between 25° to 30° C.; it is Gram-negative and nitrate-reducing. Metcalfe [9] describes and gives the dimensions for Bacterium 'A': rods, 1·75 by 0·8 μ, motile, with 2 to 5 peritrichic flagella. 'B': short rods, 1·4 by 0·75 μ, motile, with 3 to 7 peritrichic flagella. 'C': long rods, 2·25 by 0·8 μ, motile, with 1 to 3 polar flagella.

The mode of infection of healthy trees is not definitely known, but fresh infections always start in the crown. They are believed to arise through wounds made by insect borers which pupate in infected trees and later act as carriers of infection [2]. Infection may also probably take place from crown to crown of neighbouring trees when the slimy bacterial exudate is splashed by wind-blown rain from tree to tree, but again the organisms are capable of penetrating the twigs

only through wounds. The bacteria do not enter by the leaves, nor are they ever present in these organs, and the effect on the foliage appears to be due to a toxic substance carried up in the transpiration stream.

The bacteria are most abundant and active in the outer ring of the wood, but are not easily detected at first, because the zone is water-logged and colourless. In the dying branches, however, the zone or ' mark ' is easier to trace because it stains the wood a deep brown and can be followed into the main stem. In the latter, the stain in the outer wood extends up and down from the point where it came in from a branch and, travelling more rapidly downwards, may eventually reach the roots, but not always. At first it occupies the one-year-old ring of wood and only slightly the adjacent autumn wood of the older ring. The brown discoloration is a secondary effect and gradually disappears after the death of the tree ; it is probably due to the infiltration into the wood of a product liberated from the parenchyma adjoining infected vessels in which an oxidation process finally brings about a change of colour to brown. In the dark-stained wood the bacteria are dead.

The bacteria are actively motile in the water permeating the walls of the vessels. They do not enter the living cells of the wood nor those of the medullary rays, but these cells may become occupied after they have been killed by infiltration of bacterial toxin from the vessels [9]. Following upon the presence of the bacteria in the wood, parenchyma cells in the vicinity of the ' watermark ' are seen to be deprived of their normal starchy contents. This is a common feature of the disease, for starch gradually disappears from within, and in the vicinity of, the watermarked parts of the wood. Moreover, in the latter there is profuse development of tyloses (Fig. 156), even in vessels which may not have accommodated any bacteria

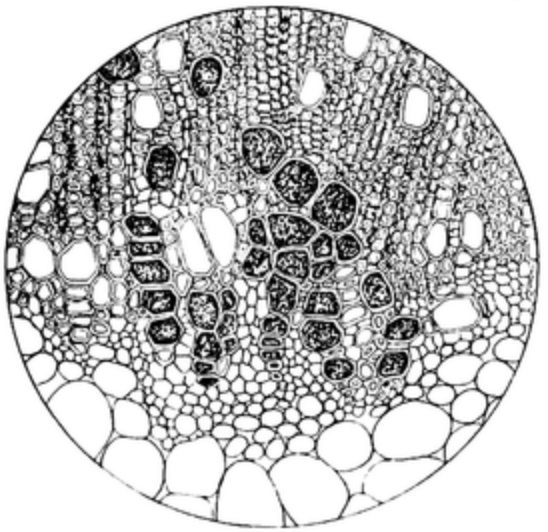


FIG. 413.—'Watermark' of willow. A group of vessels near the pith of a two-year-old twig blocked up with bacteria ; starch is absent from the medullary rays and outer cells of the pith (adapted, from *Oxford Forestry Memoir*, 3, by permission of W. R. Day)

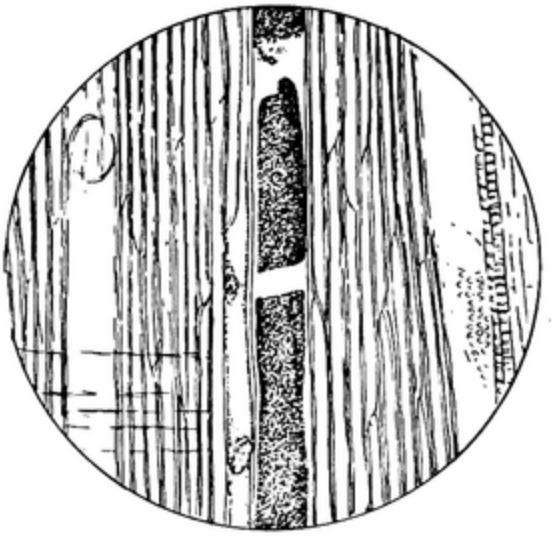


FIG. 414.—Watermark disease. A column of bacteria blocking a vessel. The column is broken in two places (adapted, from *Oxford Forestry Memoir* 3, by permission of W. R. Day)

at all, but dead bacteria may often be seen in vessels occupied by tyloses.

The ultimate effect of watermark disease on the woody elements is not a delignification of the walls, but only a dissolution of the middle lamellae. In consequence, small cracks develop in the wood along these primary walls and there follows in time extensive communication between infected regions of the stem, the cracks sooner or later becoming invaded by bacteria (9). In trees diseased for two years or more, as a result of secondary infections from various bacteria and fungi, the woody cylinder of the larger branches and often of the main stem becomes much split and water-logged, and with changes induced in the wood (which cannot entirely be ascribed to the true parasite) the wood in the last stages of decay emits a very disagreeable odour.

To check watermark disease, first attention should be given to good drainage and cultivation. The trees, though water-loving, do not thrive well when the roots lack aeration. About 20 to 25 ft., it is estimated, should be allowed between the trees, planted preferably in single rows. Stagnant localities and impermeable soils should be avoided.

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Black Canker of Willow, *Physalospora miyabeana* Fukushi

Willow canker, denoted by blackened leaves and small cankers on the stems, is common on numerous species and varieties of the tree in many parts of Britain. The same disease, along with ' scab ' of willow (*Fusicladium saliciperdum*) described below, also occurs in North America, where the two together are reported to cause much greater damage to willows in certain areas, as in New England, than in any parts of Britain (7).

This disease attacks willows in all stages of growth. Infections at the tips of the growing shoots are the most destructive and young branches may suffer from die-back early ; attacks late in the season are not so serious.

First symptoms are usually seen about the end of May, on young rods about two feet high or more. On the leaves the disease starts as reddish-brown or black areas either along the margin or at the tip, spreading later towards the midrib and down the petiole into the stem. If the discoloured area (the colour varies according to the variety of willow attacked) involves the apical half, or so, of the leaf, the discoloured part wilts and bends back at right angles to the remaining part of the lamina, but in other instances entire leaves and petioles are blackened. Infected leaves eventually dry out and shrivel but remain attached to the stem for a long

time before they drop, often leaving behind them, however, the dead stumps of the leaf stalks. Early lesions on the stem are small shield-shaped areas, 2 to 3 cm. long. As these areas develop into cankers they get more and more sunken into the bark and are especially conspicuous at the nodes where they arise, these parts being naturally the first points of invasion of the stem from the affected leaves. The lesions may sometimes completely girdle the stem, and when the cankered tissues finally break open, the wood is often laid bare (Fig. 415). Such deep cankers obviously detract greatly from the appearance and value of the rods, rendering them unsaleable and worthless for basket-making. There is little evidence that any extension of cankers occurs on the rods after cutting or while standing in the pits [6]. As infection begins at the very tip of a shoot, or close behind it, and progresses downwards, the disease is more serious when it starts on the tips of young rods about 12 to 18 inches high, causing the affected part to die back and bend over. But young laterals may suffer in the same way and, if infection at their tips travels down along their whole length, fresh cankers will also arise at the junctions of laterals with the main stem [5].



FIG. 415.—Black canker of willow (*Physalospora miyabeana*). *A*, *B*, *C*, portions of three rods of *Salix triandra* showing cankers on the peeled rods. *D*, shoot of *S. vitellina* var. *basfordiensis* showing lesion on stem caused by infection through leaf; inset, leaf of *S. pentandra* 8 days after inoculation (photos by Nattrass communicated by Barker, *Trans. Brit. Myc. Soc.*)

Black canker of willow was first attributed to *Physalospora* (*Botryosphaeria*) *gregaria* [4], but a study of the fungus causing this disease on *Salix purpurea* var. *angustifolia*, in Japan, showed the species to be *P. miyabeana* [3], a member of the Pyrenomycetes. The organism develops both acervular and perithecial fructifications (Fig. 416). The conidial acervuli appear in early summer, crowded, sometimes joined together, often arranged concentrically on young stem lesions [5], and sometimes, in some varieties of willow, on the leaves as well [2, 3]. From a basal stroma of mycelium within an acervulus, densely arranged conidiophores 30 to 45 by 4 to 6 μ wide arise, producing at their apices unicellular, ellipsoid, slightly curved, pale-pink or hyaline conidia, 12·5 to 21·5 by 4 to 7 μ (average, 17·5 by 6·5 μ) [5]; other dimensions given are, 13·0 to 23·0 by 3·8 to 6·8 μ (mostly, 17·0

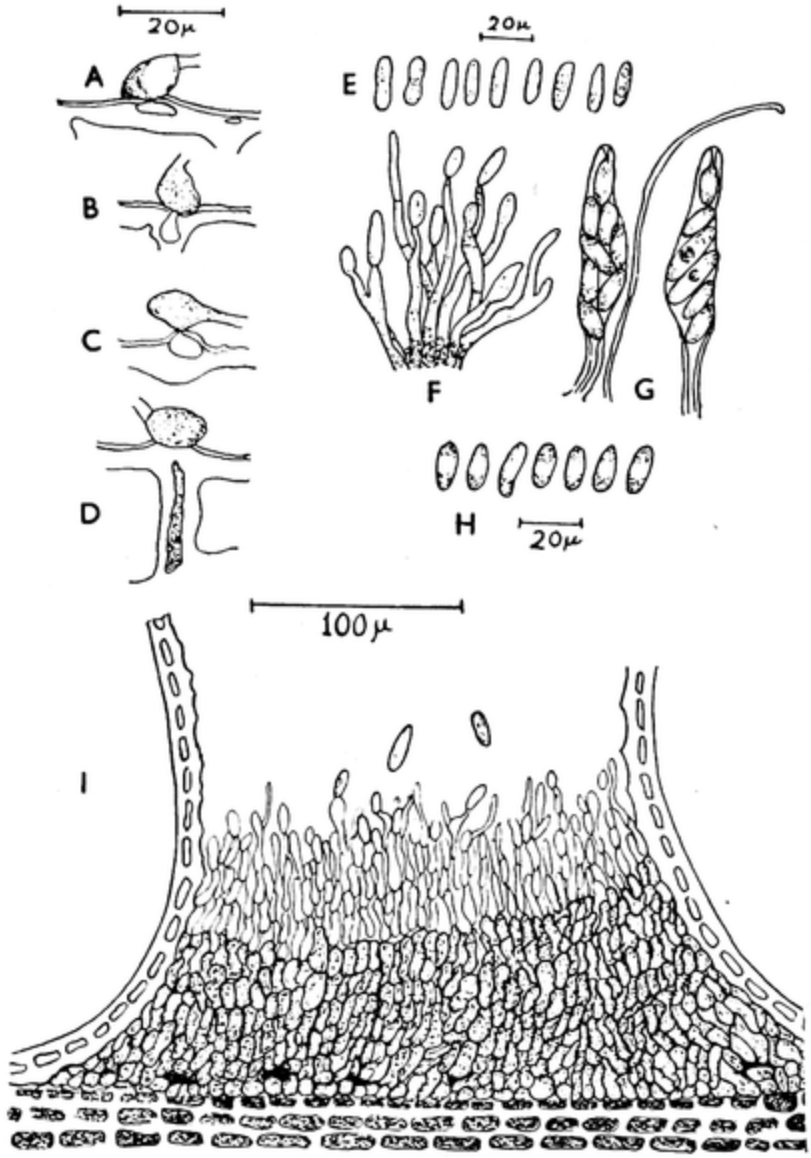


FIG. 416.—*Physalospora miyabeana*. *A–D*, penetration of the outer wall of leaf epidermis. *E*, *F*, conidia and conidiophores. *G*, *H*, asci, paraphysis, and ascospores. *I*, section through an acervulus (after Nattrass, *Trans. Brit. Myc. Soc.*)

to 19·0 by 4·5 to 6·0 μ) [3]. The dark-brown perithecia arise a little later singly or in small groups on the stems on the same lesions as bore acervuli, but the two fructifications are sometimes found together. The perithecia are globose to flask-shaped, 140 to 200 μ in diameter, immersed, with a short protruding neck; the 8-spored numerous asci measure from 65 to 86 by 10 to 11 μ, and are interspersed with slender paraphyses which eventually disappear; the 8 spores are arranged more or less in two rows in the ascus, and are unicellular, hyaline, oblong to ellipsoid, sometimes slightly curved; the spores measure from 14 to 17 by 5 to 6 μ, according to Nattrass [5]; and other given dimensions of the perithecia are from 100 to 170 μ in diameter, asci from 55 to 57 by 11 μ, and the ascospores from 15 to 17 by 5·5 to 7 μ, the asci being interspersed with slender paraphyses 66 by 1·2 μ in diameter [3]. The organism shows considerable variation in culture and some isolates are apparently not so pathogenic as others [5].

The conidia are not adapted for over-wintering, but during the summer they cause widespread secondary infections. They are disseminated in great numbers when the rods, wet with dew or rain, sway in the wind. The spores are splashed on to the leaves or washed down the stem, where they are trapped in the leaf axils. The perithecia survive the winter on cankered rods left uncut for a second-year crop, or on portions of stem left behind after cutting, or on infected twigs left on the ground. Infections are greatly assisted when the trees are grown in dense formation.

Infection of leaves and young growing tips of shoots has been established with or without previous injury, but is much more rapid through wounds. The leaves of *Salix americana* yielded to conidial infection without wounding, but

those of *S. purpurea* only after pricking the cuticle, and the latter species is the only one so far investigated which has shown the acervular fructifications on the leaves [2, 3]. Under damp conditions infections make good progress at a temperature of 25° C. Penetration is cuticular (Fig. 416 A–D) and the mycelium is both inter- and intracellular: progress of infection is rapid, for in 6 days following infection the fungus is found in the petioles, and after 12 days lesions bearing acervuli appear at the stem nodes; and after 36 days perithecia were also developed [5]. From passing down the petioles from leaves to stem, or travelling direct from the growing tip of the stem, the fungus, favoured by warm, close weather in early summer, has been known to work down the whole length of young rods and actually to invade the stool itself [6].

In stem infections the fungus penetrates as far as the wood and causes a brown discoloration which extends beyond the limits of the infected part. The tissues mostly affected are the phloem and cambium, and, when portions of the latter are killed, a depressed area is developed which becomes the seat of canker formation. Moreover, the vessels of the affected wood become blocked with gum and the primary xylem and medullary rays are also discoloured. By the formation of cork below the lesion the progress of a canker may be checked, but at times development of cork is tardy and a canker may grow so as to girdle the stem entirely. From much concentration of infection at the stem nodes, axillary buds also suffer severely and may be killed outright, their tissues becoming clogged with gum.

Osier willows in close planting suffer more than isolated trees. The varieties *Salix alba* and *S. alba* var. *cardinalis* (golden willow) and *S. purpurea* (bitter willow), all valuable basket-making kinds, are very susceptible to black canker. The much-grown *S. triandra*, according to the comparative acreage given to it in Britain, probably suffers heavier losses than all the other varieties put together [6].

Since willow canker is initiated through leaves and shoot tips, considerable control over it can be obtained by spraying the trees with Bordeaux or Burgundy mixture. First application should be made when the rods are about 6 to 9 inches high, subsequent treatments being given at intervals of 3 weeks [5, 6]. All diseased twigs should be cut out during the winter before the dormant cankers disperse their spores. The rods should be cut close to the stools to avoid the over-wintering of cankers on the snags, and all infected rods and debris must be burnt. Winter protection may be given by washing with copper sulphate (4 lb. in 100 gallons of water), or with lime sulphur (1 in 30) applied in the spring [1]. Only healthy sets, free from all traces of canker, should be planted [6].

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Scab of Willow, *Venturia chlorospora* (Ces.) Karst. = *Fusicladium saliciperdum* (Allesch. & Tub.) Tub.

This disease is common on many species of willow in Britain and on the Continent, where it is known as ' bark scorch ' [2, 3, 6, 12, 13]; it is also found in North America [8].

Willow scab attacks the leaves and twigs, causing them, in parts, to turn black, a piebald effect produced by the dark spots on the light-coloured stems being a characteristic feature [2]. There is considerable loss of foliage, often accompanied by a die-back of branches following canker formation on the stem; dead twigs are usually curved or hooked at the tips, and such twigs with their diseased leaves may be of a reddish-brown or black colour, according to the kind of willow attacked. Severe defoliation of affected trees for two or three years in succession is often followed by the death of the tree, and even after the first year's premature loss of foliage the tree seems to have little capacity for making good the loss by adventitious growth and eventually dies from starvation [8]. The dark blotches on the leaves soon become covered over by the conidia of the causal fungus, the spores forming an olive-brown velvet pile which contrasts sharply with the green colour of the healthy leaf. On mature leaves, only a few isolated black spots of variable size appear, but when young leaves are attacked the disease spreads chiefly along the midrib to the base, killing the tissues as it advances, and then passing into the stem at the junction with the leaf, spreading both above and below the node. After the destruction of the lamina, the withered midrib often remains as an extension of the petiole, still attached to the stem.

This disease is caused by *Fusicladium saliciperdum* [1, 4, 14], a fungus closely related to that causing apple scab (p. 732); the perfect ascigerous stage *Venturia chlorospora* [7] (some doubt has been expressed about this designation [12]) has not been found in Britain [3]. Other fungi are also associated secondarily with this disease [3, 13]. The conidia more or less cover the blackened leaf areas, but follow mostly the course of the larger veins and particularly the midrib. The spores, truncate at the base, rounded at the apex, are olive to reddish-brown, 1-septate usually (the basal cell is the larger), and measure from 12 to 25 by 6 to 10 μ; the spores are developed singly at the ends of brownish, closely compacted conidiophores [3, 8]. The perithecia of *V. chlorospora* have been found on dead twigs on the ground, rarely on the tree [11]. The fungus grows slowly in culture and spores may be formed, or the culture may remain almost sterile; in the latter case blackish mats composed of brown, septate hyphae are produced, and these become covered with brownish aerial hyphae which remain sterile [9]; conidia have been observed to develop from single ascospore cultures [9, 11]. The optimum temperature for growth is 20° C.; at 28° and 30°, and at low temperatures of 5° down to −2° C., growth was greatly reduced [9, 11].

Scab of willow survives from season to season under the bark of young twigs in much the same way as that adopted by the fungus of apple scab (p. 735). Conidial pustules on the twigs can survive through a dormant period and are probably the source of origin for primary infections of the leaves in the spring [3]. The spores from the twig pustules are washed down to young leaves in the opening

buds, and thereafter secondary infections are widespread during wet seasons. The outbreaks are especially severe after periods of flood following a series of dry seasons [12]. The disease is favoured by excess of nitrogen and lack of potash in the soil [5, 10, 12].

Different varieties of the willow appear to be variable in their resistance to scab, according to the locality where they are grown [8, 11]. The following programme of spraying with Bordeaux mixture, 4 : 4 : 50, gives good control, (*a*) before the buds open, (*b*) when the leaves first emerge, (*c*) when the leaves are from one- to two-thirds grown, and (*d*) when they are nearly full size [8]. A winter wash of copper sulphate, 4 lb. in 100 gallons of water, or spraying with lime sulphur, 1 in 30, also gives good control [2].

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Dutch Elm Disease, *Ceratostomella ulmi* Buisman

The so-called Dutch elm disease, or elm die-back, probably first reached Europe during the war of 1914–18 [1, 12, 15], but there is nothing whatever to suggest that it originated in Holland. It was first seen in that country in 1919 [20] but probably in France and Belgium earlier [6, 15]; spread through Europe from the Baltic to the Mediterranean and from the Atlantic to the Volga followed. In England it was found in 1927, when it was evidently of some years' standing [2, 25, 26]. America was probably reached on several occasions from 1928 onwards in elm timber from Holland, the most likely origin also of the English infections. The attempt to limit spread in the United States has been more or less successful, but early surveys in England revealed such a condition in East Anglia, the Midlands, and the South-East as would have rendered nugatory any similar efforts. Natural limitation by little-understood ecological factors affecting either the parasite or its beetle vectors (or both) appears to be operative in Great Britain and north of a line from Dorset to the Wash the disease decreases in intensity. There was no report of it from Scotland apart from a single case of its occurrence on a dead log in 1939, until quite recently when it was located on both sides of the Border, near Kelso [2, 3]. Seasonal fluctuations also occur; there was a definite check to its advance in England in 1932 [17], then a renewal and a further decline in 1938–9.

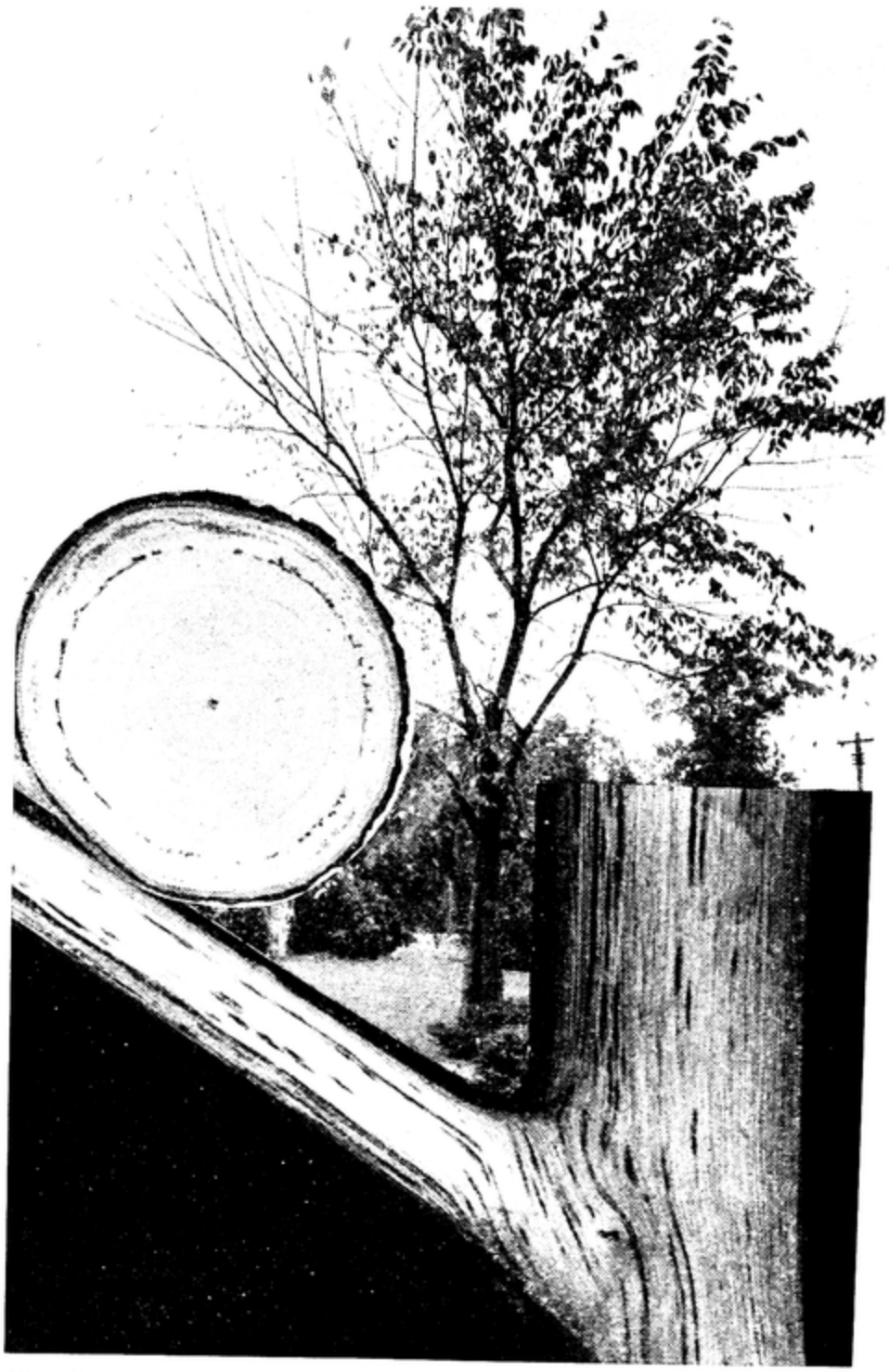


FIG. 417.—Elm disease (*Ceratostomella ulmi*). American elm showing one-sided defoliation of the crown (photo by Curtis May, by permission of Bur. Plant Indus., *U.S. Dept. Agric. Circ.* 392). Insets, cross-section of wood showing rings of discoloration, and peeled branches showing discoloration (photos by Peace)

The most noticeable early symptom is the dying back of a part of the crown of the tree, the leaves on which become yellowed over the whole blade and shrivel (Fig. 417). The tips of the affected twigs (which tend to retain their leaves longest) droop [23], and after they die remain hook-shaped [20, 24]. The progress in the tree is generally slow but varies enormously [12]: it is not often that trees

are killed in a single season; in others death occurs limb by limb and it may be several years before all are affected; other trees, again, apparently recover [17] and a tree attacked one year may not be attacked the next, unless it is visited again by a carrier beetle [2]. A characteristic feature of severe attacks is the growth of numerous adventitious shoots along the trunk or the formation of many stunted twigs with small leaves. It is not until the crown shows some 75 per cent. diseased that the tree can be considered to be doomed. In such trees the holes or tunnels bored by the bark beetles are a sure sign of internal injury (Figs. 80, 81). An early symptom which can easily be missed is the absence, in January, of flower buds from the branches that are later found to be diseased [8]. Internal mischief may be far advanced before any outward signs are visible, so that the only really safe method of diagnosis is the culture of the parasite from wood suspected of infection: a case, indeed, has been reported in which this method revealed infection in half of a stand of apparently healthy trees [4]. Even when gross symptoms are evident, cultural methods may be required to separate this disease from the wilt caused by *Verticillium dahliae*.

Elm die-back is essentially a disease of the vascular system, damaging particularly the functional wood of the current season and interfering with the water-supply to the growing parts. A brown discoloration visible as spots or rings in transverse sections of the spring wood or as elongated streaks of varying length on stripping the bark lengthwise, is a characteristic symptom, though not always present and liable to disappear after death (Fig. 417). This browning may extend as far as the petioles and the roots, and may later affect deeper annual rings by passage of the parasite through the medullary rays [5]. The brown colour is associated with a copious formation of gum and tyloses arising from the wood parenchyma and entering the vessels through the pits in their walls [7]. This makes it difficult to detect the parasite in the tissues. As tyloses also form in adjacent non-infected vessels the interference with the transpiration stream is considerable. Cellulose is not attacked but it has been suggested that the fungus exerts a toxic action on the protoplasts of living cells in advance of the limits of its growth [27].

In 1922 the cause of the disease was determined as *Graphium ulmi*, a coremial member of the *Hyphomycetes* [9, 19]. Ten years later its perfect form, a Pyrenomycete *Ceratostomella ulmi* (*Ophiostoma ulmi*), was described [10]. Mycelial conidia resembling those of the Hyphomycete genus *Cephalosporium* and a yeast-like budding stage arising from these are also produced (Fig. 418 M).

The mycelium consists of rather sparse, recumbent hyphae growing readily in culture in which they are 4 to 6 μ in breadth while sometimes less than 1 μ across in the vessels. The *Cephalosporium* conidia (Fig. 418 A) develop at the tips of short, often verticillate stalks, on which they form mucilaginous white or yellow heads; they may be from 4 to 10 by 2 to 3·5 μ in diameter and germinate either by germ-tubes forming a mycelium with similar spores, or by budding out in enormous quantity yeast-like cells, averaging 5·7 by 2·6 μ [24].

The coremia of the *Graphium* stage (Fig. 418 B) develop almost exclusively on woody substrata and are found in nature in protected places such as cracks in the bark or galleries

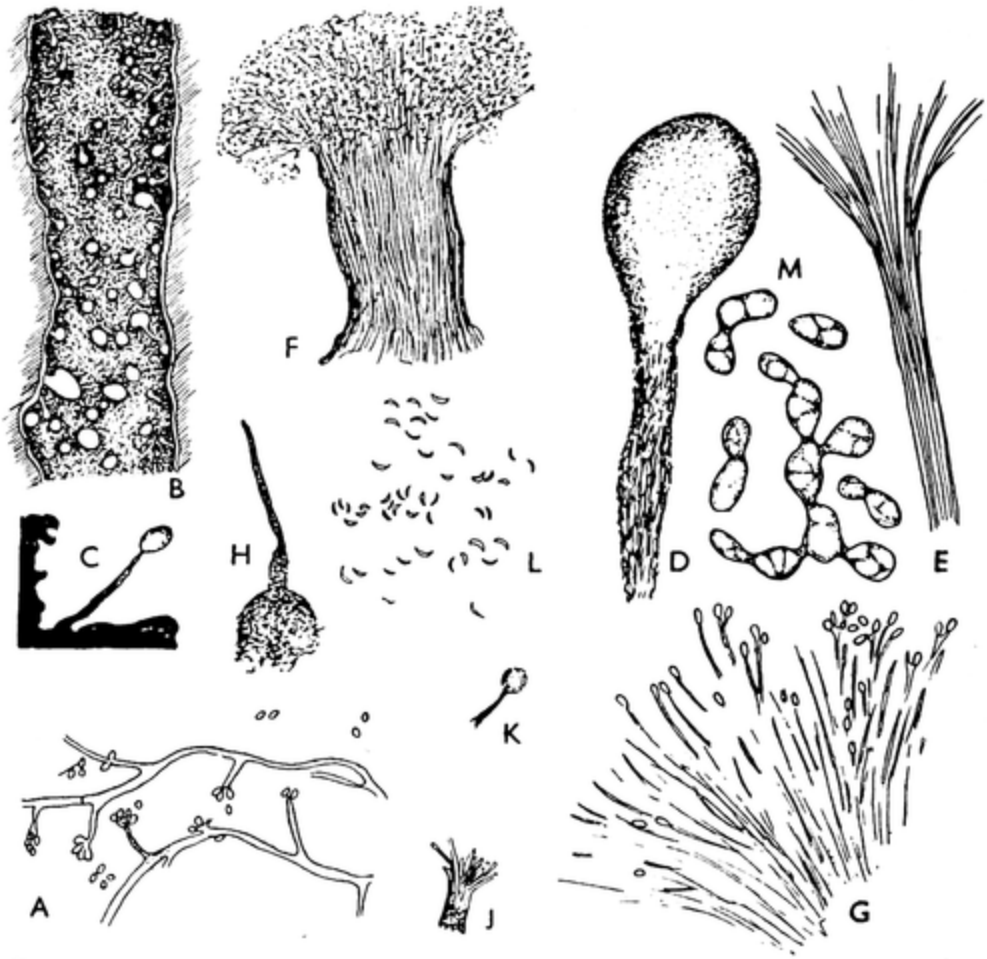


FIG. 418.—*Ceratostomella ulmi*. *A*, mycelium producing the *Cephalosporium* stage. *B*, coremia of the *Graphium* stage, growing in a tunnel in the wood, made by *Hylurogopinus*. *C*, an isolated coremium. *D*, a viscid mass of spores at top of coremium. *E*, the coremial hyphae. *F*, a short, thick coremium. *G*, coremium hyphae sporing. *H*, a perithecium of *C. ulmi*. *J*, the fimbriated ostiole of a perithecium. *K*, ascospores expelled in a viscid drop at ostiole. *L*, ascospores. *M*, yeast-like cells found in the sap in the vessels, and in sap displaced from naturally infected trees (*A–L*, after Clinton & McCormick, *Conn. Agric. Exp. Stn. Bull.* (adapted); *M* (× 1550), after Banfield, *J. Agric. Res.*)

bored by beetles (Fig. 80 A). They average about $\frac{1}{16}$ inch high, have a solid unbranched stalk, dark below and whitish above, and bear at the tip a dense brush of branched conidiophores, about 30 by 24 μ wide, on which the pear-shaped unicellular conidia, 3·3 to 4·8 by 1·7 to 2·1 μ, are agglomerated into a white or yellowish glistening head about 350 μ in diameter. Six physiologic races are reported, showing variability in temperature relations during the germination of the coremial spores [21A].

The perithecia (Fig. 418 H) occur in nature in the borer galleries [22], or below the bark and on exposed surfaces of infected wood. They are round, dark, about 105 to 135 μ in diameter, and have an elongated neck up to 350 μ in length by 38 (below) to 10 (near the tip) broad. The asci are diffluent and embedded in mucilage ; they contain about 8 spores, each shaped like a segment of an orange and measuring 4·5 to 6·0 by 1·5 μ (Fig. 418 L). The species is heterothallic and sclerotia occur [12] which may be aborted perithecia that have not been diploidised by a compatible strain.

The mycelium grows over a temperature range of 8·5° to 34° C. (optimum in culture 25°). High moisture content of the substratum is required for the development of coremia and perithecia. The *Graphium* spores withstand several weeks of drying but

none of the spore-forms is suited for air-borne dissemination [11, 14]. Spores washed down into the soil have not been found to infect unwounded roots [21].

Natural infection usually takes place in the upper part of the tree, as a result of attack by certain insects which carry the spores in and on their bodies. Infection through the roots to neighbouring trees by root unions is also possible but not common [2]; infection of the American elm readily occurred when the spores were placed in the soil in contact with wounded roots [21]. In Europe the chief vectors are the small bark beetles *Scolytus scolytus* and *S. multistriatus* (Fig. 80 B, C), while the latter, together with *Hylurogopinus rufipes* and *Saperda tridentata*, are amongst those implicated in the United States [13, 16]. The female *Scolytus* beetles excavate egg-laying galleries in the inner bark and sapwood and the emerging larvae penetrate farther into the sapwood. *Scolytus* has two generations in the year, one in May hatching out in late May and June, and the other in August to September not emerging from the larval stage until the spring [23]. In emerging they are liable to pick up spores from the tunnels and under the bark of infected branches and they have been repeatedly found carrying infection to the succulent twigs of healthy trees, where they feed on the bark and outer wood. These beetles become rarer north of a line joining the Mersey to the Humber and their range in Scotland is not known but roughly the range of the vectors appears to be in correspondence with the distribution of the disease in Great Britain.

The disease is restricted to the genus Ulmus and the closely allied Japanese *Zelkova keaki*. All species and varieties of elms ordinarily grown in Britain are susceptible, though the Jersey elm, *U. stricta wheatleyi*, suffers relatively little injury beyond the loss of individual small branches or twigs [3, 18]; the commonest English and American species, *U. campestris* and *U. americana*, are very susceptible. The search for resistant forms and the hybridisation and selection of promising types have been actively carried out in Holland, Italy, and the United States. Varieties of *U. foliacea* and the Asiatic dwarf elm, *U. pumila*, have shown a considerable degree of resistance, but the latter are too small to replace the western species and do not do well in England. The wych elm (*U. glabra*) is the most susceptible of our native species; the Cornish elm (*U. stricta*), the English elm (*U. procera*), and the plot elm (*U. plotii*) all appear to be comparatively resistant [16a]. The best variety produced in Holland is *U. foliacea* var. *Christine Buisman*, a selection from a Spanish elm. Immunity does not seem to be found in any species and it is too early yet to attempt to assess the prospect of breeding large resistant types that could replace the many fine trees found in England, or to gauge the possibilities of survival of existing elms in sufficient numbers to preserve one of the stateliest features of the English landscape.

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Oak Mildew, *Microsphaera alphitoides* Griff. & Maubl.

Of recent years mildew of oak has proved to be an increasing source of trouble in the raising of young oak in Britain. Though its attacks are most serious on nursery stock and oak coppice, it occurs on trees of all ages. Beech and sweet chestnut are also susceptible to the same disease but, so far, there are no reports of its occurrence on the latter in Britain [39]. The trouble commonly follows upon the defoliation of the trees by caterpillars and, while established trees may be able to renew their leaves after one or two successive attacks, a second or a third defoliation so weakens the trees that they can no longer resist attacks from mildew [21, 33]. In the same way, a second crop of foliage is very liable to mildew, after the first lot has been killed by frost.

The disease is believed to have made its first appearance in 1907, in Portugal [11], and two years later had spread practically throughout the whole of Europe and quite soon after into Asia [37]. It was first recorded in Britain in 1908 [7]. The mildew is not so serious on the sessile oak (*Quercus sessiliflora*) as on the pedunculate oak (*Q. pedunculata*) [24] (though in some localities the latter is highly susceptible [32]), partly, perhaps, because of the relative immunity of this variety from caterpillar attack and, therefore, from mildew, but the seedlings of both are about equally susceptible, and both are attacked in coppice ; and it is on coppice oak that mildew does the greatest amount of harm [8].

Oak mildew is caused by the fungus *Microsphaera alphitoides* Griff. & Maubl. [2, 12] (*Erysiphaceae*), which covers the leaves and young shoots with its whitish conidial fructifications to such an extent that the trees look as if dusted over with flour. Early symptoms on the leaves consist of small cinnamon-coloured spots which gradually spread over the entire lamina. These primary infections

usually occur early in May, and secondary infections occurring about the middle of June account for a prolific production of conidia. The latter become less and less during July and the white, powdery effect is not then so pronounced. Eventually the leaves curl up at the edges, turn brown, and fall off prematurely. Brown patches of dead epidermis may also appear on infected twigs, and young shoots so affected fail to ripen properly and in consequence are usually killed off by the frosts of the following winter [37]. The fungus is believed to survive the winter in buds which had contracted infection during the previous season [38, 39]. When such buds break into leaf the reviving mycelium gives rise to conidia (or *oidia*, as they are sometimes called) which infect the young leaves as they unfold. Affected buds are usually seen at the base of the previous year's shoots, the upper portions of which, as above stated, have already been killed off by frost and mildew.

The mycelium of oak mildew is entirely superficial on the host ; the hyaline, septated hyphae are about 5 μ in diameter. For a long time, only the white, oidial or conidial stage of the fungus was known, but the perfect, cleistocarpic stage attributed to the American species *Microsphaera quercina* (Schw.) Burr. was discovered in France in 1911, on moribund leaves of the sessile oak [1]. The conidia are developed mostly on the upper, fewer on the lower surface of the leaves, in long chains, from erect, septated conidiophores 50 to 115 μ long ; the oval-shaped conidia measure, on an average, 29 by 18 μ [31]. The cleistocarps, though found in many parts of Europe [4, 26, 30, 36], have only lately been seen in Britain [32 a]. These were found on leaves of *Quercus robur*, in 1945 at Bricketwood, Hertfordshire, and in 1947 at Aberystwyth [15 a] and agree closely with the species *Microsphaera alphitoides* (Griff. & Maubl.) [12] ; the cleistocarps with twenty to twenty-four appendages measured from 180 to 200 μ in diameter ; the ascospores ranged from 18 to 24 by 6 to 13 μ. It is interesting to note that the type collection of *M. alphitoides* is actually the material collected in south-west France and identified by the finders [1] with the American species *M. quercina* (Schw.) Burr., under which name the perfect stage of oak mildew has long been familiar in Europe and America. In this American species the cleistocarps are reported to arise in groups on fading or dead leaves still hanging on the trees, occurring as many as 40 to 50 together, on the upper or under side [5], the more mature cleistocarps towards the centre being black and the outer, immature ones yellow-brown in colour mostly on the margin of a group [19, 25]. In 1918 cleistocarps were found in Macedonia on bushes of *Q. conferta* at an elevation of 1000 feet, on a dry hillside fully exposed to the sun. The fructifications are apparently formed under dry conditions, when transpiration of the fungus is excessive in relation to the supply of food obtained from the host [18, 35, 37]. The fungus is believed to be heterothallic [29], and it is probable that sexually compatible strains necessary for their development are very rare in Britain ; but it is also suggested that the mean temperature and humidity relations are unfavourable to their production in this and other countries [14]. The spherical cleistocarps attributed to *M. quercina* vary from 100 to 150 μ in diameter, and are furnished with about 15 to 20 spreading, dichotomously branched appendages ; asci, from 1- to 8-spored, are numerous, ovoid, 60 by 30 μ ; the ellipsoid ascospores measure from 22 to 30 by 12 μ [12, 19]. There can now be little doubt, however, that the designation *M. alphitoides* is the correct one for the fungus of oak mildew in Europe. The ascospores have not been observed to germinate, and the cleistocarps appear to play no prominent part in the over-wintering of the fungus [34] ; but in Moscow, in 1924, *Q. pedunculata* was successfully inoculated with the spores from over-wintered cleistocarps on fallen leaves [6]. Apparently,

these fructifications, like the cleistocarps of other mildews, are only detachable from the host after being wetted, and, after being conveyed to a new substratum as the water evaporates, become firmly fixed thereto by the appendages [40]; this is contrary to the view that the separation from the original host is effected by the appendages [22, 23].

As already stated, the organism survives the winter in the form of a resting mycelium within the buds. But some authors report the occurrence of thick-walled chlamydospores, 6 to 12 μ in diameter, on infected leaves, capable of passing through the winter on the fallen leaves, on the ground. The chlamydospores are believed to initiate the infections of the opening buds in the spring [27, 28, 31], but these spores have not been found in Britain.

When infected buds open in the spring the rejuvenated mycelium early produces conidia which attack the young emergent leaves. Possibly only a few infected buds may be present on a small number of trees, but once conidia begin to be formed, fresh infections from these wind-borne spores are widespread. Infection takes place only when the leaves are young, before a thick cuticle is developed and at a time when the leaf possesses a high water content [37]. Primary infections occur on the under surface of the leaf, but subsequent infections may take place at either surface, so that the superficial mycelium may eventually cover the entire lamina. The fungus appears to vary considerably in respect of temperature relations, due possibly to the existence of different strains, for the optimum temperature for conidial germination is given by one author as being between 24° and 32° C., with fungal growth increasing steadily with rise in atmospheric humidity [10], while another states the optimum to lie between 18° and 20° C., germination being reduced to a half at 25° and practically ceasing at 30° C. [3]. Penetration of the epidermis is direct and is immediately followed by the expansion of the infection hypha in the epidermal cell to form an oval-shaped haustorium. The entered cell as well as a number of unentered epidermal cells in its vicinity turn brown, and the discoloration may start even before actual penetration, indicating that a toxic substance is apparently given out by the germ-tube as soon as it makes contact with the leaf. There is no further penetration beyond the epidermis, and the massive, white mycelium which develops is kept in position on the leaf entirely by anchorage of its numerous haustoria in the epidermis, and by the close contact which the rapidly interweaving mycelium makes with the leaf. The tips of young twigs, still uncovered by cork, may become infected by conidia in the same way, and many young branches develop such a dense covering of mildew that they are overwhelmed and killed. It is at this time, too, that young buds developing in the axils of mildewed leaves contract infection. When the bud scales are, as yet, soft and imperfectly closed, mycelium, and possibly conidia, from the attendant leaves enter through the unprotected parts and infection becomes established in the softer tissues of the scales and also, later, between the leaf primordia, there to lie dormant until the spring. As the scale leaves become toughened and hardened to meet the winter, it is interesting to note that the dormant mycelium in them is not furnished with haustoria as in the case of vegetative mycelium exposed on the leaves, and so it remains inactive but secure from harm, protected within the bud. When it revives in the spring at the opening of the buds, conidia are early produced and primary infections are started anew [39].

A remarkable phenomenon frequently found in association with the mycelium of oak mildew is the presence of another fungus, *Cicinnobolus*, which actually preys upon the mildew to such an extent that it forms its dark-coloured pycnidial fructifications, frequently in abundance, on mildewed oak leaves. This intruder is also parasitic on other members of the *Erysiphaceae* (p. 646), and it would be of interest to know how far it is effective in suppressing the activities of the mildew on the plant host.

It has been established that varieties of the oak which break into leaf comparatively late in the season are more prone to be attacked by caterpillars than early leafing kinds, and are therefore more subject to mildew. An early maturing variety of the susceptible *Q. pedunculata* was found in the forest of Vierzon [20] to suffer much less in this respect than a later leafing variety of that species ; and if this valuable oak is to be preserved it is clearly desirable to improve upon it, by selection, until an early maturing variety becomes established. In Britain the sessile oak is less susceptible than the pedunculate, and the Turkey oak (*Q. cerris*) is immune, but not in its native country, south Austria [39]. In Belgium the varieties *Q. macrocarpa*, *Q. nigra*, *Q. phellos*, and *Q. palustris* are resistant [3].

Where practicable, oaks should be planted in mixed wood with other non-susceptible trees ; they are then not so liable to the ravages of caterpillars as they are in pure stands, and therefore not so prone to fungus attack. Suitable ' mixtures ' are oak-ash, or oak-ash-elm-hornbeam, or oak-alder-ash-aspen, according to locality [15]. Regeneration from sucker shoots, so liable to the mildew, should not be encouraged, and young plantations should be allowed to grow forward into high forest [9]. Coppice oak is especially liable to mildew [8].

For the protection of young seedlings, spraying should be done before June (the critical period of infection) with colloidal sulphur, 2 to 4 lb. per 100 gallons ; three or more applications may be found necessary [13, 17, 39]. The value of sulphur treatment against mildew may be noted from the fact that oak trees in Austria, in the vicinity of factories emitting sulphurous fumes, remained perfectly free from mildew [16].

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Leaf Blotch of Sycamore, *Rhytisma acerinum* (Pers.) Fries

Black blotches resembling tar spots are common on leaves of the sycamore from midsummer to leaf fall, and persist on the fallen leaves throughout the winter. The spots are far more numerous on leaves of the lower branches of the crown than on those higher up, but the topmost leaves of the tallest trees do not escape infection. They do little harm to full-grown trees unless successive infections are heavy, but young trees and seedlings are adversely affected through impairment of the leaf functions [8]. The disease is caused by *Rhytisma acerinum*, a member of the Phacidiales group of the Discomycetes [3, 4, 5, 6].

First signs of infection in June or July consist of small pale spots which soon turn yellow when about 1 to 2 mm. in diameter, and some four or five days later each spot becomes speckled with minute black dots which join together until the spot is uniformly black save for a narrow border still remaining yellow (Fig. 419 A) [1]. The blackened spot consists of a portion of the upper epidermis where the fungus inside a group of the upper epidermal cells has caused them to split in half, parallel with the leaf surface, and within the split epidermis the fungus builds up, first, the spermagonial and, later, the apothecial fructifications (Fig. 420).

Prior to the appearance of spermagonia the surface of the blackened spot is perfectly smooth but soon develops a number of small round or oval pimples towards the centre where these fructifications are being formed (Fig. 29). Each pimple develops a raised margin and, later, one or two tiny ostioles at the top, through which myriads of spermatia exude to the surface in a thin milky film. The spermatia are very narrow, bacilliform, slightly curved, hyaline, and uninucleate, measuring 6 by 1 μ; they have not been observed to germinate (Fig. 29 D). Spermagonia continue to be formed from June till August and, thereafter, the black stroma, still increasing in area, forms again a smooth, blackened region surrounding the defunct spermagonia. The spots do not expand indefinitely

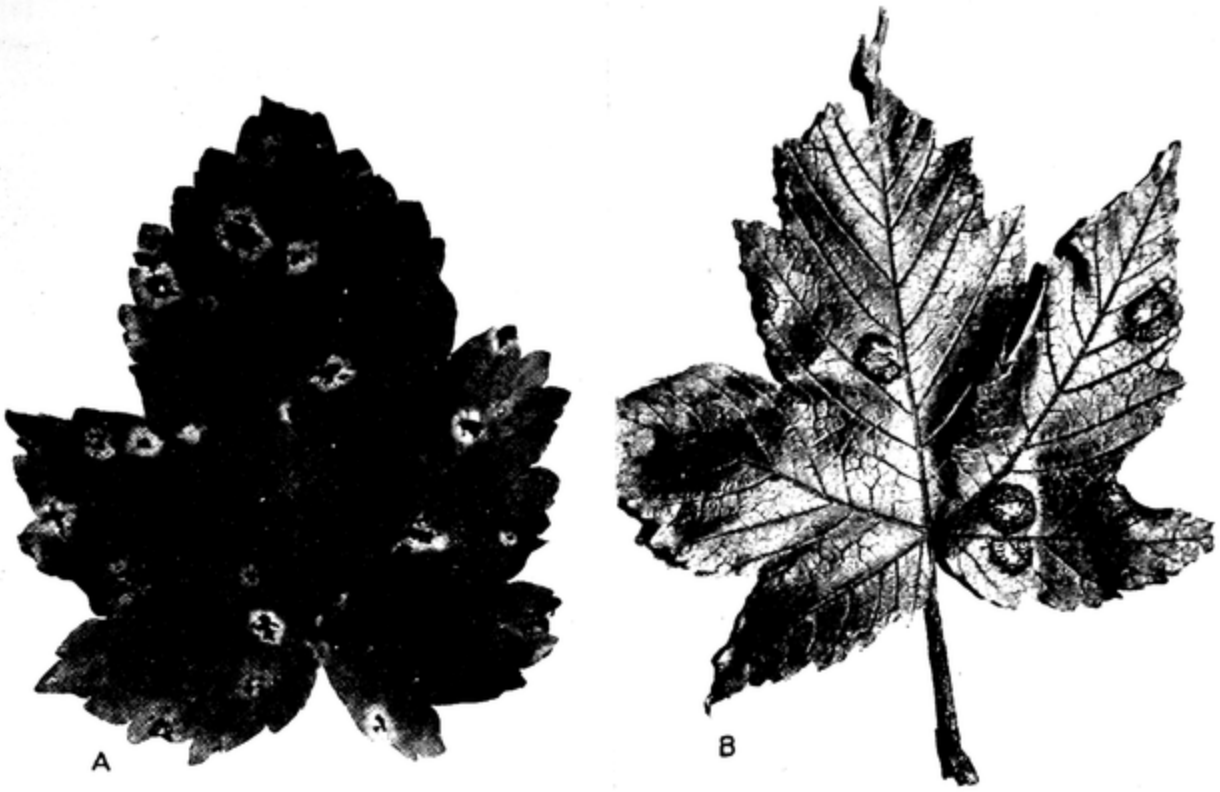


FIG. 419.—Leaf spot of sycamore (*Rhytisma acerinum*). *A*, early infection (July) showing black spots surrounded by a yellow margin; spermagonia will soon appear on these stromata. *B*, a leaf on the ground (January), well protected in a layer of the leaf humus, showing almost mature apothecia around the margin of the spots

and are usually checked when about 1·5 cm. in diameter, but infected areas often join together to form larger spots of diverse shape.

The extended zone around the spermagonia soon becomes wrinkled and numerous apothecia develop within the split epidermis in this area (Fig. 29 E). Apothecia may also arise within old spermagonia but occur mostly in the extended zone around them. This zone during development becomes divided up by solid partitions of fungus pseudoparenchyma, which radiate roughly from the centre of the spot like the spokes of a wheel, the hub representing the spermagonial region, and for the accommodation of the spoke-like partitions the fungus has meanwhile increased the tangential splitting of the epidermis all round. Each roofed space between the spokes accommodates an apothecium and, in preparation for the time when these fructifications have to spend the winter in the fallen leaves on the ground, both the roof and the floor of the apothecial cavities are greatly thickened by closely woven fungal pseudoparenchyma which is further reinforced by much deposit of black substance cementing the cells firmly together (Figs. 23, 420 B). Leaf fall is now imminent, but before it takes place the hymenium is so far developed in the apothecia that groups of ascogenous cells arising at frequent intervals along the whole length of the elongated apothecia have already been developed. The leaves are cast off at the usual time and in the usual manner, for the fungus interferes in no way with absciss formation and never extends in the lamina much beyond the limits of the black spots. If the fallen leaves are heaped up or other-

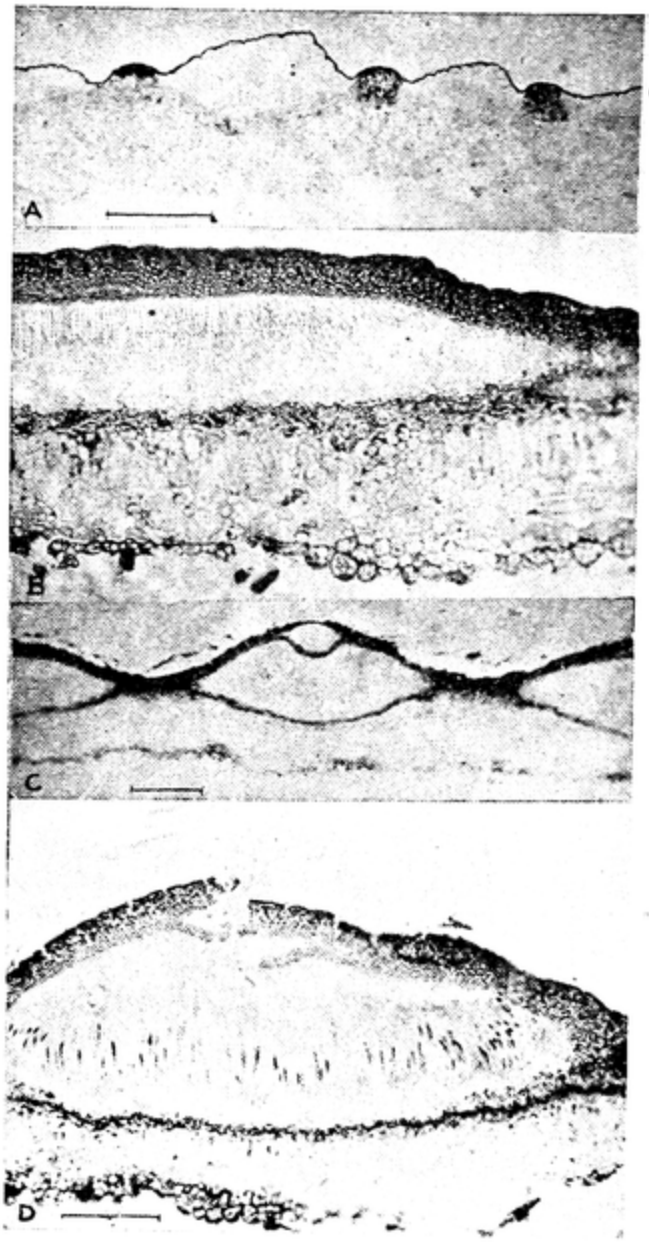


FIG. 420.—*Rhytisma acerinum*. *A*, transverse section of portion of a spermagonial stroma; note the split epidermal layer, its upper half with the cuticle carried up; the short spermatiophores, forming a dense layer, produce an enormous mass of minute spermatia; limits of a spermagonium indicated by the black pillars. *B*, portion of transverse section of leaf, after production of spermagonia is over, showing mycelium in all cells of leaf; note hypothecium with vertical hyphae which are building up the roof (epithecium) of the future apothecium. *C*, transverse section of almost mature apothecia, in leaves on the ground. *D*, a mature apothecium, the roof split open; the hypothecium shows asci in all stages of development of ascospores. (See also Figs. 5, 23, 29.) (The lines on the prints = 100 μ.) (From microphotos by Jones)

wise protected from desiccation, the stromata survive the most rigorous conditions of winter and apothecia may be found as early as January (Figs. 419 B; 420 C) to have well-differentiated asci and paraphyses [1].

The asci are uninucleate for a long time and continue to develop up to the end of March or later. The stromata do not survive in dry, wind-swept leaves, but tide the winter successfully in leaves on the lee side of a hedge, in damp crevices of walls, or deep clefts in tree crotches where moisture collects, or wherever there is a thick carpet of leaf mould. An apothecium dehisces along its entire median ridge (Figs. 29 F, G; 420 C, D) and under humid conditions spore dispersal takes place over a long period and the same stromata may continue to shoot spores intermittently, if moistened. The club-shaped, 8-spored asci are 120 to 130 by 9 to 10 μ; the ascospores are unicellular, uninucleate, filiform, and hyaline, and furnished with a thick, firm gelatinous sheath [2]. On a culture-medium of complex composition the fungus developed stromata and apothecia at room temperature after being exposed to low winter temperature [7].

Infections at the lower surface of the leaves are stomatal and begin soon after the unfolding of the buds. The ascospores become attached to the cuticle by their sticky gelatinous sheaths and the single germ-tubes entering the stomata branch profusely to form a septated, hyaline mycelium which is entirely intracellular (Fig. 5) except when the hyphae cross the intercellular spaces of the mesophyll to pass from one cell to another. The host tissues occupied by the fungus retain their shape to the end and the fungus-filled mesophyll and

lower epidermis, much reinforced with black substance, form a firm foundation to the stromata during all stages of development.

The disease can be entirely prevented if the fallen leaves are destroyed in the autumn before the stromata are matured, but this is hardly practicable unless the trees are few, and there is no certainty that they may not again become infected from ascospores carried by wind from distant trees. Spraying with Burgundy mixture is recommended for young trees [8].

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Root Rot of Larch, *Armillaria mellea* (Fr.) Quél.

Root rot is a severe affection of coniferous trees. It is caused mainly by *Armillaria mellea*, the well-known ' honey agaric ', which is no doubt the most destructive of all fungi that attack the underground parts of trees and other plants. Its hosts are numerous and range from herbs to forest trees. Moreover, it can thrive for indefinite periods on the remains of these hosts in the soil as a saprophyte. Herbs and young trees may be killed within a comparatively short time of infection, while woodland and forest trees may harbour the fungus for years before it finally destroys them.

Armillaria is a native of woodland soils, and its occurrence in cultivated land can always be traced to a previous occupation at some time or other by trees. It is a most virulent parasite of coniferous trees and is also destructive to deciduous, broad-leaved trees and fruit trees. After the death of these hosts it thrives on the stumps left in the ground and, if these remains are removed, even the smallest bits of infected root or stem left behind are often sufficient to keep the fungus alive in the soil. The organism cannot maintain itself if severed from its natural food base.

Although the parasitic activities of *A. mellea* are undoubted, some believe that it becomes aggressive only when the living plant is in a weakened condition such as would follow upon drought, frost, or human interference with natural conditions [6]. Thus, in a wood of closed canopy, of beech for instance, which maintains the ground in a dry condition, the fungus is not common, but in an open wood, such as one of Scots pine which allows considerable rain to reach the roots, the conditions are favourable to infection ; again, it is recorded that in a mixed wood of beech, pine, spruce, and silver fir the fungus became parasitic only after the densely leaved beech had been removed : the gaps left behind, becoming weed-ridden and by conserving moisture, enabled the fungus to attack the conifers, and it was significant that the shallow-rooted spruces were the first to be affected, followed by the deeper rooted pines, and finally the silver firs [21].

Leading an active parasitic life *Armillaria* causes a type of dry root rot, and its victims include, among conifers, pine, spruce, larch, deodar, araucaria, Douglas fir, etc. ; among broad-leaved trees, oak, beech, chestnut, walnut, citrus, and others ; in the orchard it causes root rot of apple, pear, medlar, plum, cherry, nectarine, black currant, raspberry, gooseberry, and strawberry ; it is reported also to have attacked potatoes in Scotland, North America, Australia, and Japan ; in Nyasaland it was recorded in 1927 to have destroyed tea plantations where infection was traced to old tree stumps (1, 8, 11, 12, 13, 14, 16, 17, 25, 26).

It is difficult to determine what factors, ecological or nutritional, encourage *Armillaria* to adopt a parasitic habit. Possibly, as long as the fungus derives its food from humus, or other organic and mineral constituents of the soil, as a saprophyte, the trees are not attacked, but if the food in the soil falls short of its requirements the fungus then attacks the living trees. It is not known to what extent *Armillaria* can compete in the soil with other fungi and bacteria, but should its nutrition suffer from the greater success of its competitors, then such conditions would perhaps induce it to seek its food from the living tree. It is suggested, again, that *A. mellea* possesses physiologic races which differ in pathogenicity, and that where the weakly parasitic or saprophytic races exist, the trees escape (4), while in other places where virulent races of the organism are present, the trees are attacked (3).

The life-history of this fungus has been fully described by Hiley (11) in its parasitism of the larch. This and other trees may be infected for many years without showing any apparent signs of ill-health, and the fungus may be present in the roots and trunk for a long time before the familiar honey-coloured sporophores appear around the base of the tree (Figs. 421, 422 A). The larch is not usually attacked before it is about fifteen years old, and may not succumb for many years after infection. The first visible symptom to appear on the crown is a yellowing of the foliage on one or more branches, followed by premature defoliation ; entire branches may be lost, and in an advanced stage of the rot there is usually a copious flow of resin from cracks in the trunk, often running to the ground and forming sticky masses with the dead leaves around the base of the tree.

Having penetrated into the roots, the fungus travels for the most part under the bark into the trunk but does not extend into the crown. In stages of advanced decay large portions of bark may be pulled off the trunk, exposing to view thick wefts or cords of stringy mycelium arranged lengthwise in parallel with the axis of the tree, several cords being joined together irregularly by cross strands of the fungus, in ladder fashion, and at other points interlacing or anastomosing to form still thicker strands, the whole system being cream-coloured when freshly uncovered but turning brown or black with exposure to the air, and then resembling a flat irregular network of black shoe-laces joined together by cross pieces of narrow diameter (Fig. 422 B). These strands are the sub-cortical rhizomorphs which extend from under the bark of the roots often to a considerable height up the trunk. If the bark is stripped off higher and higher the rhizomorphs are seen to lose their stringy form, spreading out gradually in the form of white sheets of mycelium extending like fans over the sappy surface of the wood (3). Again, if the soil around the roots is carefully cleared away the rhizomorphs may be followed

out from the roots into the soil, but unlike the flat shoe-lace form, they are round, cord-like growths of a dirty white colour, and these soil rhizomorphs may often be traced to their origin from a decayed stump at a considerable distance away [1, 7].

At an advanced stage of the rot, the fungus having penetrated into the woody tissues, a cross-cut of the felled tree shows the diseased wood to be marked by black lines of discoloration cutting off, here and there, triangular or roughly circular portions of the wood, more or less filled with fungus mycelium. These enclosed masses of fungus-filled wood delimited by the black zones are called xylostromata (or pseudosclerotia [2]), and have already been described (Figs. 20, 21) (see Chapter I, p. 17).

It is not clear what the exact conditions are which lead to the appearance of the sporophores. These fructifications may arise singly, but occur more frequently in clusters around the base of the tree or diseased stump, or near by on the ground but in all cases they are attached to rhizomorphs (Fig. 421) or to mycelium beneath the bark ; they are rarely found on the trunk above ground level. The mushroom-like sporophores vary from 7·5 to 15 cm. in height ; the stalks are of a dull orange or brown colour, sometimes black at the base, with a ring or ' armilla ' about three-fourths of the way up ; the cap, measuring from 5 to 15 cm. in diameter, is tawny or honey-coloured, with dark-brown scurfy scales covering its raised centre ; the gills are white, and the hyaline basidiospores measure from 8 to 9 by 5 to 6 μ.

The spores are easily grown on a variety of media such as plum decoction, sterilised manure, and media rich in carbohydrate, starch or glucose preferably, with peptone for nitrogen [8, 11, 16, 20]. Suitable conditions are a reaction of *p*H 5·0 and a temperature range of 18° to 25° C. Mycelial growth, at first white, later dark-brown, becomes heaped up at the centre of the colony to form a dense mass like a sclerotium, from the margin of which clumps of mycelium or stringy branches resembling small rhizomorphs are produced. The surface of the sclerotial body is formed of hyphae with dilated terminal cells very similar to the hyphae that fill the tracheids of the wood when a xylostroma is being formed (Fig. 20) [3, 11]. From the manner of its growth on the surface of artificial media and its strict partiality for penetrating well-aerated tissues, such as the cortex, the mycelium of *Armillaria* is strongly aerobic, and were it not for the internal adaptation of the rhizomorphs for storing air (Fig. 19), the fungus would not be able to exploit the deep-seated tissues of the tree [20]. Saltations are frequent in cultures on malt agar.

It is not at all clear what the rôle of the sporophores can be in the spread of root rot amongst living trees in the open [17]. It appears, however, that the spores can grow on dead wood of stumps or fencing posts. In the orchard, on the soil or grass around the trees, there may often be seen white floury deposits of spores dropped from the sporophores, and it is probable that from such centres, by the production of a considerable amount of mycelium from the spore masses, infection may extend through the soil to the roots of living trees [26]. Individual spores are apparently incapable of infecting the plant, and whether they succeed in mass has not been proved. But possibly primary infections are started in this way from massed spores of potentially pathogenic strains. Moreover, it is probable that there are strains of *A. mellea* some of which differ not only in degrees of pathogenicity but also sexually, so that only compatible strains form sporophores and





FIG. 421.—The sporophores of *Armillaria mellea* at the base of a dead yellow birch (photo by permission of Spaulding and Bur. Plant Ind., *U.S. Dept. Agric.* '*Tree Pest*' *Leaflet* 21). The sporophores of *A. mellea* at the base of a recently cut tulip tree showing the fruiting bodies on the path formed from the same infection (photo by Reid). Three sporophores of *A. mellea* attached to a rhizomorph (from Hiley's *Fungal Diseases of the Common Larch*, by permission of Oxford Clarendon Press)

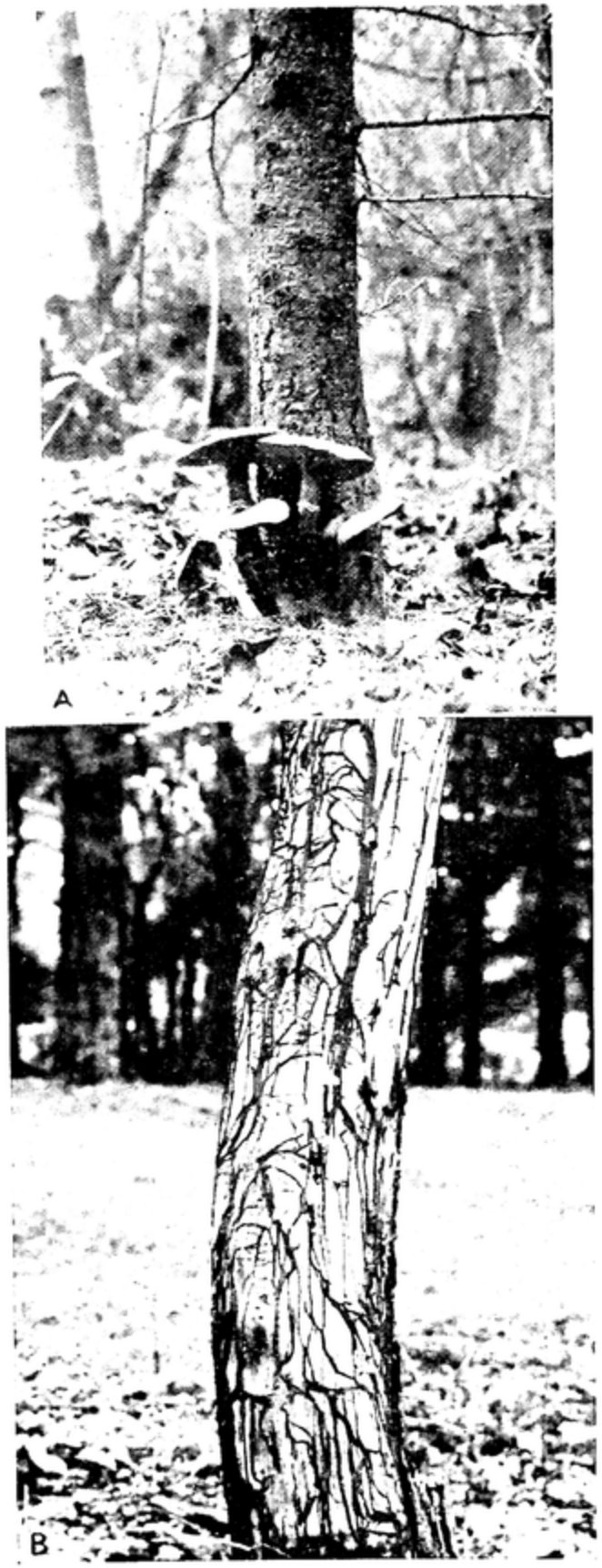


FIG. 422.—*A*, root rot of larch (*Armillaria mellea*). The fructifications growing at the base of a young dead larch (*Principal Decays of Softwoods used in Great Britain*, by permission of H.M.S.O.). *B*, showing the network of the shoe-lace type of rhizomorphs, under bark of a dead pine (*Principal Decays of Softwoods used in Great Britain*, by permission of H.M.S.O.)

some strains may be virulent parasites in a vegetative capacity and yet be incapable of producing fructifications.

Infection of the living tree is effected by the rhizomorphs in the soil. Some authors state that the fungus can only enter through wounds on the root or at the base of the trunk, such as cracks in the bark near soil-level made through the action of frost, boring insects, rodents, or by mechanical injuries during cultivation [9, 21]. Seedlings of Corsican pine in a sand culture inoculated with mycelium of *A. mellea* were attacked at the points of emergence of the lateral roots, each point being the origin of a separate infection [19]. But investigations have established that cork-protected surfaces of root or stem can be penetrated by the rhizomorphs without previous injury [5, 16, 23]. A rhizomorph, as above stated, is a firm cord-like aggregation of hyphae, endowed moreover with apical growth. The tip of one of its numerous branches is not unlike the root tip of a young seedling, but when examined closer the outside consists of somewhat loose thin hyphae extending outwards like frayed string (Fig. 19). Below these superficial loose hyphae there is a ' cortex ' of closely interwoven cells forming a well-consolidated tissue ; the core or ' medulla ' of the rhizomorph is, however, of much looser texture, and as the rhizomorph expands by growth a central cavity is formed in the medulla and this becomes the main aerating system of the rhizomorph. Young rhizomorphs, especially at their tips, give out a sticky fluid which in older parts sets hard like a crust or cement so that the loose superficial hyphae become lost in older rhizomorphs, the firm cortex then becoming the hard rind which by exposure to air turns dark brown or black. When a rhizomorph is about to produce new branches, at numerous points along its length, each branch arises as a smooth-topped dome of hyphae in the inner layers of the ' cortex ' and eventually the young growing dome breaks through the rind of the parent strand [11]. Rhizomorphs in active growth are endowed with a peculiar luminescence which is apparently due to auto-oxidation [18]. Numerous enzymes have been extracted from rhizomorphs [15]. When the free end of a rhizomorph growing through the soil comes into contact with the cork-covered root of a susceptible host, it becomes attached to it by means of the sticky mucilage secreted by the tip, which soon sets hard like cement so that firm contact is made between rhizomorph-tip and the host. The part behind then comes to lie alongside the root, sending out on that side in contact with the root a number of short branches the tips of which become glued to the host in the same way as the apex of the parent rhizomorph. Thus numerous points of contact with, and penetrations of, the root, if successful, are established. Moreover the apex of the parent rhizomorph is still free to proceed with further growth, and to give rise to further side branches, because its original anchorage to the root was secured only by the loose hyphae around and behind the tip, so that the latter is free to grow forward just like the growing point of a shoot. Actual penetration of the cork layers appears to be mainly from mechanical pressure exerted by the tips of the short rhizomorphic branches, each secured in place by the cementing external hyphae, and as pressure is maintained by continued growth of each branch, the cork layers become compressed at each point of contact (cf. Fig. 112). But there is no splitting apart of any of the cork cells, nor of any kind of solvent action on them by the thrusting tips. How the final break-

through is accomplished is not clear, but it is apparent that each rhizomorphic tip is penetrating *as a whole* and there are no signs of its disruption into strands or hyphae that would insinuate themselves between the cork cells. But a significant feature is that there are still some two or three layers of the innermost cork cells, next to the cortex of the root, which are resisting the compression from without and some of the cortex cells immediately below these cork layers are turning brown and apparently becoming plasmolised, the cells appearing to be gradually killed from '*an influence felt below the cork*' [23]. It is difficult to understand how internal plasmosysis can arise from the effect of some diffusible substance from the rhizomorph passing through suberised layers, and possibly the effect of the localised pressure (which may be quite considerable) is to break down the turgor of the affected cortical cells and so release some *influence from within* which finally assists in the complete break-through, for ' as the pressure continues, the suberised walls in direct contact with the rhizomorphic tip are seen to disappear as if acted upon by some dissolving force, and the rhizomorph pushes through into the cells below ' [23].

As soon as the rhizomorph has penetrated the cork it branches profusely within the cortex to form a thick white flaky mass of mycelium, or if penetration goes deeper, the flakes are laid down in the phloem or, deeper still, in the cambium. In any of these regions the spreading fungal growth must naturally become compressed owing to the proximity of the firm woody cylinder and consequently is forced to extend itself fanwise. By means of fine hyphal branches given off at right angles from one or both sides of a fan, the host tissues adjacent become further infected, and it is from the inner surface of a fan, usually of one which had become established in the cambium, that fungal hyphae travelling in a radial direction chiefly through the living parenchyma of the medullary rays are able to penetrate the woody tissues of the tree. The living cells of the medullary rays play a vital part in the distribution and feeding of the fungus as the hyphae proceed to occupy the tracheids, for all the tracheids of the wood are everywhere at one level or another, in contact with the living parenchyma of a medullary ray. Not only are these living cells exploited and invaded, but the fungus, at the expense of being nourished by them, is enabled to proceed from tracheid to tracheid by means of boring through the lignified walls.

In the larch, for a long time after infection, there is comparatively little mycelium within the woody cylinder and little interference with transpiration and the normal functions of the tree, so that many years may pass without any visible signs of disease above ground. When, however, it is seen that the leaves are turning yellow and falling prematurely, with branches here and there becoming denuded of leaves, such symptoms indicate that the woody tissues in root and stem are in progress of occupation by the fungus, but it may still take some years before the xylem towards the base of the tree fails entirely in its supply of water to the head. With the death of the crown, however, decay of the trunk proceeds apace. This advanced stage of the disease has already been referred to when describing the xylostromata in the wood (p. 17). The discoloration of the diseased wood is apparently due to the occurrence of a gummy substance which is of a light colour when first formed but soon darkens to black, especially when exposed

to the air (Fig. 21). In the final stages of the rot great changes occur in the xylostromata, due no doubt to the action of numerous enzymes, and resulting in a disintegration of the wood. When the dead bark finally falls off, the dry, rotted tissues fall apart, some in thin black flakes (from the black zones) and some in clumps, with stringy white strands attached to them and, mostly under the bark, the characteristic shoe-lace rhizomorphs may be found in abundance. *Armillaria* thus kills the tree from the roots up, extending first under the bark and then into the wood of the trunk.

A. mellea in its parasitism demands a high degree of humidity and does not usually attack stored or felled timber except under very damp conditions (3).

Owing to its capacity for existing as a saprophyte *A. mellea* is difficult to eradicate from the soil. But as far as practicable all infected stumps and roots should be dug up and burned. Careful watch should be kept for the sporophores; it is not sufficient to destroy them, but they should be traced to their origin and the connecting rhizomorphs and food sources removed. As this method is very laborious, a frequent practice is to dig trenches around infected stumps deep enough to ensure that no rhizomorphs are left uncut. 'Ring barking' carried out sometime before the trees are felled, is also resorted to as a means of starving the fungus in the roots, thus preventing the flow of carbohydrates; deciduous trees, in general, become depleted of carbohydrates more rapidly if they are allowed to break into leaf before being ringed (17). In some localities 'gassing' of infected stumps and contaminated soil is carried out with carbon bisulphide, and the treatment is said to give better results in soils of low moisture content (24). Iron sulphate sprinkled around the base of infected trees is stated to prevent spore germination and development of mycelium (8).

Broad-leaved trees, in general, are more resistant than conifers to *Armillaria* root rot, but in mixed woods of closed canopy the susceptible conifers are not so liable to be attacked. It is significant that in pure natural stands of coniferous forest *Armillaria* is rare, but if pure stands of conifers are established on the sites of old broad-leaved forest the fungus flourishes luxuriantly (6).

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Heart Rot of Conifers and Other Trees, *Fomes annosus* Fr. (Cooke)

Fomes annosus is the commonest cause of heart rot of the larch in Britain, and is responsible for very heavy losses on certain types of land. The fungus attacks other conifers [4, 6, 9a] as well as various hardwoods [6, 20, 21]. In Denmark, where it is often known as spruce red rot, it causes much injury to Norway spruce (*Picea abies* or *P. excelsa*), Sitka spruce (*P. sitchensis*), and the Weymouth pine (*Pinus strobus*). *Pinus contorta* and the silver fir (*Abies alba*) show an intermediate degree of susceptibility, while Douglas fir (*Pseudotsuga douglasii*), larch (*Larix decidua*), and Scots pine (*P. sylvestris*) are fairly resistant [15]. Scots pine may, however, in some areas, as in south Scotland [2], show severe damage, but in coniferous plantations in Great Britain this tree appears, in general, to suffer less than Norway spruce and European larch [10]. The disease, described below on the larch, is known throughout Europe and North America, and is found also in Australia, India, and northern and eastern Asia.

Heart rot is most destructive to young larches up to 10 years old ; the infected trees turn reddish brown and drop their leaves, death ensuing in a year or two in severe cases. In older trees in the plantation the disease seems to appear when the canopy has become closed [1, 2] ; from 15 to 45 years of age the trees are seldom killed outright, but the infection is tolerated and pursues an insidious course, gradually rotting away the heartwood until the trunk becomes hollow. There are few symptoms, apart from some loss of colour in the foliage, and even the sporophores of the parasite are not often to be found on the larch, so that it may be necessary, in order to judge the extent of the infection, to tap the trunks for a hollow sound or to take a boring from the heartwood.



FIG. 423.—Heart rot of larch (*Fomes annosus*). Base of a larch trunk which has been excavated underneath. The lateral roots show *figured rot*, whilst the tap-root and an anchor-root are completely rotted (Hiley's *Fungal Diseases of the Common Larch*, by permission of Oxford Clarendon Press)

Infection occurs underground and seems to be usually through the tips of the deeper roots which have been killed back from one cause or another [14, 15]. The fungus

enters through the dead tissues and passes upwards to the trunk through the deeper layers of the wood. The lateral, surface roots may be reached by an extension of the central infection from the collar of the tree [12] or by direct attack from the surface soil in which the fungus grows and where it promotes acidity with consequent root-killing [1]. In young trees the surface roots are completely decayed (Fig. 423) and thin sheets of mycelium may be found under the bark or there may be small pustules of dirty white mycelium on the bark. The surface roots of older trees are decayed only in the centre of the woody cylinder and may remain functional for a long time ; in the trunk decay is confined to the heartwood and is at first localised in small groups of tracheids with medullary rays. These become surrounded by a deposit of ' wound gum ', forming a black zone which may for a time isolate the decayed islands of tissue from the sound heartwood beyond [12]. The result is a pitted condition which increases as new islands of decay form, until the wood is honeycombed with cavities. Sometimes the advancing decay is annular owing to multiple invasion from the roots, and solid spikes of hard wood several feet in length are left isolated from a decayed outer ring of heartwood. More usually, however, the whole of the heartwood is involved : the cavities become confluent and a loose, yellow, spongy or fibrous mass remains which may be further broken down by wet rotting due to secondary bacteria and fungi. The sapwood remains sound and functioning, separated from the rotted centre of the trunk by a gummy or resinous zone across which the fungus does not pass.

The mycelium reaches the inner wood through medullary rays in dead roots and enters the tracheids through the simple pits. Here its extension is mainly axial, but the coarser axial hyphae give off fine branches which enter neighbouring tracheids through very fine bore-holes made by enzymes excreted only at the hyphal tip [16]. Dark, thick-walled hyphae with swollen ends accumulate here and there in the heartwood of the trunk, causing a black pitted appearance in section. These have been thought to be centres of enzyme energy, the product of which leads to solution of the middle lamella between the cells and the delignification of the cell walls. Both lignin and carbohydrates are destroyed [18]. No fewer than sixteen enzymes were produced by this fungus isolated from red cedar (*Juniperus virginiana*) attacked in north Carolina [4]. A bleached whitish cellulose residue is left for a time but



FIG. 424.—*Fomes annosus*. Decay in the larch, causing small white pockets of rot, shown in the delignified specks in the decayed wood (*Principal Decays of Softwoods used in Great Britain*, by permission of W. R. Day)

ultimately this is also dissolved. The fungus belongs to the 'white rotting' group of wood destroyers (Fig. 424), attacking all constituents of the wood; it is one in which action on the cellulose is delayed [1]. The wood is left light, dry, and yellowish in colour, often with persistent black spots [12]. Its alkali solubility increases with advancing disintegration [18].



FIG. 425.—*Fomes annosus*. Top, the sporophores on the root and crown, and on lateral roots, the latter probably overlying cavities or rodent burrows in the soil (photo by Rostrup; by permission of P. Spaulding and Bur. Plant. Ind., *U.S. Dept. Agric.* '*Tree Pest*' *Leaflet*; root rots of conifers, No. 18). Below, the sporophores at base of an old pine stump (*Principal Decays of Softwoods used in Great Britain*, by permission of W. R. Day)

Sporophores form more freely on other conifers than on the larch. They develop on the stem after death, either as hoof-shaped brackets at the base of the trunk or as resupinate crusts on the under side of lateral roots overlying cavities in the soil due to surface wash or the burrows of rodents (Fig. 425). The upper surface is reddish-brown, the lower, white or biscuit-coloured and porous. The pores are lined with a hymenium of usually 4-spored basidia, 30 to 40 by 8 to 10 μ in diameter, intermingled with paraphyses. The obovoid basidiospores are hyaline and measure 5·5 to 7 by 4·5 to 5 μ. The sporophore is perennial, forming its fertile porous surface in layers year after year. Dissemination is partially by air, but burrowing rodents are believed to be liable to carry the fungus to new roots.

Fomes annosus grows well in culture, forming a colourless mycelium which becomes matted and fawn-coloured and may eventually develop a porous surface. Ellipsoidal terminal chlamydospores, 9 to 15 by 6 to 12 μ, and *Eurotium*-like conidiophores bearing obovoid hyaline conidia, 5 to 8 by 3·5 to 6 μ, are also produced. These conidia, which appear in no other species of Fomes [5], germinate like the basidiospores and conidial cultures have yielded small sporophores [12]. The mycelium forms acid vigorously [7, 8, 19] and has its optimum reaction for growth about $p\mathrm{H}_4$ to $p\mathrm{H}_{4\cdot5}$ or $p\mathrm{H}_6$ [13, 20]; in nutrient media it tends to adjust the reaction towards this level [20]. Its optimum temperature for growth is about 23° C. [19].

The disease is more severe in plantations on arable than on pasture or moorlands [14, 17]; possibly owing to deficient aeration of the 'plough pan', early die-back of the vertical roots occurs in the former type of soil [13]. It is also more severe when conifers are grown in a second stand immediately following the first, presumably because the accumulation of raw humus leads to soil sourness and

death of lateral roots [1]. Soil acidity is one of the most important predisposing factors in infection, which is also more virulent in the larch on sandy soils than on clay [15], though the reverse occurs in spruce [17].

Strongly acid soils should if possible be avoided in planting conifers, owing to their tendency to induce heart rot. In moderately acid soils much can be done by preliminary removal of plants that promote acidity — bracken, heather, the Vacciniums, and the like — and leaving time for the decomposition of the raw humus at the surface of such soils. (In 1940, in Norway, however, moorland plants of the heather type were believed to assist materially in the transmission of the parasite [17a].) Lime and insoluble phosphates should be freely used in planting to counteract acidity. Young and middle-aged trees are prone to infection when the closing of the canopy leads to an accumulation of raw humus, so that heavy thinning is recommended when the stands are 20 to 25 years old [4, 15]. In starting a larch plantation physical and biotic requirements of the tree need to be taken into account [9, 10]; seed from a racial stock of home origin is preferable to nursery seedlings, and during cultivation every effort should be made to induce shallow rooting and avoid deep penetrating roots, on account of their liability to asphyxiation in the subsoil [1, 12, 15].

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Larch Canker, *Dasyscypha willkommii* (Hart.) Rehm

Larch canker is widespread in Europe, from humid lowland to high alpine situations; infections found in America on introduced European larches (*Larix decidua* and *L. leptolepis*) are believed to have been eradicated [13].

There is much difference of opinion concerning the etiology of larch canker, a disease which causes great injury in young plantations in Britain. By some it is

attributed to the attack of the Discomycete *Dasyscypha willkommii*, by others to frost, and others again invoke both these agencies acting together ; the last is the view which seems most satisfactorily to explain the symptoms and course of the disease.

Dasyscypha has, however, been considered to be the primary cause of larch canker [12, 13a, 17, 18]. It is possible that failure to produce cankers by inoculation has sometimes been due to confusion between the parasitic species, *D. willkommii*, and the purely saprophytic *D. calycina* which almost invariably accompanies it, though it has also been claimed that they should be regarded as forms of a single species since all intermediate stages between them occur [14]. In the United States it is claimed [12] that *D. willkommii* alone is associated with canker formation and a strain of the fungus received from America has been found in England to cause necrosis and die-back of twigs of *Larix decidua* in the absence of frost injury [4, 13a]. Nevertheless, according to some, if frost action were to be substituted for the action of the fungus the sequence of events in the development of the cankers as described below need not be seriously altered [2-9, 14].

In young larch the cankers develop rapidly and are usually fairly well established within the first six years of age ; they may completely girdle and kill seedlings a year or two old. Injury diminishes with growth and practically ceases after 30 or 40 years [10, 19]. Cankers on the main stem almost invariably appear at the base of a lateral branch which has died back (Fig. 426), or near a dormant bud [3, 14]. At first they are simple swellings but later become open wounds forming an amphitheatre floored with dead wood and surrounded by raised tiers of swollen tissue. A copious flow of resin comes from them and may stream down the stem before hardening to a white crust.

In Britain the larch is susceptible to frost injury in the spring from the end of March to the middle of May and again in the autumn from about mid-September [2]. The former period coincides with the renewal of cambial activity and the new cambium starting from the buds and progressively developing downwards, often in localised patches around a bud or twig, is particularly liable to suffer from frost. This may kill or damage its tender cells while leaving the exterior tissues unscathed. The injury may vary from tree to tree or even in different parts of the same tree in accordance with the stage of cambial growth around a particular bud or shoot.



FIG. 426.—Canker of larch. Canker showing origin from a branch, and subsequently healed on one side ($\times \frac{2}{3}$) (after Hiley, *The Fungal Diseases of the Common Larch*, by permission of Oxford Clarendon Press)

The sequence of events leading to the formation of a canker may be attributed, as above mentioned, wholly to frost action or wholly to fungal infection. In Fig. 427, 1, infection (or frost action) has occurred at a patch on the stem marked X. The effect is not to kill but to cause a portion of cambium AB to be stimulated to form

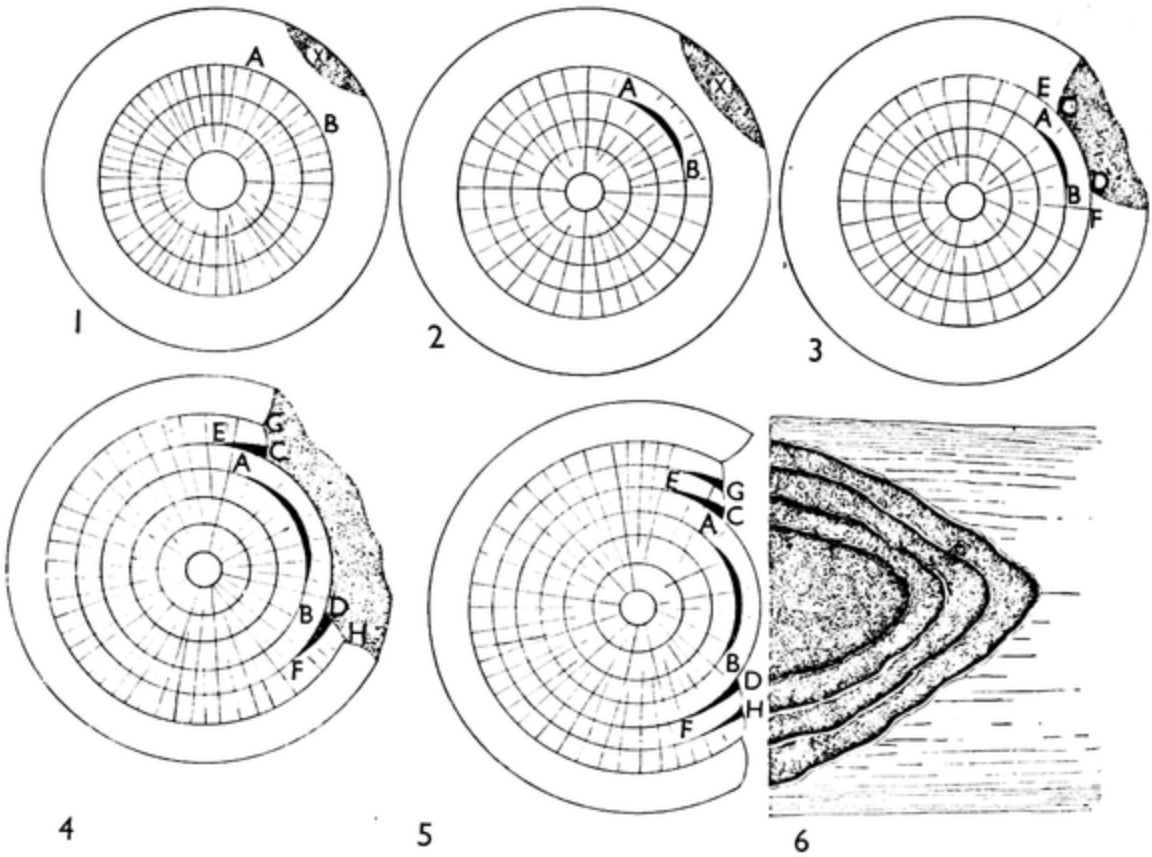


FIG. 427.—Larch canker. Diagrams showing stages in the formation of a canker. 1, infected patch of stem at X, the invading fungus about to affect a portion of the wood cambium at *AB*, directly opposite. 2, extension of the ' stimulus ' at X, but *AB*, not yet killed, has formed a layer of ' abnormal wood ' (see Fig. 135) which has become covered over by normal wood. 3, extension of fungus right down to the cambium at *CD*, which has been killed, so that no wood has been formed in this part, and here the external tissues are collapsing. 4, the fungus has affected portions of cambium *CE* and *DF*, as in *AB* (2), and abnormal wood is formed here. 5, with exposure of the wood, the fungus extends further, as in *CE*, *DF*, and is starting at *G* and *H*. 6, diagram of surface of a canker showing development in form of an amphitheatre, each step corresponding to a ring of wood in the previous diagrams; note the curling back of the wood at the inner margin of each ' step ', where the fungus is affecting the cambium as at *CE* and *DF* in 5 (1-5, after Hiley, *Fungal Diseases of the Common Larch*, by permission of Oxford Clarendon Press)

abnormal wood, which, as we have already seen, consists of iso-diametric parenchyma containing starch and usually becoming lignified in place of the normal tracheids and medullary rays (Fig. 135). As the effects pass off, normal xylem is again formed from the recovered cambium. Progressive infection, however, or a further spell of frost the following season, may actually kill this part of the cambium now shown in CD after adding an increment of normal wood over the abnormal AB. In the cambium contiguous to that killed, arcs of wound wood from injured but not dead cells again form (CE, DF) and abnormal wood is formed here as in AB. Meanwhile growth in thickness goes on, leaving the dead part (CD) behind, its flanks now fully exposed; and the cambial cells which form the new flanks (G and H) are, in their turn, killed by fungal action or frost. With intermittent cambial growth and continued infection (or periodic incidence of frost) less and less wood forms on the infected (frosted) side of the stem, and with each addition of secondary wood beyond the killed zone, the latter (CD) recedes farther

from the surface of the stem, remaining as the floor of an amphitheatre [14] whose step-like tiers are laid down on foundations of wound wood (Fig. 427, 6). In these tiers the margins of the cambiums are dangerously exposed to further infection or frost injury, the more so because they are known to show precocious activity in the spring and unduly delayed growth in the autumn. The result is that the steps of the amphitheatre get lifted and their edges torn away by recurring frosts and infection.

During this process the canker finally opens to the surface. In the earlier stages it is covered by rather thin and inelastic layers of phloem and cortex. These become stretched by the increasing mass of wound wood so as to reach a state of tension. The rupturing of the bark occurs especially at thaws, when the melted ice in the cells swells the tissues ; the bark of the smaller twigs is raised into ridges and blisters which readily crack, while that of the larger stems may be violently torn. In the open wound a repetition of frost injury to the base of the steps enlarges the canker year by year. Shredding of the bark may be considerable and new frost cankers may form near the first and even fuse with it.

At any stage a hardy tree may succeed in healing off and burying the earlier injured part under rings of normal wood. Very many cankers appear to become obliterated in this way, including nearly all forming in the older trees. In this process it is probable that the presence or absence of *Dasyscypha willkommii* is of critical importance. The fungus may be able to gain entry through the cork barriers of the stem by way of the dead snags or buds around which the deep-seated injury first starts. Even through these paths of entry, free passage is checked by a cork barrier which extends continuously from the surface to the xylem of the snag or base of the bud and is continued across the xylem by a gum barrier. Passage through this is believed to occur mainly at the junction of cork and gum barrier. The enzymes of the fungus disorganise the cells of the cortex and outer phloem but appear to be ordinarily unable to injure the actively growing tissues near the cambium. It has been claimed, however, that when an incipient frost canker has developed in the cambium of the stem at the point reached by the fungus the latter can continue its advance into the wood, especially if an arc of cambial cells has been killed. The new tissues at the edge of the canker are also, it is claimed, made more susceptible to frost injury by the action of the enzymes in raising the freezing point of the sap and reducing osmotic pressure through the production of tannin at the expense of the sugars [15]. This leads to further killing back of the edges and their colonisation by the fungus. When eventually the wood is invaded it is turned a deep reddish brown and the functionless tracheids of the inner wood become choked with hyphae and wound gum.

On this view the fungus acts as a saprophyte or a wound parasite, injuring some tissues by enzyme action in advance of its growth and enhancing liability to frost damage by raising the freezing point of living cells reached by its enzymes. Attempts to induce canker formation by inoculation with *Dasyscypha* frequently fail, and where they have succeeded it is not always certain that the action of adverse influences of the environment, such as frost, have been excluded. The disease is now widely regarded as primarily due to these influences in relation to the periodic activity of the cambium [3-9, 11, 15, 20].

The fructifications (apothecia) of *Dasyscypha willkommii* (Fig. 39) are formed in clusters around the cankers, especially from August to March [19]. They resemble small shallow egg-cups, 2 to 5 mm. across, with short stalks, white outside and lined with a smooth orange-red hymenium which gets paler with age. They occur also in profusion on dead twigs and branches of the larch. They arise from small white cushions breaking through the bark and forming from February onwards (Fig. 428). The club-shaped asci measure 150 to 200 by 10 to 14 μ and contain in a single row 8 ascospores, which range from 20 to 23 by 9 to 10 μ in diameter. The fungus is reported to be homothallic [19]. Its complete life-cycle can be obtained in culture, ascospore germination being favoured on media with extract of larch cortex, glycerine [16], and a temperature of 15° to 22° C. Growth is impeded by an alkaline reaction [10].

The incidence of larch canker is reported by some investigators to be related to aspect, susceptibility increasing, for instance, on slopes receiving the morning sun and therefore thawing earlier than those shaded until later in the day [4-8]. But the frequency of cankers associated with *Dasyscypha willkommii* is said to be independent of aspect [14]. Deep and well-aerated soils promote vigorous growth of the larch and the healing of the cankers. In mixed plantations the deeper rooted hardwoods open up the soil, with the same result.

Some measure of control over this disease may be secured by early pruning of the lateral branches which would normally die back when the tree is 5 or 6 years old. Painting over of growing cankers in older trees with tar is reported to have

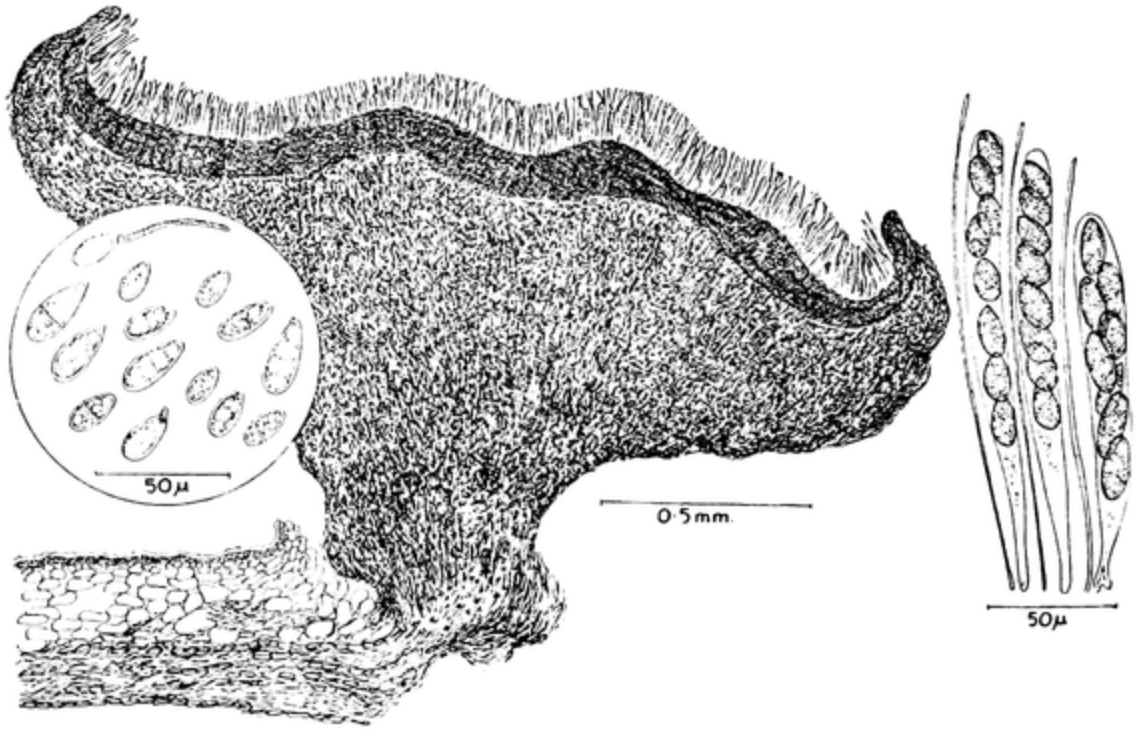


FIG. 428.—Larch canker. Section of a ripe apothecium (from material collected in March) showing the dense hymenium of asci and septated paraphyses, as shown on right. The fungus has disrupted the deep-seated cork layers in the stem (cut longitudinally), and is present between these layers and those of the phelloderm in greater quantity than in any other tissues of the stem. Inset, the ripe ascospores, some germinating (in water); they are mostly unicellular (from a slide made by Jamieson)

given good results [21]. Frost injury can often be diminished by silvicultural methods directed specially to protect the young trees from cold currents of air [8]. The most effective measure that can be taken, however, is the selection of species, varieties, or races of larch adapted to the habitat [2, 17, 18]. A fairly wide range of types is available and others may become so by methodical breeding and selection. In Britain the best control of canker may lie in extending the propagation of the Scottish larch in suitable localities [1, 3], just as in Germany a type from the Sudeten border of Bohemia appears to enjoy comparative immunity from canker injury in certain areas with a similar climate to its original habitat [11, 18].

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Meria Needle Cast of Larch, *Meria laricis* Vuill.

This disease of the European larch (*Larix decidua*) and the western American larch (*L. occidentalis*), in Britain, attacks the trees chiefly in the nursery. It occurs in Europe on the former species, but so far there is no report of the disease in America. The Asiatic species (*L. gmelini*) and the Japanese larch (*L. kaempferi*) are resistant; this type of leaf cast is, therefore, fairly specialised on its host.

Leaf cast of larch was first discovered in France in 1895 [5]. It is caused by *Meria laricis* [8, 9, 10], a member of the Fungi Imperfecti, about whose affinities very little is known; it possesses different strains and cultural races [6].

The fungus is confined entirely to the leaf, and the intercellular, septated mycelium occupying the mesophyll consists of broad hyphae which give rise to much finer branches, both kinds having unusually thick walls; there are no haustoria. No definite fructification is formed on the infected leaf, and prior to spore formation the mycelium merely aggregates to form cushions in the substomatal cavities, and from these hyphal masses tufts of conidiophores are passed out through the stomata.

The conidiophores are 1- to 3-celled, and short sterigmata are developed from each cell, terminal from the apical cell and lateral from the others; from each sterigma one or more spores are produced in succession. The spores are colourless, unicellular at first, later bicellular prior to germination, a septum being laid down across the constricted middle of the spore; they measure on an average 9·3 by 3·1 μ, at the broader diameter. The spore masses are very difficult to detect on the leaves in dry weather, but in a damp atmosphere conidiophores and spores are developed in abundance. Germinating spores sometimes produce small microconidia (3·6 by 1·4 μ) of unknown function [6].

The disease attacks mostly one, two, or three-year-old seedling trees in the nursery, causing the leaves to turn brown and drop. It is only troublesome, however, in the one-year beds, though older trees up to ten feet high, or so, may also suffer from defoliation. Mortality is naturally higher in the seedling beds, but the disease does little harm to established trees except in so far as the loss of foliage impoverishes the wood and so brings about reduction in the height and girth of the tree.

Early signs of disease in the nursery are seen a little later than on older trees. Seedlings in their first year are attacked usually about the end of June, the cotyledons, as the only leaves formed by that time, being the first to suffer; on older plants the disease appears on the young needles towards the commencement of May or the end of April [6]. At first, the early symptoms resemble somewhat the effects of frost on the leaves [2], but are quite distinctive. Frost attacks the needles first at the extreme tips, and then browns and kills off the whole leaf almost immediately. This disease, however, generally commencing early in May, begins as one or more spots towards the middle of a leaf and thereafter the browning progresses downwards very gradually towards the base of the leaf, which finally goes brown all over. The disease extends no farther than the base of the leaves, which soon fall off, but frosted needles often remain on the tree for a long time [3, 4].

In March or April young nursery trees which have developed dwarf shoots are early attacked, and the first spring infections occur on the leaves of these small side shoots, the source of infection being the old leaves on the ground. The leaves on the long shoots of unlimited growth, which do not appear until later, about May usually, are also in turn attacked. On the long shoots the needles may become infected at the tips, and the brown or yellowish-brown discoloration again travels down gradually towards the base of the leaf, but before it reaches that point the leaves are often cast off. But the long shoots do not lose their leaves from the apex down; the disease starts on those leaves which are situated some two or three inches from the apex, and as the forward growth of the shoot is not interfered with, fresh leaves continue to develop. The new leaves, in turn, are then attacked and meanwhile the older infected leaves below are cast off. With continued growth of the long shoots, which slows down towards September, progressive disease with defoliation results in considerable portions of these shoots becoming stripped of their leaves, but there is always a tuft of young leaves at the tips and these may remain until the tree divests itself of its foliage in the normal course of seasonal change.

The majority of infected leaves fall to the ground and the fungus survives in them throughout the winter, but it is often observed, especially among young trees, that quite a number of infected leaves may still remain on the trees during the winter and may not be shed until late in the following spring. When, therefore, growth is resumed and new leaves unfold, it is not improbable that the latter may get infected from spores carried in splashing rain-drops from leaves retained on the tree. The fungus, moreover, survives and even spreads in these retained needles during the winter, and in the fallen leaves may remain alive for a long time under moist conditions; in dry weather it tends to die out of the needles on the ground.

While the ideal conditions for natural infection appear to be dependent on high atmospheric humidities, successful inoculations of the leaves with spores are obtained when there is a low rate of evaporation from the leaf surface, coupled with the absence of wind so as to maintain a moist environment about the leaves until infection has become established. Infections usually appear in two to four days following a brush-over with the wet spores when the temperature is from 10° to 25° C., but is much slower at low temperatures of 0° to 5° C., while high temperatures up to about 30° C. prevent them entirely.

The appearance of this disease in fresh localities can usually be traced to the planting of young stock on which infection may have passed unnoticed. There is definitely no evidence that the trouble can be carried with the seed, and indeed the best method of avoiding the introduction of the fungus into new areas is to start with seed planting.

On a small scale, removal and burning of all infected leaves on the ground would obviously prevent to a great extent the reappearance of the disease in the following season, but the method is hardly practicable in large plantations. Effective control, if carried out before the symptoms of disease appear, may be obtained by spraying, and for this several preparations are recommended, namely 'amberene' at 1·5 per cent. strength; 'sulsol' at 0·3 per cent. with the addition of a wetting compound; liver of sulphur at 0·7 per cent.; or lime sulphur at 1 per cent. Any of these materials should be applied at the rate of 4 gallons of solution for every 100 square yards, spraying being carried out on dull but not rainy days; the first application should be given at the end of February or early in March, and subsequent treatments should follow at intervals of a fortnight or three weeks. After the end of July, if the weather appears settled, spraying may be discontinued, as loss of foliage after that date does not cause serious harm to the trees [7].

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Needle Cast of Scots Pine, *Lophodermium pinastri* (Schrad.) Chev.

Young seedlings of Scots pine (*Pinus sylvestris*) as well as trees up to 20 years old (mainly up to 5 years) often suffer severely from loss of foliage due to needle cast disease or pine blight. It is caused by the Ascomycete *Lophodermium pinastri*, a member of the Hysteriales. The same disease attacks many other pines such as *P. montana*, *P. nigra*, *P. cembra*, *P. ponderosa*, and *P. lambertiana* [3, 6, 7, 8, 10, 16]. It is very prevalent throughout North America and northern Europe, chiefly on the Scots pine, and is liable to appear after cold wet summers [5], or very dry weather in early spring [7], or after heavy frosts in the autumn [1].

On very young nursery seedlings the first signs of infection consist of small greyish spots, first on the primary leaves, then on the double needles from below upwards. This indicates that infection starts from spores conveyed by wind from the old leaves in which the fungus has wintered on the ground. On the young double needles, on older trees, as they emerge on the opening shoots, infections are very difficult to detect, and the general observation is that the needles are liable to attack just when they are about to attain their full size. This period of susceptibility is said to coincide with a condition of minimum acidity in the leaf tissues [19]. Early infections on these, almost mature needles, consist of spots of variable colour, sometimes grey or yellow, or tinged with purple or red, but later, as infection becomes better established, the spots, at variable intervals along the leaves, are mostly brown in colour. Towards the autumn, affected needles develop a series of very narrow black rings which girdle the leaves at irregular intervals from base to tip, thus isolating the spots, singly or in groups, and dividing the needles into portions which soon turn a yellow-brown colour from the action of the spreading fungus within (Fig. 429). These black rings are actually the edges of diaphragms consisting of a few layers of altered mesophyll cells killed by the fungus, and thereafter crushed and impregnated with a black gummy substance which on ' setting ' makes these

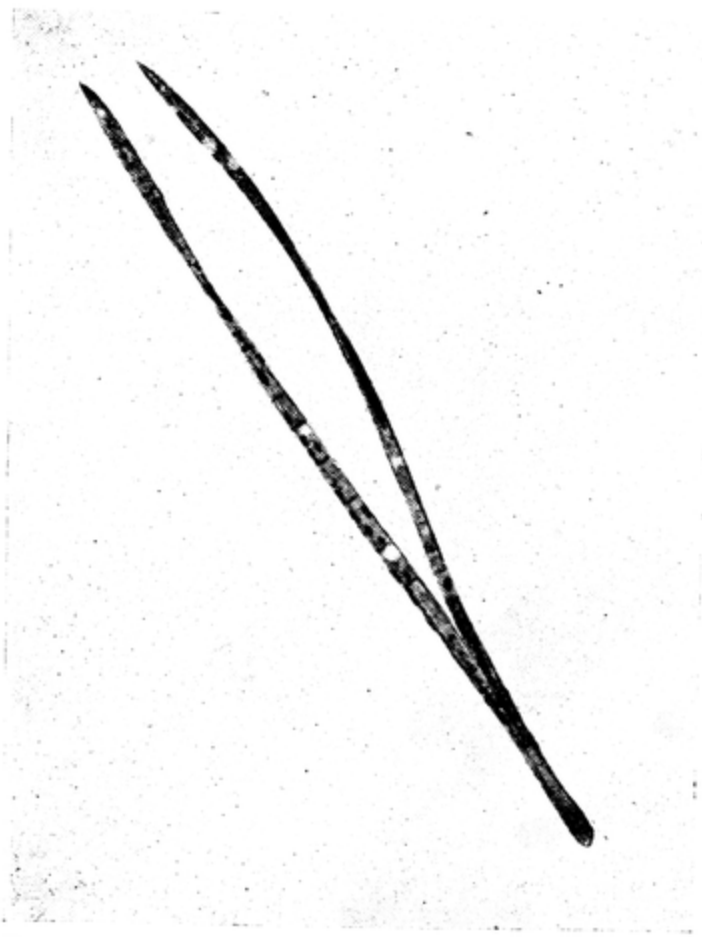


FIG. 429.—Needle cast of Scots pine (*Lophodermium pinastri*). Pine needle showing the apothecial spots; note the black, transverse rings (see also Fig. 123)

partitions tough and resistant (Fig. 123). The black diaphragms do not go right across the leaf and extend only as far as the endodermis, so that the vascular bundles in the needles are still functional and will remain so almost until defoliation. But the cells of the mesophyll between the diaphragms are practically all destroyed by infection, and within these portions of the needles thus partitioned off from each other the fungus increases rapidly. The mycelium having replaced most of the mesophyll proceeds to become established at various places between the epidermis and hypodermis, and it is between these two layers, or more correctly within the tangentially split epidermis, that it builds up its spermagonial and apothecial fructifications.

The spermagonia are developed first, but the time of their appearance is variable, depending apparently upon locality and climate. In Britain they may be seen about the end of summer, as minute black specks, opening by an ostiole, or otherwise breaking irregularly around the margin. For the accommodation of a spermagonium the fungus, by dissolving the lower half of the epidermal wall, first displaces the thick sclerosed linings from a group of the epidermal cells (Fig. 150), so that when finally formed the spermagonium is covered by a portion of the cuticle and the outer half of the epidermal wall, the sclerosed thickenings meantime having been removed and deposited at the base along with the developing hypothecium. The narrow, bacilliform spermatia, 4 to 8 by 0·5 μ, are abstricted in great number from short pyriform cells on the hypothecium, and are extruded to the surface, embedded in mucilage. The apothecia follow later, and, while their development begins on leaves still on the tree, it is not completed until after leaf fall and the fungus remains dormant in the fallen leaves until the spring. They arise in the same way as the spermagonia, intra-epidermal, but are protected by a much thicker roof of pseudoparenchyma made by the fungus after displacement of the sclerosed inner walls of the epidermis (Fig. 150 C) [9, 17]. Apothecia may be laid down within defunct spermagonia or arise anew, very frequently along the angles of the leaf, singly or in opposite pairs, rarely fusing to form a stroma (Fig. 430). The apothecia are small, ellipsoid-conchoidal, about 1·0 by 0·5 mm., and shiny black (Fig. 429). The hymenium consists of numerous club-shaped asci which are 8-spored; the ascospores are filiform, sheathed, unicellular and uninucleate, slightly curved, and measure from 90 to 140 by 1·5 to 1·7 μ; paraphyses are slender; dehiscence occurs along a median ridge lined with periphyses (Fig. 430). *L. pinastri* is stated to be heterothallic [12].

The apothecia pass the winter on the fallen leaves, more or less covered by needle litter. From the end of June onwards ascospores are ejected and infections may start in July and continue until September or later. On moistened leaves the ascospores adhere firmly by virtue of the gelatinous sheath and penetration by a single germ-tube is stomatal. From a sub-stomatal vesicle one or more delicate hyphae first penetrate the guard cells and thereafter the fungus proceeds quickly into the mesophyll, which is destroyed soon after preparation has been made to establish the black diaphragms or partitions above mentioned (Fig. 123 B). After spermagonial development is over and apothecial initials are established, the fungus crosses the endodermis into the stele, to occupy the pericycle and phloem, but the lignified tracheids and the pitted cells of the transfusion tissue contain but few hyphae. Leaf fall is now imminent and may be brought about in two ways, either in the normal fashion by absciss cork layers at

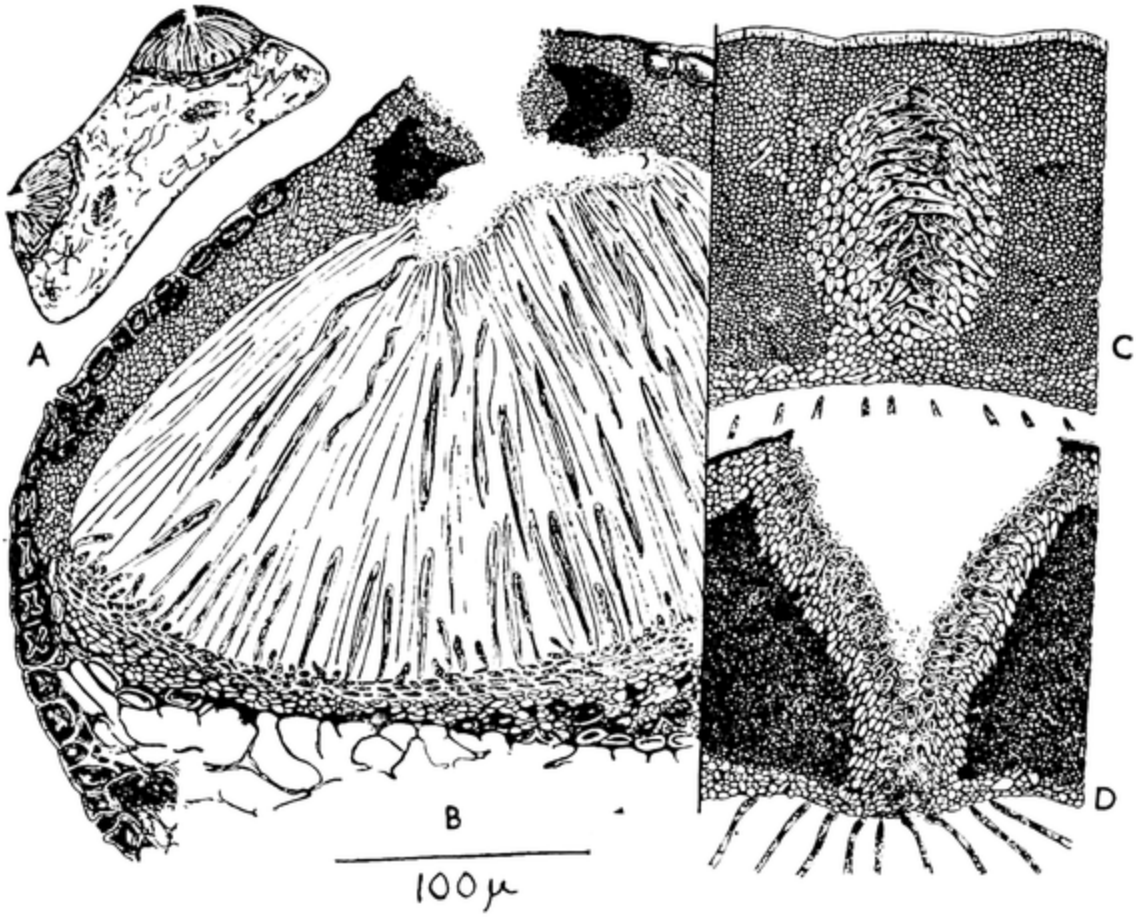


FIG. 430.—*Lophodermium pinastri*. *A*, diagram of transverse section of pine leaf showing two apothecia. *B*, portion of section of an apothecium at time of spore discharge. *C*, the interlocking periphyses along the median line of dehiscence. *D*, dehiscence at the median ridge almost complete, showing pressure of the roof on the tips of the paraphyses (after Jones, *Ann. Bot.*)

the base of the dwarf shoot, or by a clogging of the tissues within the dwarf shoot with black gummy substance made by the fungus (Fig. 123 C). In the latter case the fungus, by penetrating the gum barrier, is sometimes successful in passing from the needles into the stem, but the extent of its invasion into the tree has not been ascertained and it is not known whether *L. pinastri* is capable of surviving in the tree in the form of a resting mycelium [9]. Primary infections appear to be due entirely to ascospores and the mycelium within the fallen leaves is said to persist for years, and is resistant to high temperatures [3, 12], but there is no evidence that the fungus can thrive as a saprophyte in the dead needles [18]. The fungus has been grown in culture, from ascospores, on sterilised dead needles (producing spermagonia but not apothecia) and on agar or gelatine media containing biomalt and citric acid [4, 12, 18].

Premature defoliation of Scots pine induced by *L. pinastri* is due mainly to the effects of excessive transpiration following, at least in part, upon the early crippling of the stomatal guard cells, and in the infected leaves hardly a stoma remains unexploited by the fungus. There follows, accordingly, much desiccation of branches due to disorganisation of the water balance, and in dry weather there is severe loss of foliage from which young trees fail to recover [12].

'Needle cast' may be controlled on nursery pines by spraying with 1 or 2 per cent. alkaline Bordeaux mixture, the first application being given towards the end of May when the needles are about half developed, and another at the beginning of July [2, 13a, 14, 15]; the spraying of older trees may be deferred until July, using 2 per cent. Bordeaux or Burgundy mixture, or a 1 in 32 solution of lime sulphur [16], but the first applications are given in some areas as early as April and continued at 2 or 3 weeks' intervals, until the autumn [8]. As the mycelium of *L. pinastri* is long-lived, all diseased plants and needles should be destroyed by burning, or deeply buried [3].

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Blister Rust of White Pine, *Cronartium ribicola* J. C. Fischer

Blister rust caused by *Cronartium ribicola* [7] occurs on the white or Weymouth pine (*Pinus strobus*) and other five-needled pines on which the aecidial stage in the life-history of this heteroecious fungus is developed; the uredospore and teleutospore stages occur on the leaves of currant and gooseberry, the *Ribes* host. The disease is also known as the pine and currant rust; it is not common on the gooseberry in this country. The rust is of much greater importance on the pines and economically of little significance on either of the *Ribes* hosts, on which it appears rather late in the season.

On the currant the disease is confined to the leaves and does no direct harm to the bushes except that, by causing the leaves to turn brown and fall prematurely, the plants are weakened from repeated annual attacks, and the fruit crop gets smaller every year. The fungus does not live in a mycelial condition in the *Ribes* host over winter [20]. But the disease on the pines is very serious, for the fungus lives and grows under the bark from year to year, forming cankers, until the tree dies, and heavy losses are incurred on trees of all ages [29] (Figs. 95, 431).

Blister rust is believed to have appeared in Europe about 1854 on currant in the Baltic provinces of Russia [7, 18], thence spreading to northern and western Europe,



FIG. 431.—Blister rust of Weymouth pine (*Cronartium ribicola*). *A*, trunk showing, at point indicated by the axe, a canker, 2 ft. long, covered with aecidia. *B*, aecidial masses breaking through the bark. *C*, the same, enlarged. *D*, early stage on twig, showing slight swelling; this part showed orange-yellow discoloration of the bark. *E*, another early stage showing swelling caused by the developing canker. *F*, note the cluster of needles on dwarf shoot, through which infection entered the branch; spermagonia are developed around the lesion. *G*, the uredo-stage on leaf of the yellow flowering currant. *H*, the teleutosori-threads on the under side of currant leaf (U.S. Dept. Agric.)

but probably did not appear in America until about 1898 [21], though a much earlier date than this is also reported [18]. It is now widely distributed in the United States and Canada, and occurs also in Japan and Saghalien, so that it seems to be fairly general throughout the northern hemisphere [6, 18, 19, 19a, 21].

Both wild and cultivated currant and gooseberry bushes are responsible for carrying the disease over to the pines, the ordinary cultivated black currant being the most commonly implicated. The rust attacks the leaves of the currant from about early June to autumn. These primary infections take place, of course, from aecidiospores conveyed by wind from the alternate pine host. The aecidiospores germinate (Fig. 432 R, S) on the lower side of the leaf, the germ-tubes entering through the stomata to establish an intercellular mycelium of binucleate cells, with haustoria, chiefly in the tissue around the sub-stomatal cavities and forming in these regions, just below the epidermis numerous sori which break out on the under side of the leaf as yellow pustules (Fig. 431 G) of uredospores [1]. These spores infect only the *Ribes* host and new generations of them arise at intervals of 10 to 14 days throughout the summer. These summer spores are viable for several months so that throughout the season there is extensive infection of currant and gooseberry bushes ; they infect the leaves in much the same way as the aecidiospores. The uredospores are orange in colour, with an echinulate wall, and measure from 21 to 24 by 14 to 18 μ (Fig. 432 A, C, D, E).

The teleutospores usually appear a little later, either in the same sori as the uredospores or independently from a similar mycelium of binucleate cells. The teleutospores of *C. ribicola* are arranged together in a very characteristic manner. They are not developed singly like the uredospores but in numerous aggregations of small thread-like columns which break out on the under side of the leaves (Fig. 431 H), and may continue to be formed to the end of the season until the leaves drop off. A column of teleutospores (Fig. 432 F-K) is a cylindrical, more or less solid pillar of cells, hyaline or pinkish in colour, every cell of a column being a teleutospore. The teleutospore cells measure on an average 16 by 42 μ, are binucleate when young, but in the mature cells the two nuclei fuse together. The columns may be straight or curved and vary greatly in size, up to 2 mm. high, as well as in number per unit area of the leaf, according to the species of *Ribes* on which they grow. On the black currant they are far more abundant than on any other kind, and this may be one reason why this species causes relatively heavier infections than any other species of *Ribes* that may be found in the vicinity of pine plantations. Moreover, its leaf is of firmer texture, and is said to defy the action of frost longer than any other of its kind [30]. The teleutospores survive for periods of from 3 weeks to 3 months, longer on fallen leaves on moist soil than on leaves retained on the trees. When they germinate, the teleutospores still remain together in their columns on the leaf (Fig. 432 J), the first to grow being the oldest at the top of the column, others in succession until the youngest at the base are reached [31]. Any number of the teleutospores forming an individual column may germinate, each producing a typical 4-celled promycelium or basidium characteristic of a rust fungus and bearing four sterigmata and sporidia or basidiospores (Fig. 432 K, L) ; the latter are small and round, and measure from 8 to 10 μ in diameter. Sporidia begin to appear about 12 hours after the promycelia are pushed out to the surface of the column, the optimum temperature for their production being from 12° to 18° C., the minimum from 0° to 1° C., and the maximum about 21° C. [29]. For a high percentage of germination, teleutospores should be from 96 to 216 hours old [12a], and need high atmospheric humidity, of 96 to 100 per cent. saturation, but direct contact with water is not essential, a mere film being sufficient, and a pre-cooling or icing stimulates the germination of sporidia [27, 32].

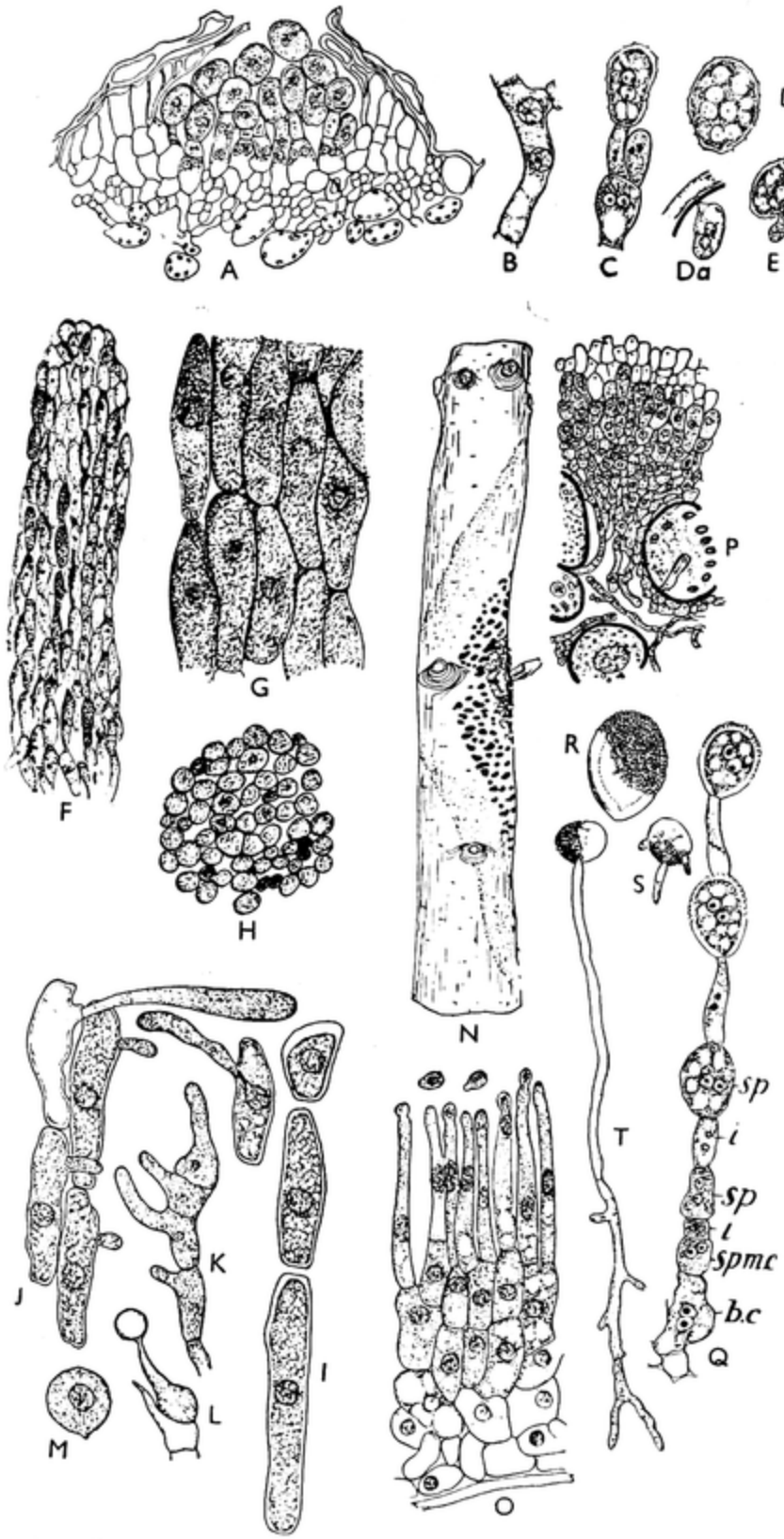


FIG. 432.—*Cronartium ribicola*. *A*, section of small uredosorus (× 250). *B*, a binucleate cell from mycelium in leaf of *Ribes*. *C*, a basal cell bearing a stalk cell surmounted by a nearly ripe uredospore, and a secondary uredospore initial (× 425). *D*, a mature uredospore (× 425). *Da*, a binucleate haustorium from a cell of *Ribes* (× 850). *E*, germinating uredospore (× 425). *F*, a thread or column of teleutospores, in longitudinal section (× 125). *G*, ditto (× 525); note nuclei. *H*, a cross-section of a column (× 260). *I*, three teleutospores, from tip and side of a column (× 260). *J*, four teleutospores germinating *in situ*. *K*, the germinating promycelium (basidium). *L*, tip cell of promycelium showing sterigma and nearly ripe sporidium (× 850).

The sporidia, conveyed by wind, are the means of carrying the disease over from the currant to the coniferous host, the pine. Infection from sporidia falling on the needle leaves occurs by direct penetration of the epidermis [11], or through the stomata, during periods of dew, heavy fog, or cloudy weather, and is favoured by rather high relative humidities and medium temperatures [12a, 23, 37, 38]. Provided they still retain their stomata, young twigs may also be infected in the same way [3, 26], otherwise it is not deemed likely that the pine stem ever becomes infected except when the mycelium travels into it from the needle leaves. First signs of infection on the needles consist of yellow to brownish spots. On the Weymouth pine infections which become established in the needles on the first- and second-year shoots usually enter the stem and any older leaves attacked fall off, in the normal habit of the tree, before the fungus has had time to pass from them into the stem. But some species of pines retain their leaves longer than the Weymouth ; thus *P. monticola* keeps them for a year longer and there is usually heavier infection of the stem from the older leaves in this species. Accordingly those species of five-needled pines which keep their leaves longest suffer more severely from stem infections than those which discard the older leaves earlier. The number of stomata per unit area in the leaves of these various species is also considered to be a factor connected with relative susceptibility to blister rust [28].

Passing in from the infected leaves into the stem, the mycelium enters the intercellular spaces of the cortex and the phloem, and later becomes well established between these two tissues, wedging them apart so that much of the phloem becomes crushed against the wood. Accordingly cambial division in infected regions is much less active than in healthy parts of the stem, but the secondary wood is normal except that thinner increments of tracheids are added to the annual rings. But, so far, there is little interference with the stem functions, and not until the fungus has accumulated so much between cortex and phloem as to burst through the bark, does the infection become serious and a canker formed. Then, with the exposure and drying-out of the delicate tissues within, the cells perish, and there follows extensive splitting of tissues with copious exudation of resin in the vicinity of the canker. The enlargement of the cankers and continuous discharge of resin from the broken tissues is accountable for considerable injury to the tree. The extension of the lesion around, so as to girdle the twig or branch, is due to progressive cracking of the bark caused by the periodic swelling and drying of the exudate. Moreover, the impregnation of large portions of the cortex and phloem in the vicinity of a canker with resin results in a complete stoppage of the translocatory functions of the phloem and there is consequently much hypertrophy around the affected part (Fig. 431 F).

M, sporidium. *N*, an infected 12-year-old stem, infection having entered at the small branch ; the spermagonial (pycnial) areas, shown in black dots, and the near aecidial area on which the bark is cracked and broken ; in another season, the aecidial area will spread over the spermagonial area, and the latter will advance as far as the boundary ($\times \frac{1}{4}$). *O*, elements of a spermagonium, spermatial hyphae abstricting spermatia (pycnospores). *P*, part of a young aecidium, showing the '*fertile cells*', host cells (black) ; note haustoria and starch grains. *Q*, a chain of aecidiospores, *bc*, basal cell, *i*, interstitial cell, *sp*, aecidiospore ($\times 425$). *R*, ripe aecidiospore ($\times 425$). *S*, germinating aecidiospore ($\times 425$); note, partly verrucose and partly smooth, spore wall ($\times 212$). *T*, later stage (after Colley (adapted), *J. Agric. Res.*)

The period of incubation between infection and the first appearance of canker varies with the location (altitude), the season, the species, and age of the tree. On young trees of *P. monticola*, not over 5 years old, incipient cankers appeared in 6 months; on trees 8 years and over, the period varied from 20 to 26 months, but only in very exceptional conditions do cankers ever appear in the year following that of infection; they arise mostly in the second year and to a lesser extent in subsequent seasons [13, 14, 25]. Provided a branch has not been actually girdled with disease there is no serious die-back, but when girdling cankers appear low down on the tree entire branches or the whole tree may be lost [15]. The stubs of cankered branches may remain alive and harbour the mycelium up to 5 years [17].

The spermagonia and aecidia are developed around the margins of the cankers (Fig. 431 A-F). The spermagonia appear first (Fig. 432 N, O), from June to September usually in the second summer after infection [12]. They are laid down between the cortex and periderm which they break through to push forth the numerous spermatia embedded in a viscid fluid or nectar; the spermatia are pyriform, 3·5 by 2·5 μ [4], and probably have a fertilising function during the development of the aecidial primordia [24]. The aecidia come later, from 6 months to $2\frac{1}{2}$ years or more, but usually appear in the spring following the first appearance of spermagonia [14]. The young aecidia arising as distinct sori, occasionally joining together, break through the periderm as thin yellow streaks at the edges of the cankers, from a bed of mycelium in which the cells contain at least two nuclei [5]. The emerging aecidia (Fig. 432 P-S) are furnished with a prominent thick peridium; the binucleate aecidiospores, separated by long, narrow intercalary cells (which finally disintegrate), are thick-walled, partly verrucose, orange coloured, and measure from 20 to 26 by 18 to 21 μ [4].

The aecidiospores are dispersed over extensive areas and are capable of germinating on currant and gooseberry bushes after many miles of travel by wind. It is estimated that the spores from pine to *Ribes* may carry in the upper air over distances of 300 to 400 miles [19a]. Aecidiospores infect the *Ribes* host in the same way as the uredospores, and from a binucleate mycelium established in the leaf, sori of uredospores begin to appear in about 10 days [1]. Aecidiospores germinate best at a temperature of 11° to 12° C., up to 28° C., and this capacity of the rust to grow over a wide range of temperatures is of importance in the infection of currant and gooseberry trees growing in widely extended latitudes [10]. The *Ribes* host is susceptible to aecidiospore infection from the time the leaves emerge from the buds, up to about 16 days or so, after which resistance increases. Infection is obviously greatly dependent on variations in seasonal conditions; thus an early spring by advancing the development of the leaves may help the currant to resist attack at a time when the production of aecidiospores is greatest. But this does not preclude infection in higher latitudes and in northerly places where spring is late and the *Ribes* host less forward than in lower latitudes. Conditions which advance spring in the north and retard it in the south tend, in general, to extend the range of conditions favourable for long-distance spread of the rust, and vice versa [16].

Few varieties of currants or gooseberries are resistant to this rust, but it is rarely found in this country on the gooseberry. The Norwegian Viking currant is highly resistant [1, 8].

The white pine (*P. strobus*) is very susceptible ; *P. monticola* is most commonly infected in North America; *P. lambertiana* and *P. albicaulis* are also subject to natural infection [19a]. *P. cembra helvetica*, *P. peuce*, and *P. excelsa* are more or less resistant [2, 8, 29, 34], but in Upper Bavaria *P. peuce* is slightly susceptible [35]. Recent studies in Wisconsin [24a] have shown that while a very high percentage of seedling selections of white pine subjected to natural or artificial inoculation developed cankers within a year, good resistance was found among grafted plants similarly treated, and trials with grafts and rooted cuttings give promise of high resistance among Wisconsin trees [24a].

As the rust is not capable of spreading to the pines without the *Ribes* host, active measures have been undertaken, especially in North America, for the systematic eradication of currant and gooseberry bushes (see Chapter I, p. 57). Where practicable, hand pulling and grubbing are resorted to, but on a bigger scale destructive spraying is adopted. By this method extensive areas of the bushes can be killed by the application of sodium chlorate, which must not be used unless mixed with calcium chloride which controls the fire propensities of the sodium compound [22, 33]. The rust on the *Ribes* host may be controlled by spraying with 4 : 4 : 50 Bordeaux mixture as soon as the buds burst into leaf [36].

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Needle Cast of Douglas Fir, *Rhabdocline pseudotsugae* Sydow

Leaf blight or needle cast of Douglas fir (*Pseudotsuga douglasii*) was first described in Scotland in 1922 [17], and the disease was probably introduced into this country about 1914 on trees from North America [2, 3]. It has since been found in several parts in south-east England and is perhaps much more widespread in Britain than already known [4]. Its spread to Europe was probably from Scotland to Germany, thence to Holland either on nursery stock or, as has been suggested, by spore dissemination [14, 15]. In Britain, three varieties of Douglas fir are susceptible to this disease to variable extent, the green *viridis* variety which is the common quick-growing type; the slower growing blue variety, *glauca*; and an intermediate form, *caesia*. The last named is the worst to suffer in all localities, followed by the blue *glauca*, while the green variety appears to be the least susceptible generally, in Britain and abroad. In America, where the Douglas fir is recognised only as a single species (*Ps. taxifolia*), all forms of the tree are apparently attacked [2]. The disease seems to be generally much more virulent in Scotland and Europe than in America [1, 10].

In North America [16] and in certain parts of Germany [6], young trees in the nursery, as well as established trees in close dense stands in the forest, up to 30 or 37 years old (mostly from 15 to 30 years), are liable to attack [2, 11], but in Britain the disease has not been found in the nursery and is usually seen here and in other parts of the Continent only on trees from 10 to 16 years' growth [8, 18]. Older trees are, in general, immune from it and in mixed stands with broad-leaved trees Douglas fir is not so frequently attacked [16].

On the blue variety *glauca* with its slightly hairy branches, the symptoms of leaf-cast disease consist of purplish-brown patches on the leaves, giving the foliage a mottled appearance, but on the smooth-branched, thinner-leaved intermediate variety *caesia*, on which, as stated, the disease is more severe, the whole needle usually becomes discoloured and loss of foliage on this tree is more extensive. In America, outbreaks of leaf cast disease are spasmodic, epidemics lasting for about two years then dying down, but this is not the experience in Britain where the trouble shows no loss of activity [17]. In general, by early spring the trees present a yellow-brown appearance and about the beginning of June are almost uniformly brown, as if suffering from the effects of frost. Indeed, the onset of frost, the effect of which may not be discernible for a long time may, by weakening the trees, render them susceptible to the disease. Repetitions of frost and disease for several seasons result in extensive defoliation, entire branches casting their needles in one year whereas they are normally retained for eight. In consequence there is a marked check to the seasonal increments in height, and the amount of secondary wood in the annual rings of affected trees becomes less and less every year [11, 16].

Leaf cast of Douglas fir is caused by *Rhabdocline pseudotsugae* [12], an Ascomycete of the group Phacidiales. It forms its fructifications (only apothecia are known) on the leaves while still on the tree, and the leaves fall after the apothecia have discharged their spores (in this respect differing markedly from those of *Lophodermium pinastri* on Scots pine, whose spores are discharged from the fallen leaves), but whether spore dissemination may still continue from the discarded leaves is not known. But infected needles of this tree may be lost spontaneously during the autumn and winter before there are any signs of apothecia on them and leaves containing the fungus may remain on the tree without forming these fructifications at all [9], a feature which suggests that *Rh. pseudotsugae* may prove to be heterothallic : several varieties of the fungus were found in America, which varied in the form of fructifications, spores, and pathogenicity [10]. The apothecia, established within the epidermis, are found on the under side of the leaf, in 1 or 2 rows on each side of the midrib ; they are brown, elliptical, and open along a median ridge exposing a deep orange-brown coloured hymenium ; the 8-spored cylindrical asci, interspersed with swollen-tipped paraphyses, are 115 to 125 by 17 to 21 μ [17] or, 113·9 to 153·3 by 15·7 to 19·4 μ [16] ; the ascospores are obliquely uniseriate or irregularly biseriate, hyaline, unicellular, but quickly become bicellular and constricted ; they measure from 17 to 21 by 7 to 10 μ [17] or 18·2 to 19·8 by 6·6 to 7·4 μ [16] ; the spores are furnished with a gelatinous sheath which expands on the wet surface of the leaf prior to germination.

Ascospores are ejected from leaves still on the tree at the time when the buds are opening, and young leaves become infected at this stage. Apothecia are developed in the following spring [17], the leaves meantime showing signs of infection before October when yellow spots appear on their under surfaces ; by the spring the spots are purplish-brown, and the apothecia developed below them are then mature, ready to disperse their spores [16]. The exact mode of infection is not known but is believed to be cuticular at either surface of the leaf [15]. Only the mesophyll appears to be exploited, the stele remaining free from invasion, and the mycelium does not pass from the needles into the stem. After accumulating in the mesophyll the fungus proceeds to build up the apothecia by collecting at various places inside the epidermis, portions of which therefore become split tangentially to accommodate the developing hymenium, in a manner similar to that described for *Lophodermium pinastri* (p. 926) or *Rhytisma acerinum* (p. 904).

The water content of infected leaves is less than that of normal leaves of the same age. Since the stomata are no longer functional, transpiration is not controlled and the presence of mycelium in the sub-stomatal spaces greatly increases the surface area of evaporation, the final effect being the loss of the leaves due to reduced water control [3].

The relative immunity of the ' green ' variety of the Douglas fir is said to be largely due to the comparatively late appearance of its buds, the tree apparently escaping the full virulence of attack because the maximum discharge of ascospores is over before the buds are open, whereas the other varieties, by maturing their buds earlier, expose their leaves when spore discharge is most active [9]. This, however, is not the general observation, for in western Oregon and Washington the green fir is liable to severe infection, though within its natural range it is not attacked to the same extent as the other varieties [2]. In general, therefore, a wider cultivation of the green variety is recommended [7, 13]. In the nursery, but

hardly practicable under forest conditions, good control has been obtained in Germany by spraying with 1 per cent. Bordeaux mixture plus alum (150 gm. per 100 litres) as an adhesive, applied at the beginning of May, and repeated at 10-day intervals for a month [5].

The Douglas fir is host for another leaf disease, called Swiss Needle Cast, also caused by an ascomycete, *Phaeocryptopus gaeumannii* (Rohde) Pet. (=*Adelopus gäumannii*) [6, 10a]. This disease has many features in common with the needle cast described, and has no doubt been long confused with it, especially as the two may occur within the same stand. It is widespread in the British Isles, especially in the western part of Britain, but is much more serious on the Continent, in Switzerland, Germany, and Austria [2a, 4a, 9a, 15a]. The black, spherical fructifications of the causal fungus are easily distinguishable from the brown, elongated apothecia of *R. pseudotsugae*; they are of the cleistocarpic kind (50 to 80 μ, diam.), formed during winter and early spring, in rows in sub-stomatal spaces on the under side of the leaves. Spore dispersal occurs in May and June. The numerous clavate asci (30 to 40 by 8 to 15 μ) contain eight uniseptate, hyaline, fusiform ascospores (11 to 15 by 3·5 to 5 μ). The spores infect the young developing leaves. Cleistocarps may continue to be formed on the affected yellowing leaves for two or three years before the leaves are cast off. Little is known about any methods for the control of this disease.

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Phomopsis Disease of Douglas Fir, *Phomopsis pseudotsugae* Wilson

This disease attacks several coniferous trees which include the Douglas fir (*Pseudotsuga douglasii*), the two species of larch (*Larix kaempferi*, *L. decidua*), the firs (*Abies grandis*, *A. alba*), *Tsuga* (*T. heterophylla*, *T. siebaldii*), the three cedars (*Cedrus libani*, *C. atlantica*, *C. deodara*), and *Sequoia gigantea*, the Californian giant fir. In Britain the disease is best known on the Douglas fir [2, 11-15]. Although the range of hosts embraces a number of American species (the Douglas fir is a

native of North America), there is no record of the occurrence of the fungus causing this disease in the North American continent and the parasite, probably a native of Europe or the Old World, attacks the American trees only after their introduction to Europe, where the disease is now widely distributed [10, 15].

The Douglas fir, on account of its phenomenal rate of growth, is extensively planted in Britain, and on the Pacific slopes of North America it is one of the most valuable of all trees, yielding a high percentage of the timber exported from that locality. In Britain (as mentioned above, p. 936) two distinct species of the tree are recognised, namely the green Douglas fir (*Ps. douglasii*), sometimes referred to as the *viridis* variety, and the ' mountain ', or blue Douglas fir (*Ps. glauca*), together with a varietal form intermediate between the two, named *Ps. douglasii* var. *caesia*; in America, however, the tree is denoted by the single species *Ps. taxifolia*.

This disease attacks the trees in early life, plants of 5 or 6 years old in the nursery being especially susceptible [1]. It causes a die-back of the leading shoot of these young trees, but older trees may also suffer severely if the younger branches are attacked and when a canker stage sets in later very heavy losses are incurred. Following upon the death of the tissues for variable distances along the young shoots or branches, partial or complete girdling of the shoots may occur, but in some cases, in addition to this loss of branches, the trunks of young trees may also be attacked at one or more places, usually about six inches above ground. These infections, occurring low down on the tree, arise from the passage of the fungus into the main stem from snags of lateral branches already diseased, and result in the formation of cankers on the trunk. It is obvious that if such cankers are developed on young trees of narrow girth to the extent that the stems are girdled, serious losses in the stand are incurred. Cankers may also arise on the larger branches resulting, if girdling takes place, in their death.

The disease may break out even within a year of planting, in the form of a die-back of the leader shoot, an observation which would appear to indicate that the young trees were either already infected or that they had contracted infection after planting out as a consequence, perhaps, of having been disturbed in the digging-up, and re-planting in a different environment, a factor which no doubt predisposes the trees to attack [12]. The fact that infection usually starts on the leading shoots or terminal parts of lateral branches, materially upsets the whole contour of affected trees, which, from deformity of growth, shorter stature, or bushy habit, contrast strikingly with the regular pyramidal form of healthy trees. Not only is the leader shoot early destroyed, but the next lateral which makes effort to replace it may also go the same way, and sometimes a third and a fourth attempt may be made by lateral shoots to carry on the growth of the tree. When the attack falls upon older, sixteen- to twenty-year-old trees, they almost invariably develop the canker phase of the disease, which, as already stated, is confined to the lower parts of the tree. In the spring diseased trees show considerable yellowing of the foliage, which deepens to a reddish brown by the middle of summer, and the trees may become an entire loss [12].

The Douglas fir is a host for several apparently closely allied species of the genus *Phomopsis* [1, 3, 7, 8, 9], of which *Ph. pseudotsugae* (Fungi Imperfecti) is the cause of the

present disease. This fungus forms its pycnidial fructifications on living and dead tissues, and though an ascomycetous type of the genus *Diaporthe*, named *D. pitya* was once thought to be the perfect stage, later work failed to substantiate this connection [8]. The pycnidia, arising under the epidermis, or bark, are densely distributed, and break through to the surface by longitudinal slits (on the Japanese larch the openings are transverse on the shoot) in the bark. They are black, partially erumpent, sub-globose, 0·1 to 1·0 mm. in diameter, bluntly conical, ostiolate; incompletely locular or multilocular. The pycnospores, on long colourless sporophores 12 to 14 μ long, are hyaline and unicellular, from 5·5 to 8·5 by 2·2 to 4 μ [12, 13]. The ripe spores are exuded through the ostiole in a whitish tendril; some larger pycnidia are multi-ostiolate. There are species of *Phomopsis* with two kinds of pycnospores, one kind fusiform in shape (the 'A' spores), as here described, and a second kind ('B') longer and curved, which this species does not possess.

The Douglas fir appears to be very sensitive to the action of frost, especially within the first 10 years after planting out [4], and in low-lying hollows filled with cold stagnant air or surrounded by a humid vegetation dominated by grasses and rushes, the tree is rendered very susceptible to this die-back and canker trouble. On the other hand, when the trees are more or less protected by a different type of vegetation such as bracken or bramble, they are much less liable to frost injury, and under such conditions are hardly ever attacked by this disease.

Infections appear to occur at places on the shoots where surface injuries have followed frost action [5, 6], or through wounds inflicted by insects, voles, rabbits, deer, or by pruning. Inoculations made into cuts on the branches develop successfully at any time of the year [12]. The die-back condition may proceed on its downward path of infection so as to involve and kill the whole of the current year's shoot, and may sometimes extend into the tissues of the second year and even into older growth. Whilst there is little discoloration at the seat of infection at the start, later a slight depression develops which becomes more and more sunken and dark brown in colour as the surrounding parts increase in girth by normal secondary thickening. The mycelium is present mostly in the cortex and resin canals, and quickly proceeds to replace the host tissues disintegrated. A canker on the trunk or on a large branch is initiated, as already indicated, from the broken end of a small lateral branch which had become infected, the mycelium spreading from it into the tissues of the stem in all directions; if the stem is comparatively thin, the canker may encircle it completely, and as its progress entails the destruction of all the tissues down to the wood cambium, transmission of elaborated food from the leaves is checked, with the result that the upper limits of the lesion become much swollen, and a hypertrophy of the stem just above the point of attack is often a characteristic symptom on four- to eight-year-old trees. In the case of older trees, however, canker formation on the trunk near the base is not usually accompanied by any marked hypertrophy, for the reason that older stems are rarely girdled by the disease. During development, the edge of a canker becomes raised by callus formation and numerous resinous blisters break out on the dead bark and around the lips of the cankers. The small black pycnidia are again in evidence around the edges of the cankers. Older trees of Douglas fir have much capacity for healing up their cankers, and on trees of twenty years and upwards all signs of cankers are usually obliterated. But despite such recovery

with advancing age, scars of the disease are still evident in the felled timber, for there must always be a blemish in the wood where abnormal secondary tissues, formed by an affected or frosted cambium, adjoin healthy wood; such lines of fault in the timber are seen as dark zones, and, while the physical effect of the flaw may be small where only a single canker occurred, numerous cankers render the wood of little value as structural timber.

Every precaution should be observed to avoid injury to young trees in the nursery, or during planting out, so as to prevent wounds. There can be little doubt that frost, especially when followed by rapid thaw, is responsible for much injury to this and many other trees [6], and, when planting in frost areas cannot be avoided, it is recommended that a protective nurse crop such as birch or mountain pine be adopted, and in order to avoid wet, cold conditions, draining the area may also be found advisable. Little appears to be known about any varietal forms of Douglas fir in respect of any differences that might exist as to the time of leafing (flushing), but it would obviously be a great advantage to get a late flushing kind that would not be so sensitive to late frosts. No sources of infection should be left unburnt near the nursery, and any young trees suspected of harbouring the disease should be removed and destroyed [12].

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Other Rots of Standing Timber

(a) *CONIFERS*

Butt rot of coniferous trees in Great Britain affects larch rather more often than spruce, with Douglas fir coming next; the pines suffer little damage from it until they are 60 to 70 years old and the silver fir (*Abies alba*) is even more resistant. *Fomes annosus* (described above, p. 915) is by far the commonest cause, with *Polyporus schweinitzii* probably next in importance. *Fomes pinicola* causes a butt rot of spruce comparatively rarely in Britain. The common butt rot of conifers, caused by *Armillaria mellea* (described above, p. 907), is due to a limited extension of the fungus from the main roots into the base of the trunk. By itself it is not a cause of serious trunk heart rot, but it frequently accompanies and may open the way to the true butt rots such as that caused by *Fomes annosus*. Heart rots of conifers that ordinarily arise as wound infections include, in Britain, those due

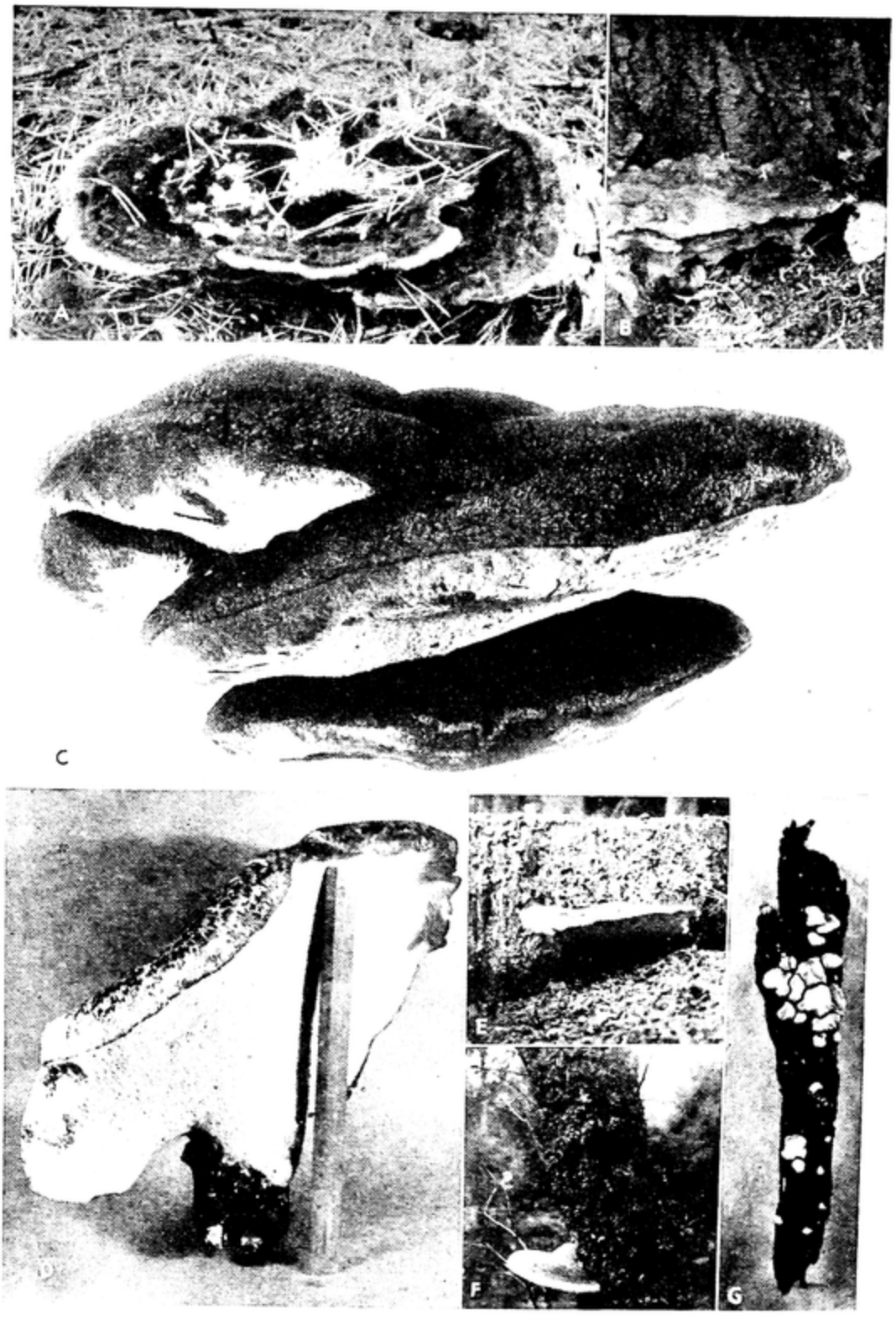


FIG. 433. *A*, *Polyporus schweinitzii*. Fruit body on soil near base of pine tree (photo by Wyatt Smith, *Principal Decays of Softwoods used in Great Britain*, by permission of H.M.S.O.) ($\times \frac{1}{2}$). *B*, *Fomes fraxineus*, on poplar (Mesopotamia) (*Ann. App. Biol.* 29). *C*, *Polyporus hispidus*, on a living ash at Nuneham Park, Oct. 1939 (*Principal Decays of British Hardwoods* and *Ann. App. Biol.* 29). *D*, *Polyporus squamosus*, from Nuneham Park, Oct. 1938 (Crown copyright reserved); reproduced by permission of the Director, Forest Products Research Laboratory. *E*, *Fistulina hepatica* (*Principal Rots of English Oak*, H.M.S.O.) ($\times \frac{1}{5}$). *F*, *Polyporus betulinus* (Crown copyright reserved); reproduced by permission of the Director, Forest Products Research Laboratory. *G*, *Stereum frustulatum* (*Principal Rots of English Oak*, H.M.S.O.)

to *Fomes pini* and *Stereum sanguinolentum*. As these frequently affect the upper portion of the bole they are sometimes designated top rots. All these fungi show little host specialisation, each occurring on various coniferous trees and are, therefore, discussed here in a general way. The histological effects on the wood of fungi of this type have been already discussed from a number of examples in Chapter VI.

Polyporus schweinitzii (Fig. 433 A) is found in larch and spruce in Britain as the cause of a brown 'butt rot' next in prevalence to that due to *Fomes annosus*. In south-eastern Scotland Scots pine seemed to be attacked after the age of 70 to 100 years [10a]. In the United States, where different isolations have been found to differ considerably in cultural and physiological characters [8], it causes much greater damage than the latter, following *Fomes pini* in prevalence and attacking various species of pine, spruce, fir, and larch, while in some of the pine forests of central Russia it is commoner even than the ubiquitous *F. pini*. In Sitka spruce and Douglas fir [26] the decay begins as faint yellowish elongated streaks in the heartwood, followed by a brownish or reddish-brown discoloration and a cubical brown rot which becomes friable and is accompanied by a great loss in the toughness of the wood (Fig. 435 E). Even in the early stages of attack Sitka spruce may undergo a large reduction in strength and in resistance to impact; part of the seriousness of 'dote' in airplane spruce is due to inability to detect this weakness. Some of the affected pines show a resin flux. It has been proved experimentally that the fungus is a definite parasite of the roots of *Pinus strobus* under certain conditions, while in second-growth Douglas firs infection in the Pacific north-west is usually through fire scars. Sporophores may be found on superficial roots as well as on the bole near the ground or, occasionally, well above ground-level.

Fomes pinicola (Fig. 434 D) also causes a red-brown cubical heart and sapwood rot of various conifers in Europe, North America, India, and Japan [21]. It is sometimes found in certain hardwoods such as poplar, birch, and willow, and has been recorded as a cause of heart rot of peach and plum in America. Like *Fomes annosus* and *Polyporus schweinitzii* there may be little external indication of its presence in the bole, but it can cause a rapid destruction of the wood with removal of cellulose and the development of cracks filled with white mycelium. Unlike most other wood-destroying fungi, considerable differences in cultural characters and physiological activity has been found in isolations of *F. pinicola* from different coniferous hosts in North America, but these differences do not extend to host specialisation [13]. As a butt rot it has been reported to cause severe injury to pine and spruce but it is comparatively rare on the latter host in Britain.

Fomes pini (Fig. 434 H) is perhaps better known to foresters as *Trametes pini*, though its perennial, stratified, porous hymenium places it better in the genus *Fomes*. It is extremely prevalent and destructive in large areas of the coniferous belt of the northern hemisphere, occurring on many hosts (pine, spruce, fir, larch, deodar, Douglas fir, *Thuja*, etc.) and causing what is often termed 'heart rot', red or ring scale, or, in the United States, conk rot. It is the most widespread of all the wood-rotting fungi of North America and is, perhaps, the most destructive of all fungus parasites of coniferous trees. Infection of the heartwood occurs ordinarily through broken branches containing heartwood, most frequently in

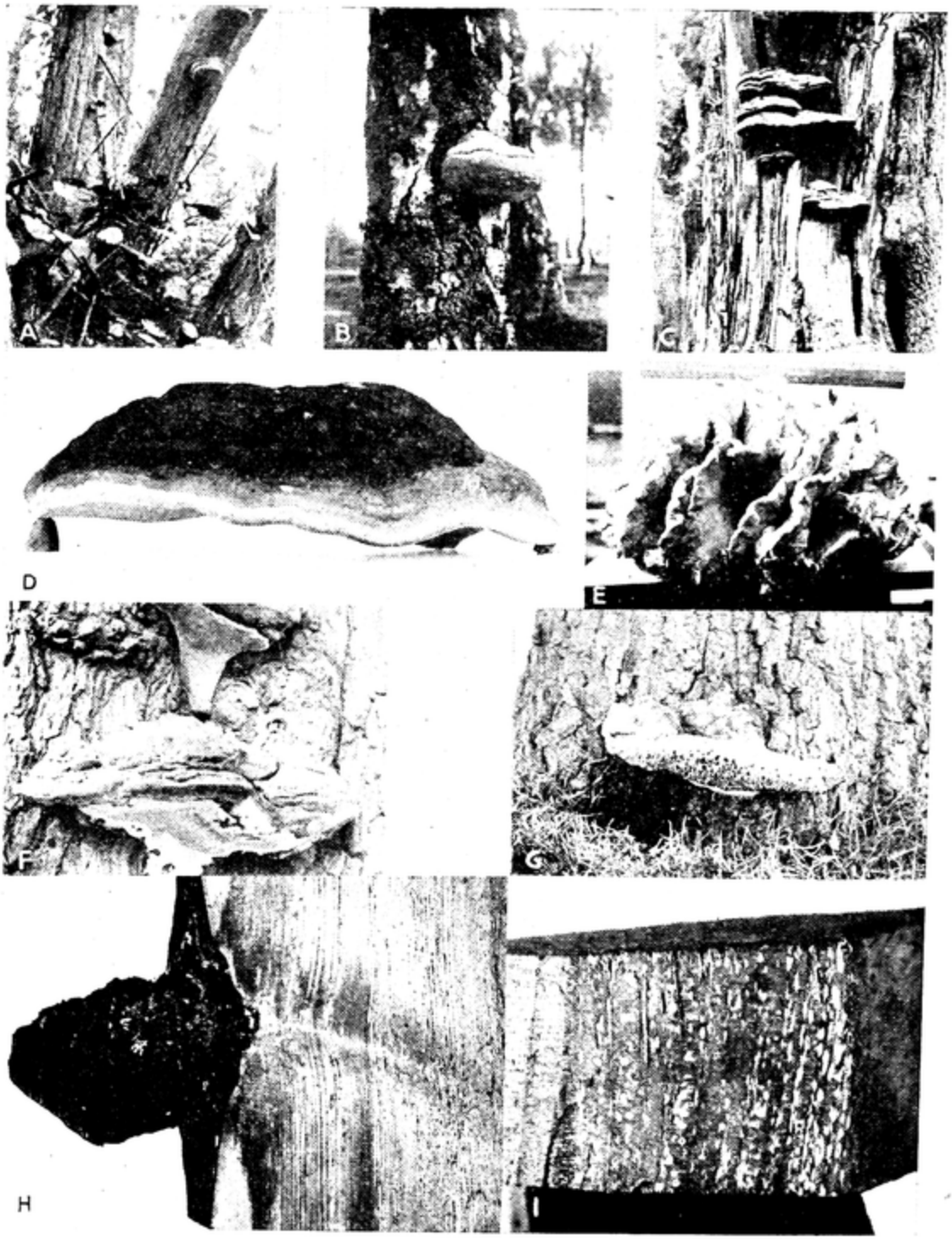


FIG. 434.—*A*, *Fomes igniarius*, fruit body on willow, Nuncham Park (Crown copyright reserved); reproduced by permission of the Director, Forest Products Research Laboratory. *B*, *Fomes fomentarius*, fruit body on birch, Scotland (*Ann. App. Biol.* 29) ($\times \frac{1}{10}$). *C*, *Ganoderma applanatum*, on living beech, Knole Park, Sevenoaks, 1933 (*Ann. App. Biol.* 29) ($\times \frac{1}{10}$). *D*, *Fomes pinicola* (*Principal Decays of Softwoods used in Great Britain*, H.M.S.O.). *E*, *Polyporus sulphureus*, from Beech Knole, Oct. 1929 (*Principal Rots of English Oak*, H.M.S.O.) ($\times \frac{1}{5}$). *F*, *Fomes ulmarius*, on common elm, Oxford (Crown copyright reserved); reproduced by permission of Director, Forest Products Research Laboratory ($\times \frac{1}{7}$). *G*, *Polyporus dryadeus*, at base of oak, Knole Park, Sevenoaks, 1934 (Crown copyright reserved); reproduced by permission of the Director, Forest Products Research Laboratory ($\times \frac{1}{7}$). *H*, *Trametes pini*, on Scots pine (*Principal Decays of Softwoods used in Great Britain*, by permission of W. R. Day) ($\times \frac{1}{3}$). *I*, *Trametes pini*, wood decayed by the fungus (Crown copyright reserved); reproduced by permission of the Director, Forest Products Research Laboratory

the upper bole, so that some of the hosts are seldom attacked before they are about 30 years old [15]. Later the rot may extend into the sapwood; sporophores are generally found at the site of the old branch stubs and there may be strands of felted brown mycelium at the base of dead twigs. A copious resin flux may prevent the external development of the fungus. Wound infection from spores is believed sometimes to occur at the base of the tree and to cause a limited butt rot, but usually the whole trunk becomes infected. The fungus is one of those causing ultimately a white rot of the wood, for it attacks lignin equally with cellulose, with the result that though the first reaction is frequently a purple or reddish discoloration turning to yellowish brown, a white pocket rot follows, marked in Douglas fir by a riddling of the heartwood with small white cavities between which the wood appears sound; different hosts vary somewhat in the type, colour, and sequence of changes produced in the heartwood [23], but infection usually brings about a great loss of strength, though the trees may live to a great age owing to the relative immunity from attack of their functionable wood. In the Aleppo pine in the south of France the spring wood succumbs to decay before the autumn wood; the fungus ceases to grow when the host is killed, but it is then often followed by *Fomes pinicola* which completes the decomposition of the wood [10]. In Germany, where *T. pini* causes losses amounting to millions of marks annually, a systematic campaign for its eradication from the State forests by felling all affected trees was in force for some time before the war.

Stereum sanguinolentum (Fig. 435 D) causes a 'red stain' top rot of the heartwood of living spruce, fir, pine, larch, and Douglas fir in Europe, North America, and Australia. Like *Trametes pini* it enters through branch stubs and may extend down inside the bole to the base of the tree, causing a butt rot in the older trees. It belongs to the white rotting group (like all species of the genus *Stereum*), causing first a reddish-brown mottle in the heartwood and then white pockets of decay. In a severe outbreak observed in Idaho [14] external symptoms were few and inconspicuous, though in Douglas firs the base of the stem showed whitish streaks and there was a marked resin flux in spruce and fir; at the collar of the trees the bark was loose and showed a white mottling on its inner surface, while the sapwood was greyish brown and soft; the course of the infection appeared to be from the bark to the sapwood and thence into the heartwood, instead of the usually reported direct invasion of the heartwood. In balsam fir (*Abies balsamea*) in Canada [18] the affected heartwood is firm, water-soaked, and reddish brown, with rays of the same colour extending out for an inch or two into the sound light-coloured wood; white mycelial sheets may form in the wood and persist until an advanced stage of decay when the wood turns light brown, loses weight, and becomes dry and friable. Sporophores are found on the standing trees and develop freely on the dead wood.

(b) *HARDWOODS*

Amongst the more important causes of decay in the wood of living dicotyledonous trees in Great Britain [7a] are *Polyporus sulphureus, P. squamosus, P. betulinus, P. hispidus, P. frondosus, P. dryadeus, Fomes fomentarius, F. igniarius, F. fraxineus, F. ulmarius, Ganoderma applanatum, Stereum hirsutum, S. gausapatum, S. frustulatum,*

and *Fistulina hepatica*. Most of these attack several hosts and their range may even include conifers, just as some of the softwood parasites already described can attack hardwoods; a few, however, are more closely specialised, as *Polyporus betulinus* on the birch and *Stereum gausapatum* on oak.

Polyporus sulphureus (Fig. 434 E) is a common cause of butt rot of the heartwood of oaks in Europe (1, 7), the United States, South Africa, and elsewhere, entering usually through wounds and fire scars; a variety of the fungus has been described as a cause of root rot of oaks in the United States. It causes a severe heart rot of sweet chestnut (*Castanea vesca*) on the Caucasian side of the Black Sea and attacks *C. dentata* in America. Other hosts include beech, birch, poplar, willow, walnut, acacia, eucalyptus, pear, plum, and cherry. It is common on conifers in Siberia and on yew near the Black Sea, while firs (*Abies*) are its chief softwood hosts in Canada where, and in the United States, Douglas fir and larch are also infected. Conifers are less liable to be attacked than hardwoods in Europe. It causes a cubical brown rot due to the formation of shrinkage cracks which isolate the wood into cubes, the cracks themselves being frequently filled with thin mycelial sheets; the water-conducting tissue may be extensively decayed while the medullary ray cells are still intact. This is followed by a dry powdery decay which ends in hollowing out of the bole; death may ensue in a few years (Fig. 435 A). Poplars suffer from a red rot and cracking of the wood.

Polyporus squamosus (Figs. 34, 433 D) occurs on various hardwoods in Europe, North America (where it is not common) (10 b), India, and Australia. In Britain it is the commonest cause of decay of the heartwood of the walnut and of the upper part of the elm, and it is frequently found on sycamore and other species of *Acer*. The latter (maples) seem to be subject to its attacks in New England, as are beeches in the Carpathians. Horse chestnuts (*Aesculus hippocastaneum*) and species of *Pyrus* are other British hosts of this fungus. It is a wound parasite, entering chiefly through broken branches possibly in the same way as *Stereum purpureum* (p. 763), since its spores are small enough to be sucked into the vessels (3a). The decay is of the white-rot type and is confined mainly to the heartwood, so that the fungus cannot be regarded as an active parasite of living elm tissues though responsible for much of the loss of large branches and even whole trees, to which the elm is notoriously liable from weakening of the heartwood. The mycelium extends upwards and downwards from the seat of infection, at first in the central tissues then extending outwards; eventually the trunk becomes hollow, following the reduction of the wood to a stringy or spongy white mass often mixed with mycelial wefts. The hyphae penetrate the cell walls freely through bore-holes which subsequently enlarge. The walls are progressively thinned, the less heavily lignified ones first. White strands or sheets of mycelium divide up the wood while black lines occur especially near the sites of the sporophores, forming the so-called xylostromata or pseudosclerotia.

Polyporus betulinus (Fig. 433 F) occurs throughout most of the geographical range of the birch, a host to which it is restricted in nature (16). In England it is much the commonest cause of decaying in standing trees, but this is not the case in the north of Scotland where *Fomes fomentarius* is equally prevalent on this host. It is a wound parasite, affecting first the sapwood and then extending irregularly

into the heartwood, leaving islands of sound tissue behind. The rot produced in sap- and heartwood is red-brown and cubical, the affected tissues becoming soft, light in weight, and crumbling to powder. Quite young birches which have been weakened or injured from other causes may become infected, as penetration may occur without exposure of the heartwood.

Polyporus hispidus (Fig. 433 C) attacks a wide range of forest, ornamental, and fruit trees in Europe, America, Australia, and Asia. In Britain it is common on the white ash (*Fraxinus excelsior*) and is less often seen on the black ash (*F. nigra*) [22], to which it is almost restricted in the United States [2]. Infection is generally through branch stubs (which may be devoid of heartwood) towards the upper part of the bole. From the medullary rays and wood parenchyma the hyphae pass through the pits into the conducting tissues, extending up and down rapidly in the heartwood and more slowly in the sapwood. Passage through the tracheid walls is by bore-holes and may lead to the lumina being choked with mycelium. The affected wood is changed to a uniformly soft spongy mass which may be chalky in appearance and is delimited by dark-brown lines, or a fine white streaky mottle may result. The wood becomes ' brash ' in texture and undergoes considerable loss of weight ; mechanical tests may be necessary to reveal early injury to it. Eventually pockets of decay form, filled with wefts of mycelium. In Asiatic Russia *P. hispidus* is very prevalent on walnut and has been reported as killing whole apple orchards ; old apple trees are commonly decayed by it in Britain, where also the elm, walnut, plane, and sycamore are occasional hosts. In France and Kashmir, mulberries (including those used for feeding silkworms) are often attacked. The fungus is also associated with a canker of the oak in the United States due to infection of the heartwood from a branch stub with the production of oval areas of white crumbling wood surrounded by swollen callused margins and covered by bark ; these may be several feet long and occur from ground-level to at least 35 feet up the stem, so that they greatly diminish the value of the wood [28].

Polyporus frondosus (Fig. 435 F) and *P. dryadeus* (Fig. 434 G) cause a white butt rot of oak heartwood in Britain. The former [5], which probably starts from the roots, resembles in its effects the commoner rot caused by *Stereum frustulatum* (Fig. 435 B) except that the white pockets of decay are less well defined. *P. dryadeus* also affects the roots but seldom extends more than a few feet into the base of the trunk and its action as a parasite is slow. In the white fir, *Abies concolor*, the sporophores of *P. dryadeus* may be found at the collar of infected trees.

Fomes fomentarius (Fig. 434 B) is widely distributed in Europe, North America, Asia, Australia, and North Africa on beech, birch, poplar, maple, olive, and some other hosts. Though the heartwood is usually first to show signs of decay in living trees, in culture the fungus can destroy heartwood and sapwood at equal rates. In the beech it may cause a vertical furrowing of the trunk, due probably to the cambium being reached and killed in elongated strips while secondary thickening proceeds on either side. Sporophores are reported to be rarely produced on living infected birch and beech in New England. In Germany and south-eastern Europe it is not uncommon on old beech stands, sometimes decaying mainly the sapwood and leaving the heartwood intact though reddened. As lignin and cellulose are attacked simultaneously [12] the fungus belongs to the ' white rotting ' group of

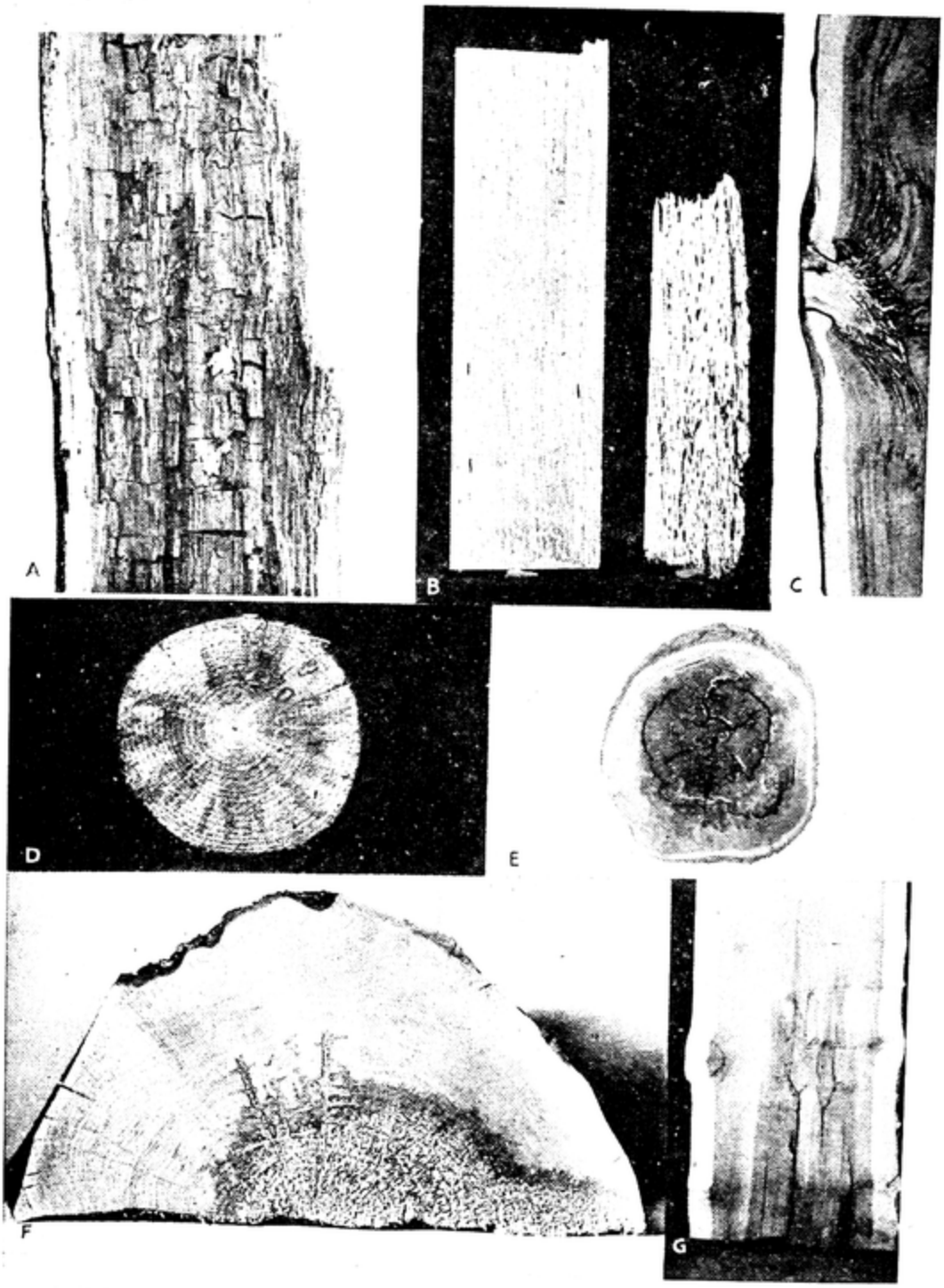


FIG. 435.—*A*, *Polyporus sulphureus*, the rot in oak (*Principal Rots of English Oak*, H.M.S.O.) (× $\frac{2}{3}$). *B*, *Stereum frustulatum* (*Principal Rots of English Oak*, H.M.S.O.). *C*, *Stereum gausapatum* (= *S. spadiceum*), showing white pipe rot; infection takes place through small dead branches and spreads into main trunk (*Principal Rots of English Oak*, H.M.S.O.) (× $\frac{1}{12}$). *D*, *Stereum sanguinolentum*, causing a red discoloration in Norway spruce (*Forestry*, 11, 1937) (*Principal Decays of Softwoods used in Great Britain*, H.M.S.O.) (× $\frac{1}{6}$). *E*, *Polyporus schweinitzii*, cross-section of decayed pine trunk; note mycelium in the cracks (about $\frac{1}{3}$ natural size) (*Principal Decays of Softwoods used in Great Britain*, by permission of W. R. Day). *F*, *Polyporus frondosus* (*Note on a Heart Rot of Oak Trees caused by Polyporus frondosus* Fr.; *Forestry*, 14) (× $\frac{1}{2}$). *G*, *Fomes ulmarius*, on elm, decayed from butt (*Ann. App. Biol.* 29) (× $\frac{1}{15}$)

wood destroyers, its effect being eventually to produce a white mottled rot with black lines demarcating the decayed areas, which become brittle. In Scotland, Germany, Russia, the United States, and Canada *F. fomentarius* is prevalent on birch, especially when over-mature. The Scottish form of the parasite has sometimes been confused with *F. igniarius* (Fig. 434 A) or *P. nigricans* but has been found to be only a hard north European form as distinct from the soft, light-coloured, rapidly growing type occurring farther to the south [17]. The rot it causes on this host has been described as laminated, as distinguished from the cubical, pocket, and powdery rots due to other fungi. The mycelium develops chiefly in the medullary rays and vessels of the wood, often completely filling the vessels. On poplars in France it is believed to enter through the wounds caused by pollarding, and its subsequent progress is slow; in beech, also, infection through the crown is frequent, but the top rot of this host caused by it commonly in Scotland and on the Continent is replaced in England, where *F. fomentarius* is very rare, by that due to *Ganoderma applanatum*. Sycamore (*Acer pseudo-platanus*) and alders are also attacked in Denmark, cork oaks (*Quercus suber* and *Q. occidentalis*) in the Caucasus, and walnuts in central Asia.

Fomes igniarius (Fig. 434 A) is not uncommonly found in company with *F. fomentarius* on birches, and the decay due to the latter fungus in Scotland was at one time often attributed to it. In Canada also it is a much less common birch parasite than *F. fomentarius*; thus, in an over-mature birch stand in Quebec *F. fomentarius* was the cause of 71 per cent. of the infections, *F. igniarius* of 8 per cent., and both together of 21 per cent. *F. igniarius* has much the same geographical distribution as *F. fomentarius* and attacks an even wider range of economically important trees than the latter, but in England it is almost confined to willows. In some parts of the United States and northern Europe it is the chief cause of heart rot of aspens (*Populus tremula*, *P. tremuloides*, and *P. grandidentata*) [19, 27]. Other hosts include white poplar, birch, oak, beech, elm, maple, willow, and alder as well as fruit trees such as apple, pear, plum, walnut, almond, and occasionally the grape-vine. There is evidence that three forms of the fungus can be distinguished morphologically and culturally in America, one limited to aspen, one to birch, and the third on miscellaneous hosts [30]. Like *F. fomentarius* it causes a white rot [27a] which, in aspen, at first affects only the central heartwood, causing the development of a yellowish-white area bounded by streaky greenish-black lines; these extend and the wood breaks down at the centre. Later the rot extends to the sapwood and may reach and kill the cambium. The later stages of decay are marked by black concentric rings; eventually the wood of both aspen and birch is completely decomposed, the fungus being one of those by which lignin, cellulose, and pentosans are all equally dissolved. Entry is through wounds and fire scars as well as through branch stubs, but infection is usually limited to a few feet upwards and downwards from the seat of infection, this point being frequently marked by the development of a sporophore. In yellow birch (*Betula lutea*) cankers covered by the bark and a hard black fungal layer may result, in the United States, in sporophores developing around their margins after death of the tree [4].

Fomes fraxineus (Fig. 433 B), in addition to the ash, has been found on living 'acacia' (*Robinia pseudacacia*) [20], elm, and beech in England, and on various other

trees in Europe and North America. It is a sapwood and heartwood parasite causing in culture on ash wood much loss of weight in the early stages; later the heartwood is converted into a crumbling whitish decayed mass. Though comparatively rare in England, it may cause a serious butt rot of affected trees.

Fomes ulmarius (Figs. 434 F, 435 G) is common on elm trees in England where it is the chief cause of butt rot, as *Polyporus squamosus* is of the upper wood rot, of this host. It causes a brown rot in which the wood separates into oblong blocks, and affects usually a conical area of heartwood at the base of the tree, sporophores developing near ground-level or inside old basal hollows. Infection seems to be ordinarily through bark wounds.

Ganoderma applanatum (Fig. 434 C) occurs on a wide range of hosts in Britain, where it is commonest on the beech and is probably the most frequent cause of decay in standing poplars; it is also often one of the causes of rot in living willows, horse chestnut and sycamore and less often in elm, walnut, and various other trees. It is found both in temperate and tropical countries, appearing in a number of growth forms which have sometimes been given distinct names (14a). It causes a white rot of the heartwood (31), beginning with whitish streaks which, as they decay, tend to separate the wood into oblong pieces. Later the decay extends, causing a uniform, white, soft spongy rot, demarcated from the sound wood by a dark-brown zone of cells containing wound gum and tyloses. This zone resembles in character and function the blocking layer which has been already described as protecting pruning wounds against infection by such fungi as *Stereum purpureum* (p. 763); on the mulberry in America it may not develop (2a) and the hyphae may extend into the apparently sound wood. Another type of dark line develops between areas of wood occupied by *Ganoderma applanatum* and those colonised by other fungi (3b). The hyphae penetrate the cell walls by bore-holes which subsequently enlarge. The fungus is a wound parasite, entering trees such as the beech through branch stubs, especially those left by loss of the larger branches. In tropical plantation crops, such as oil palms, tea, coffee, and the like, the roots and base of the stem are sometimes the sites of greatest injury.

Stereum hirsutum is found usually on the oak but also attacks beech, stone fruit, and the vine in Europe and eucalyptus in South Africa. It can cause a serious decay of heartwood and sapwood of the white rotting type but beginning as brown spots interspersed with whitish strips and patches. The heartwood may resist attack long after the sapwood has been invaded. The form on the vine, regarded at one time as a distinct species, *S. necator*, is the cause of the serious disease known as 'esca' or 'apoplexy' in southern and eastern Europe and the Levant. The fungus is a common and very active cause of decay in cork oaks (*Quercus suber* and *Q. occidentalis*) in the Caucasus and of the former species in North Africa, but in English oaks it seems to cause injury mainly to the wood after felling.

Stereum gausapatum (Fig. 435 C) is similar in its effects to *S. hirsutum* but is restricted to the oak, to which it is a major cause of damage in the United States (9, 24), France, and certain British forests. In the latter its prevalence has been ascribed to over-wide spacing of the young trees, with the result that when the canopy closes many of the lower large branches that are killed contain heartwood and afford channels of entry to the fungus. In pollarded oaks entry is apparently

directly into the parent stump. The fungus causes a white ' piped rot ' of heart- and sapwood, beginning as small brown oily spots and then spreading in concentric zones. Eventually yellow or brown bands form, especially in the spring wood, separated by white elongated streaks which give a characteristic mottled appearance. In the final stages all the wood becomes pale, brittle, and useless. Spread downwards is rapid but upwards very slow ; the butt usually escapes but the rest of the trunk may be internally decayed. Sporophores are usually found near the points of entry and furnish the best means of distinguishing between the rots caused by *S. gausapatum* and *S. hirsutum* since the decay caused by both is very similar. The name *S. spadiceum* which has been used in France and Britain is now usually considered to be a synonym.

Stereum frustulatum (Fig. 433 G) is the cause of ' partridge wood ' of the oak, a decayed condition of the heartwood in which irregular white spots appear in the browned wood, usually of the upper part of the tree, and develop into oval or spindle-shaped cavities, separated by firm brown walls. External symptoms are slight, but the infected wood rapidly becomes useless. Like *Stereum gausapatum* entry is probably through wounds exposing the heartwood, but it also takes place through fire scars both in the base and upper part of the trunk.

Fistulina hepatica (Fig. 433 E), the ' beefsteak fungus ', causes a sap and heart rot of the oak and sweet chestnut, producing, before infection has advanced sufficiently to injure the strength of the wood, the so-called ' brown oak ' which is much valued by timber merchants [6]. Decay ceases to progress when the timber is prepared for use, the fungus losing vitality gradually after felling. The fungus is common on pollarded oaks, presumably as a wound parasite, and is stated to cause a rot similar to, but less common than that due to *Stereum gausapatum* in the United States ; in Britain the rot is greyish brown, resembling tortoiseshell. In several species of eucalyptus, including the valuable jarrah wood (*E. marginata*), *Fistulina hepatica* produces what is known in Australia as ' pencilled wood ' [29]. The wood is marked by dark streaks about a millimetre broad extending vertically and also radiating outwards from the centre. They are caused by an infiltration of the lumina of fibres and vessels with ' kino ', a heavy, dark reddish-brown gum. The fungus seems to remain active in the wood of living trees for many years, causing a heart or pith rot, a decay of pockets of sapwood, and yellow-edged pin-holes, in addition to the pencilling [25].

Control of Fungi causing Decay in Trees.—It is evident that few of these fungi are susceptible to direct control. Indirect measures such as close planting to prevent die-back of large branches containing heartwood when the canopy closes, or the growth of mixed instead of pure stands, have a certain value against some fungi. More important is the choice of races of the host adapted to the locality in which they are grown. A sharp distinction must be drawn between the conditions prevailing in the virgin forests which still cover a good deal of the land surface of the globe and the replanted woodlands of the British Isles and much of continental Europe. In the former, decay is predominantly due to fungi that attack the older trees and may do little damage to those of the age classes below 60 or 80 years ; in the latter it is mainly caused by species like *Fomes annosus* that can

infect young stands. In the first type a knowledge of the optimum date of felling is all important, and once a regulated rotation is established with this in mind, great improvement may be expected [11]. It has been pointed out, for instance, that *Fomes igniarius* is unlikely to cause much damage to aspen plantations grown in rotations of about 50 years. Selective felling of infected trees is not easy to carry out in regularly worked forests, but reference has been made above to the German efforts to accomplish it as a means of reducing injury from *Trametes pini*, and it may sometimes be practicable in other cases. In closely managed forests on the Continent decay is not uncommonly reduced by the removal of damaged side branches close to the trunk so as to secure a cleanly healing wound. In park and avenue trees this should be regularly done and a wound dressing applied when practicable.

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